

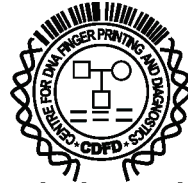
सी डी एफ डी **CDFD**

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अधिदेश
Mandate

अघिदेश

सीडीएफडी सोसाइटी के संगम ज्ञापन तथा नियम एवं विनियमों में बताए गए अनुसार डीएनए फिंगरप्रिंटिंग एवं निदान केंद्र की स्थापना जिन उद्देश्यों के लिए हुई वे निम्न प्रकार हैं :

- i. पितृत्व विवाद, आप्रवास और अस्पतालों में नवजात शिशुओं की अदला-बदली जैसे मामलों में निजी पक्षों सहित विविध अभिकरणों के लिए पर्याप्त अदायगी पर डीएनए प्रोफाइलिंग और उससे संबंधित विश्लेषण का वैज्ञानिक अनुसंधान करना।
- ii. अपराध अन्वेषण अभिकरणों को डीएनए फिंगरप्रिंटिंग और उससे संबंधित विश्लेषण तथा सुविधाएं प्रदान करना।
- iii. अपराध अन्वेषण और परिवार मामलों में डीएनए प्रोफाइल विश्लेषण और उससे संबंधित तकनीकों के साक्ष्य संबंधी मूल्य को समझने में पुलिस कर्मियों, न्यायिक वैज्ञानिकों, वकीलों तथा न्यायपालिका की सहायता करना।
- iv. आनुवंशिक अव्यवस्थाओं को संसूचित करने हेतु डीएनए नैदानिक विधियां सिद्ध करना और इस प्रकार के संसूचन के लिए संपरीक्षाएं विकसित करना।
- v. पादप और जंतु कोशिका माल, कोशिका लाइनों के प्रमाणीकरण के लिए डीएनए फिंगरप्रिंटिंग तकनीकों का उपयोग करना और ऐसे प्रयोजनों के लिए आवश्यकतानुसार नई संपरीक्षाएं विकसित करना।
- vi. डीएनए फिंगरप्रिंटिंग तकनीकों पर प्रशिक्षण प्रदान करना।
- vii. मूलभूत, अनुप्रयुक्त अनुसंधान एवं विकास कार्य करना।
- viii. देश में चिकित्सा संस्थाओं, जन-स्वास्थ्य अभिकरणों और उद्योग को परामर्शी सेवाएं प्रदान करना।
- ix. केंद्र के उद्देश्यों से संगत क्षेत्रों में विदेशी अनुसंधान संस्थाओं एवं प्रयोगशालाओं और अन्य अंतरराष्ट्रीय संगठनोंके साथ सहयोग करना।
- x. अनुसंधान छात्रों को स्नातकोत्तर उपाधियों के लिए पंजीकृत कर सकने के प्रयोजन हेतु उच्चतर अधिगम के मान्यता प्राप्त विश्वविद्यालयों एवं संस्थाओं के साथ संबंध स्थापित करना।
- xi. भारत सरकार, राज्य सरकारों, देश में स्थित पूर्व संस्थाओं / न्यासों, व्यक्तियों और अन्य गतिविधियों के लिए अंतरराष्ट्रीय संगठनों सहित विदेशी स्रोतों से आर्थिक सहायता प्राप्त करना।
- xii. केंद्र सरकार के पूर्व अनुमोदन से प्रशिक्षण कार्यक्रमों, वैज्ञानिक अनुसंधान और अन्य गतिविधियों के लिए अंतरराष्ट्रीय संगठनों सहित विदेशी स्रोतों से आर्थिक सहायता प्राप्त करना।
- xiii. केंद्र की गतिविधियों को चलाने के लिए जैसा आवश्यक या सुविधाजनक हो, कोई भी संपत्ति चल या अचल या भवनों एवं निर्माणों को निर्मित करने, सुधार करने, परिवर्तित करने, गिरा देने या मरम्मत करने हेतु उपहार, क्रय, विनियम, पट्टा, भाडे पर लेने द्वारा या अन्यथा किसी भी तरह अर्जित करना।
- xiv. केंद्र के प्रयोजन हेतु, भारत सरकार और अन्य प्रोनोटों, विनियम पत्रों या अन्य परक्राम्य लिखतों को आहरित करना और स्वीकार करना, तैयार करना और पृष्ठांकित करना, बट्टा करना और परक्रामण करना।
- xv. केंद्र को सौंपी गई निधियों या धन को निवेश करने के लिए, ऐसी प्रतिभूतियों को खोलने या ऐसे तरीके से, जो कि समय-समय पर शासी परिषद द्वारा निर्धारित किए जाते हैं, इस प्रकार के निवेश को विक्रय या पक्षांतरण करना।

- xvi. उक्त सभी उद्देश्यों या उनमें से किसी उद्देश्य की प्राप्ति के लिए सभी ऐसे अन्य विधिसम्मत कार्य, जैसा आवश्यक, प्रासंगिक या सहायक हो, करना।
- xvii. केंद्र के उद्देश्यों को वास्तविक बनाने के लिए प्रोफेसरों, अन्य संकाय पदों, अभ्यागत अध्येतावृत्तियों सहित अध्येतावृत्तियों, अनुसंधान एवं संवर्ग पदों, छात्रवृत्तियों आदि को संस्थापित करना।
- xviii. केंद्र के वैज्ञानिक एवं प्रौद्योगिकीय कार्य के लिए प्रयोगशालाओं, कार्यशालाओं, भंडार, पुस्तकालय, कार्यालय और अन्य सुविधाओं को स्थापित करना।
- xix. तकनीकी जानकारी को उद्यमकर्ताओं और उद्योगों से प्राप्त या उनको अंतरण करना, और
- xx. पेटेंटों, डिजाइनों एवं तकनीकी जानकारी जो कि केंद्र द्वारा विकसित की गई हो, को पंजीकृत करना और केंद्र के हित में ऐसे पेटेंटों / डिजाइनों / तकनीकी जानकारी के किसी भाग को अंतरण करना।

MANDATE

The objectives for which the Centre for DNA Fingerprinting and Diagnostics (CDFD) was established, as enumerated in Memorandum of Association and Rules and Regulations of CDFD Society, are as follows:

- i. To carry out scientific research pertaining to DNA profiling and related analysis in civil cases like paternity disputes, immigration, and exchange of newborns in hospitals, for various agencies including private parties, on appropriate payment;
- ii. To provide DNA fingerprinting and related analysis and facilities to crime investigation agencies;
- iii. To assist police personnel, forensic scientists, lawyers and the judiciary in understanding the evidential value of the DNA profile analysis and related techniques in crime investigation and family matters;
- iv. To establish DNA diagnostic methods for detecting genetic disorders and to develop probes for such detection;
- v. To use DNA fingerprinting techniques for the authentication of plant and animal cell material, cell lines and to develop new probes where necessary for such purposes;
- vi. To provide training in DNA fingerprinting techniques;
- vii. To undertake basic, applied and developmental R & D work;
- viii. To provide consultancy services to medical institutions, public health agencies and industry in the country;
- ix. To collaborate with foreign research institutions and laboratories and other international organizations in fields relevant to the objectives of the Centre;
- x. To establish affiliation with recognized universities and institutions of higher learning for the purpose of enabling research scholars to register for post-graduate degrees;
- xi. To receive grants, donations and contributions in cash or in other forms from the Government of India, State Governments, Charitable Institutions/Trusts, individuals and industry within the country;
- xii. To receive, with the prior approval of the Central Government, monetary assistance from foreign sources including international organizations for training programmes, scientific research and other activities;
- xiii. To acquire by gift, purchase, exchange, lease, hire or otherwise howsoever, any property movable or immovable or to construct, improve, alter, demolish or repair buildings and structures as may be necessary or convenient for carrying on the activities of the Centre;
- xiv. For the purpose of the Centre, to draw and accept, make and endorse, discount and negotiate Government of India and other Promissory Notes, Bills of Exchange, Cheques or other Negotiable Instruments;

निदेशक का संदेश
From the Director's Desk

निदेशक का संदेश

यह बहुत गौरव पूर्ण अवसर है कि मैं वर्ष 2017-18 के लिए वार्षिक रिपोर्ट प्रस्तुत करता हूं, एक ऐसी अवधि जब सीडीएफडी उप्पल में अपने परिसर में स्थानांतरित हो गया। केंद्र की उपलब्धि को उजागर करने से पहले, मैं पद्म श्री डॉ. लालजी सिंह को श्रद्धांजलि अर्पित करना चाहूंगा जिनका निधन 10 दिसंबर 2017 को हो गया। सीडीएफडी की स्थापना का विचार डॉ लालजी सिंह द्वारा आरंभ किया गया था। वे न केवल सीडीएफडी के लिए बल्कि भारत में संपूर्ण वैज्ञानिक बंधुता के लिए प्रेरणा के स्रोत बने रहेंगे ।

सीडीएफडी अनुसंधान के एक अद्वितीय हाइब्रिड मॉडल के रूप में उभरा है जो न केवल आधुनिक जीवविज्ञान के सीमांत क्षेत्रों में अनुसंधान करके शैक्षिक उत्कृष्टता के लिए प्रयास करता है, बल्कि खास तौर पर डीएनए फिंगरप्रिंटिंग और अनुवांशिक विकारों के निदान के क्षेत्र में सामाजिक रूप से संगत कार्य के साथ भी बनाया गया है ।

पिछले वर्ष, सीडीएफडी ने 117 मामलों के लिए डीएनए प्रोफाइलिंग सेवाएं प्रदान कीं, जो न्यायपालिका और विभिन्न राज्य सरकारों की कानून लागू करने वाली एजेंसियों द्वारा अग्रेषित की गईं। यहां 4300 से अधिक रोगियों का मूल्यांकन किया गया है और सीडीएफडी द्वारा हैदराबाद के निजाम इंस्टीट्यूट ऑफ मेडिकल साइंसेज में स्थापित चिकित्सा अनुवांशिकी विभाग द्वारा विभिन्न अनुवांशिकी बीमारियों के लिए परामर्श दिया गया है। इस विभाग द्वारा चिकित्सा व अनुवांशिकी में डीएनबी कार्यक्रम भी सफलतापूर्वक चलाया जा रहा है। बासमती डीएनए विश्लेषण के लिए एपीडा-सीडीएफडी सेंटर ने पेटेंट बासमती 8 -एसएसआर मार्कर पैनल का उपयोग करके शुद्धता के लिए कुल 133 बासमती नमूनों का परीक्षण किया।



ट्रांसक्रिप्शन प्रयोगशाला में माइकोबैक्टेरियोफेज प्रोटीन पर शोध के लिए नई दिशा प्रदान की गई, जो माइकोबैक्टीरिया को मारने में सक्षम है और रो-निर्भर समाप्ति द्वारा एंटीबायोटिक संवेदनशीलता को नियंत्रित करने का तंत्र स्थापित किया गया है। बैक्टीरियल अनुवांशिकी प्रयोगशाला द्वारा आर लूप गठन और ई. कोलाई में एंटीसेन्स प्रतिलेखन के जीनोम-व्यापी संबंधों में नई अंतर्दृष्टि के साथ योगदान दिया गया।

सेल सिग्नलिंग प्रयोगशाला में दर्शाया गया है कि सह प्रोटीन सी-मिक के आईपी 7-मध्यस्थ पाइरोफॉस्फोराइलेशन में समय पर गिरावट सुनिश्चित करने की आवश्यकता है। इस समूह ने यह भी दर्शाया है कि आईपी 6 के एक नॉकआउट चूहों में, गोल शुक्राणुओं में क्रोमैटॉइड पिंडों की हानि मुख्य शुक्राणुजनित जीन के समयपूर्व अनुवाद और परिणामी शुक्राणुजनित विफलता के साथ सहसंबंध बनाता है। कोशिका मृत्यु और कोशिका उत्तरजीविता प्रयोगशाला में 143 मानव फॉस्फेट्स के मैण्ड इंटरैक्शन नेटवर्क हैं और कई फॉस्फेट्स को नई कोशिकीय प्रक्रियाओं से जोड़ दिया है जिसमें काइनेटोकोर असेंबली और कोशिकीय ग्लूकोज अपटेक शामिल हैं।

पादप-माइक्रोब अंतःक्रिया प्रयोगशाला द्वारा नवीन फेरिक लोहा बाध्यकारी प्रतिलेखन कारक की विशेषता ज्ञात की गई है जो लोहे के उपापचय और विषाणु के विनियमन के लिए आवश्यक है। कवक रोगाणुजनन प्रयोगशाला में दर्शाया गया है कि एसवायके (स्पिलीन टाइरोसिन काइनेस) के एस्पार्टिल प्रोटीएज़-मध्यस्थ संदमन - निर्भर आईएल - 1 बीटा उत्पादन सी ग्लैब्राटा के अंतःकोशिकीय अस्तित्व के लिए आवश्यक है।

ड्रोसोफिला तंत्रिका विकास प्रयोगशाला में दर्शाया गया है कि मूल हेलिक्स-लूप-हेलिक्स परिवार का एक प्रतिलेखन कारक ग्रेनी हेड, तंत्रिका स्टेम कोशिकाओं (एनएससी) में एपोप्टोसिस करता है, जिससे न्यूरोनल प्रजनन कोशिकाओं की आबादी को नियंत्रित किया जाता है। स्तनधारी आनुवंशिकी प्रयोगशाला में मेजबान मैक्रोफेज के संक्रमण के दौरान एपिजेनेटिक्स सर्किट्री के मांड्यूलेशन में एम. ट्यूबरकुलोसिस द्वारा एन्कोडेड एपिजेनेटिक प्रभावक अणुओं की भूमिका निर्भाई है।

कम्प्यूटेशनल और कार्यात्मक अनुवांशिकी प्रयोगशाला द्वारा माइक्रोबैक्टेरियम ट्यूबरकुलोसिस के विभिन्न फैड आर-जैसे प्रोटीन को प्रतिलेखन विनियमन और कोशिकीय शरीर क्रिया विज्ञान के रख-रखाव में उनकी कार्यात्मक भूमिकाओं को समझने के लिए चित्रित किया है। प्रतिरक्षा विज्ञान प्रयोगशाला में एनएफ-के बी के अवरोध और पी 53 के सक्रियण के माध्यम से ट्यूमर कोशिकाओं के विरुद्ध प्राकृतिक रूप से होने वाले टेट्रान या ट्राइटरपेनोइड (अज़ाडिराक्टिन) की भूमिका की पहचान की है। एम. ट्यूबरकुलोसिस के विरुद्ध नवीन हस्तक्षेप कार्यनीतियों को डिजाइन करने के लिए उनके अध्ययन के एक हिस्से के रूप में, आण्विक कोशिका जीवविज्ञान के प्रयोगशाला में फेगोसोमल परिपक्वता प्रक्रिया में एक नवीन प्रोटीन, रैब7एल-1 की भूमिका को समझ लिया है।

आण्विक ओन्कोलॉजी प्रयोगशाला ने गैर-हॉटस्पॉट उत्परिवर्ती पी53 के नवीन प्रतिलेखन लक्ष्यों की पहचान की है। इस समूह ने एक्स-पीएनपीईपी3 को कैन्ऑनिकल डब्ल्यूएनटी / बीटा-केटलिन सिग्नलिंग के नवीन प्रतिलेखन लक्ष्य के रूप में भी पहचाना है। कोशिका चक्र विनियमन की प्रयोगशाला में प्रोटीन किफ 2ए के स्पिंडल माइक्रोट्यूबुल स्थानीयकरण के विनियमन में एमएलएल परिसर की भूमिका की पहचान की है जो कि उचित गुणसूत्र पृथक्करण और कोशिका विभाजन के दौरान स्पिंडल गठन करता है। क्रोमैटिन बायोलॉजी और एपिजेनेटिक्स प्रयोगशाला में दिखाया गया है कि पोग्बे सिर्टुइन एचएसटी4 द्वारा डीएनए क्षति के उत्तर में यूबीक्रिटिन लाइगेज एससीएफ मध्यस्थ प्रोटीलाइसिस द्वारा निमंत्रित किया जाता है। जीनोमिक्स और प्रोफाइलिंग अनुप्रयोगों के प्रयोगशाला में नौ एसएनपी की खोज की गई जो विविध भारतीय आबादी के बीच मेलानिन सूचकांक भिन्नता के 31% को समझा सकता है।

पिछले वर्ष सीडीएफडी ने उच्च शोध सहकर्मी में अंतर्राष्ट्रीय शोध पत्रों की समीक्षा में अपना शोध परिणाम प्रकाशित किया है। हमारे संकाय को इस साल कई पुरस्कार और सम्मान प्राप्त हुए थे, जिनमें डॉ संगीता मुखोपाध्याय शामिल थीं, जिन्हें आईसीएमआर चतुर्वेदी घनश्याम दास जयगोपाल मेमोरियल अवॉर्ड - 2015 और डीबीटी की टाटा इनोवेशन फैलोशिप से सम्मानित किया गया था। डॉ. रूपिंदर कौर और डॉ सुभदीप चट्टर्जी को गुहा रिसर्च सम्मेलन के सदस्य के रूप में निर्वाचित किया गया, डॉ एम सुब्बा रेड्डी को वर्ष 2016 के लिए जीवविज्ञान में बीएम बिड़ला विज्ञान पुरस्कार से सम्मानित किया गया। डॉ रंजन सेन को भारतीय राष्ट्रीय विज्ञान अकादमी, भारतीय विज्ञान अकादमी और तेलंगाना एकेडमी ऑफ साइंसेज के अध्येता के रूप में निर्वाचित किया गया था। सीडीएफडी छात्रों को ईएमबीओ लघु अवधि अध्येतावृत्ति, डॉ जी पी तलवार युवा वैज्ञानिक पुरस्कार आदि सहित कई प्रतिष्ठित पुरस्कार,

एवं अध्येतावृत्तियां और यात्रा अनुदान भी प्राप्त हुए। इस अवधि के दौरान 15 छात्रों को पीएचडी डिग्री से सम्मानित किया गया।

अंत में, अपने सभी सहयोगियों की तरफ से, मैं इस अवसर पर जैव प्रौद्योगिकी विभाग, सीडीएफडी संस्थान के प्रतिष्ठित सदस्यों, शासी परिषद, अनुसंधान क्षेत्र पैनल - वैज्ञानिक सलाहकार समिति, प्रबंधन समिति, वित्त और भवन समितियों के प्रति निष्ठा, पूर्वक धन्यवाद प्रेषित करता हूं। उनके प्रोत्साहन, सलाह और अचूक समर्थन के बिना हमारी अधिकांश उपलब्धियां संभव नहीं होतीं।

देबाशीष मित्रा

31 मार्च, 2018

Director's Message

It is with great pride that I present the Annual Report for the year 2017-18, a period when CDFD shifted to its own campus at Uppal. Before I highlight the achievement of the centre, I would like to pay homage to Padma Shri Dr. Lalji Singh who passed away on the 10th December 2017. The idea of establishing CDFD was conceived by Dr. Lalji Singh. He will always remain as the source for inspiration not only for CDFD but also for the entire scientific fraternity in India.

CDFD has emerged as a unique Hybrid model of research that strives, not only for academic excellence, by undertaking research in frontier areas of modern biology, but is also knitted with socially relevant work, especially in the areas of DNA Fingerprinting and Diagnosis of genetic disorders.

In the past year, CDFD provided DNA profiling services for 117 cases, forwarded by the judiciary and law enforcing agencies of the Union and different State Governments. More than 4300 patients have been evaluated and counselled for various genetic diseases by the Medical Genetics Department established at Nizam's Institute of Medical Sciences, Hyderabad by CDFD. DNB Program in Medical Genetics is also being successfully run by this department. The APEDA-CDFD Centre for Basmati DNA Analysis tested a total of 133 Basmati samples for purity using our patented Basmati 8-SSR marker panel.

Laboratory of Transcription provided new direction, to the research on mycobacteriophage proteins that are capable of killing mycobacteria and established the mechanism of controlling antibiotic sensitivity by Rho-dependent termination. The Laboratory of Bacterial Genetics contributed with new insights into the genome-wide relationships of R-loop formation and antisense transcription in *E. coli*.

Laboratory of Cell Signalling has demonstrated that IP₇-mediated pyrophosphorylation of the oncoprotein c-Myc is required to ensure its timely degradation. This group has also shown that in *Ip6k1* knockout mice, the loss of chromatoid bodies in round spermatids correlates with the premature translation of key spermiogenic genes and consequent spermiogenesis failure. Laboratory of Cell Death & Cell Survival has mapped interaction network of 143 human phosphatases and have linked several phosphatases with new cellular processes including kinetochore assembly and cellular glucose uptake.



The laboratory of Plant-Microbe Interactions has identified and characterized a novel ferric iron binding transcription factor which is required for the regulation of iron metabolism and virulence. Laboratory of Fungal Pathogenesis has shown that aspartyl protease-mediated suppression of the Syk (spleen tyrosine kinase)-dependent IL-1 β production is essential for the intracellular survival of *C. glabrata*.

The Laboratory of Drosophila Neural Development has shown that Grainyhead, a transcription factor of basic helix-loop-helix family, brings about apoptosis of neural stem cells (NSC), thereby controlling the population of neuronal progenitor cells. Laboratory of Mammalian Genetics has dissected out the role of *M. tuberculosis* encoded epigenetic effector molecules in the modulation of the epigenetic circuitry during infection of the host macrophages.

Laboratory of Computational & Functional Genomics has characterized different FadR-like proteins of *Mycobacterium tuberculosis* to decipher their functional roles in transcription regulation and maintenance of cellular physiology. Laboratory of **Immunology** has identified the role of naturally occurring tetranortriterpenoid (Azadirachtin) against tumor cells through inhibition of NF- κ B and activation of p53. As a part of their study to design novel intervention strategies against *M. tuberculosis*, Laboratory of Molecular Cell Biology has deciphered a role of a novel protein, Rab711 in phagosomal maturation process.

Laboratory of Molecular Oncology has identified novel transcriptional targets of non-hotspot mutant p53. This group has also identified *XPNPEP3* as a novel transcriptional target of canonical Wnt/ β -catenin signalling. Laboratory of Cell Cycle Regulation has identified the role of

MLL complex in regulation of spindle microtubule localization of the protein Kif2A to proper chromosome segregation and spindle formation during mitosis. Laboratory of Chromatin Biology & Epigenetics has shown that ubiquitin ligase SCF mediated proteolysis in response to DNA damage is regulated by *S. Pombe* sirtuin Hst4. Laboratory of Genomics and Profiling Applications has discovered nine SNPs that could explain 31% of the Melanin Index variation amongst the diverse Indian population.

In the past year CDFD has published its research outcome in high profile peer reviewed international journals. Our faculties were recipients of several awards and honours this year, including Dr. Sangita Mukhopadhyay, who was awarded the ICMR Chaturvedi Ghanshyam Das Jaigopal Memorial Award – 2015 and the TATA Innovation Fellowship of the DBT. Dr Rupinder Kaur and Dr. Subhadeep Cahtterjee were elected as members of the Guha Research Conference, Dr. M Subba Reddy was awarded the B.M. Birla Science Prize in Biology for the

year-2016, Dr. Ranjan Sen was elected as a Fellow of Indian National Science Academy, Indian Academy of Sciences and the Telangana Academy of Sciences. CDFD students also received several prestigious awards, fellowships and travel grants including the EMBO short-term fellowship, Dr. G.P. Talwar Young Scientist Award, etc. 15 students were awarded Ph.D Degrees during this period.

Finally, on behalf of all my colleagues, I take this opportunity and extend our sincere thanks to the Department of Biotechnology, distinguished members of the CDFD Society, Governing Council, Research Area Panels-Scientific Advisory Committee, Management Committee, Finance and Building Committees for their encouragement, advice and unstinted support without which much of our achievements would not have been possible.

Debashis Mitra

March 31, 2018

सेवाएँ
Services

LABORATORY OF DNA FINGERPRINTING SERVICES

Scientist In-charge	Madhusudan Reddy Nandineni	Staff Scientist
Other members	SPR Prasad	Senior Technical Officer
	Devinder Singh Negi	Technical Officer
	Sanjukta Mukerjee	Technical Officer
	Pooja Tripathi	Technical Officer
	Kiranmai Joshi	Technical Officer (till Feb. 2018)
	Vijay Amrutarao Girnar	Technical Assistant
	Shruti Dasgupta	Technical Assistant
Co-ordinator	D P Kasbekar	Haldane Chair (till July 2017)
	Sanjeev Khosla	Staff Scientist (since July 2017)

Objectives:

- 1) To provide DNA fingerprinting services in cases forwarded by law-enforcing agencies / judiciary of State and Federal Governments, relating to murder, sexual assault, paternity, maternity, child swapping, deceased identification, organ transplantation, etc.;
- 2) To develop human resources skilled in DNA fingerprinting, to cater to the needs of State and Federal Government agencies;
- 3) To impart periodical training to manpower involved in DNA fingerprinting sponsored by State and Federal Government agencies;
- 4) To provide advisory services to State and Federal Government agencies in establishing DNA Fingerprinting facility;
- 5) To create DNA marker databases of different populations of India.

Karnataka (10), Andhra Pradesh (8), Puducherry (5), Delhi (5), Bihar (3), Maharashtra (3), Jammu & Kashmir (2), Uttar Pradesh (2), Andaman & Nicobar Islands (1), Madhya Pradesh (1), Tripura (1) and West Bengal (1).

Details of services provided in the current reporting year (1st April 2017 to 31st March 2018):

Breakup of the cases during this reporting period is given below under following heads:

Biological Relationship	27
Identity of Deceased	43
Paternity/Maternity	42
Sexual Assault (Rape)	05

Total number of cases	117
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Summary of services provided until the beginning of the reporting year (1st April 2016 to 31st March 2017):

A total of 143 cases were received for DNA fingerprinting examination during the reporting period 2016 – 2017. Of these, 38 cases were related to identification of deceased, 12 cases pertained to sexual assault (rape), 70 cases were related to paternity / maternity, 2 cases pertained to murder and 21 cases pertained to biological relationship (organ transplantation). Fifteen States and two Union Territories of India had availed DNA fingerprinting services from CDFD during this period. Chhattisgarh forwarded the highest number of cases (41) followed by Punjab (22), Goa (14), Telangana (12), Tamil Nadu (12),

Prominent cases during April 1, 2017 to March 31, 2018

1. Five cases from National Investigation Agency (NIA) involving national security and public safety.
2. Two cases forwarded by the Anti-Terrorism Squad, Maharashtra involving national security and public safety.
3. Two cases from Armed Forces Medical College, Pune pertaining to the identification of Indian Air Force personnel in IAF helicopter crash at Itanagar and Sukhoi fighter jet crash at Tezpur.

Deposition of evidence in Courts of Law

During this reporting year, the DNA experts defended their reports in **21 cases** in various Honorable Courts of Law throughout the country.

Training/Lectures/Workshops: 2017 - 2018

1. Training was provided to the officer from Maldives Police Service, Maldives on mtDNA profiling from 16.07.2017 to 04.08.2017 at LDFS, CDFD.
2. Lecture was delivered for the benefit of students of Dept. of Biotechnology, Periyar Maniammai University Vallam, Tanjavur, Tamil Nadu at CDFD on 28.09.2017
3. Lecture was delivered for the benefit of students of Daulatram College, Delhi University at CDFD on 26.10.2017
4. Lecture was delivered for the benefit of students of Department of Genetics and Plant Breeding, College of Agriculture, Rajendranagar, Hyderabad on 01.12.2017.
5. Lecture was delivered for the benefit of students of Apeejay School, Nerul, Mumbai

at CDFD on 21.12.2017

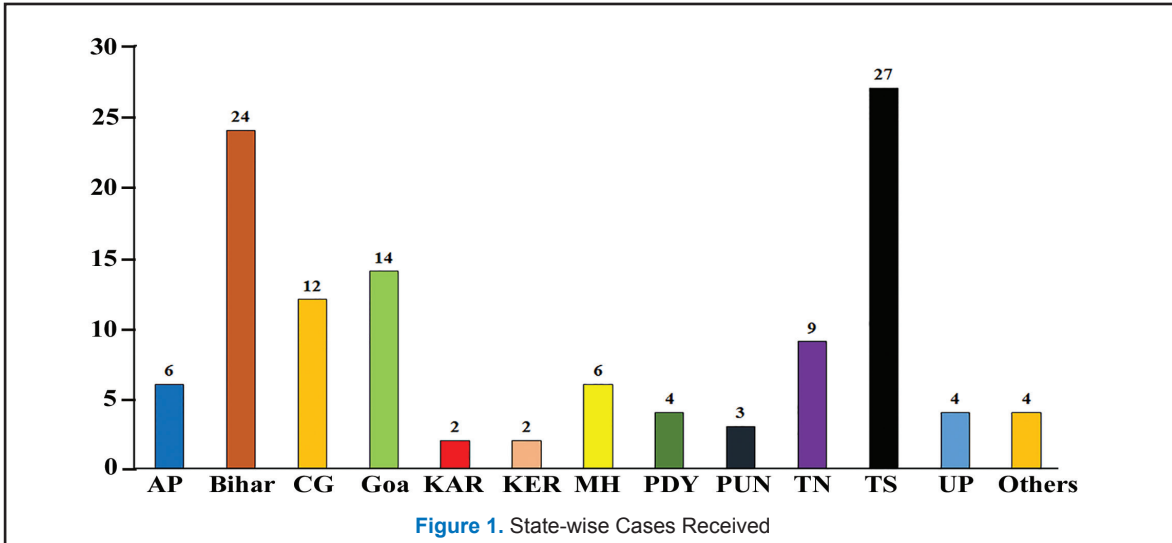
6. Lecture was delivered for the benefit of students of Biotechnology, Guwahati University on 09.01.2018
7. Lecture was delivered for the benefit of students of Department of Life Sciences, Jain University, Bangalore at CDFD on 29.01.2018
8. Lecture was delivered for the benefit of students of Statistics, Savitribai Phule University, Pune on 01.02.2018.
9. Invited talk by Dr. N. Madhusudan Reddy, during the 39th Annual Conference of the Indian Academy of Forensic Medicine (IAFM) at Puducherry on 03.02.2018.
10. Invited talk by Dr. N. Madhusudan Reddy, on the theme of "Science for Human Welfare" as part of National Science Day Celebrations organized by the Telangana Academy of Sciences, in association with Jignasa Science Forum, Jogulamba Gadwal District, Telangana State on 06.03.2018.

Summary of the State-wise breakup of DNA Fingerprinting cases:

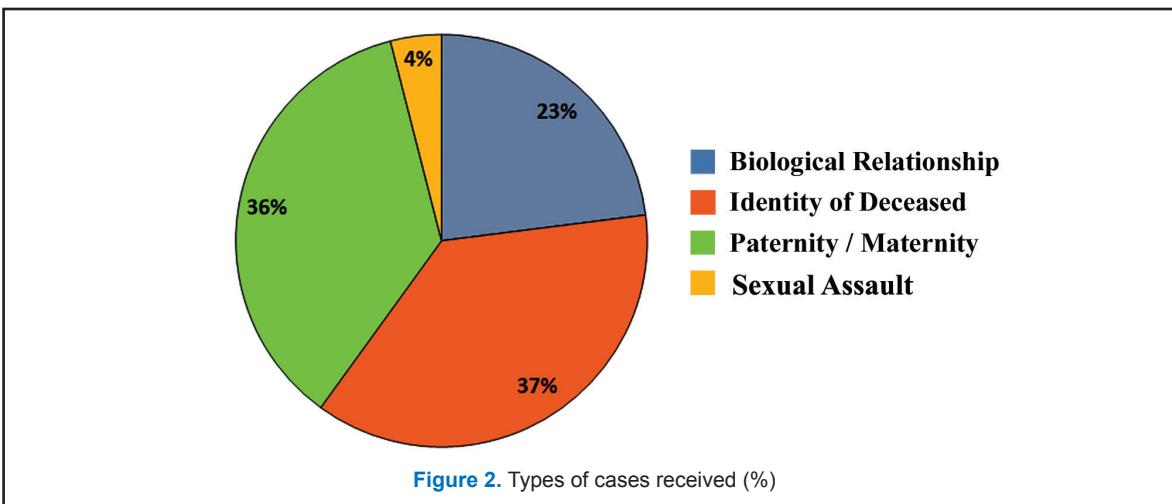
Name of the State	Biological Relationship	Identity of Deceased	Maternity / Paternity	Sexual Assault (Rape)	No. of Cases
Andhra Pradesh		1	5		6
Assam		1			1
Bihar		16	5	3	24
Chhattisgarh			12		12
Delhi		1			1
Goa		6	8		14
Karnataka			2		2
Kerala		1		1	2
Maharashtra		5	1		6
Puducherry		3	1		4
Punjab		2		1	3
Tamil Nadu	9				9
Telangana	18	2	7		27
Uttar Pradesh		3	1		4
Uttarakhand		1			1
West Bengal		1			1
Total No. of Cases.	27	43	42	5	117

A total of 117 cases were received for DNA fingerprinting examination during the current reporting period (2017 – 2018). Of these cases, 42 cases were related to maternity/paternity, 43 cases were related to identity of deceased, 27 cases were related to biological relationship and 5 cases were related to sexual assault. 14 States and two Union Territories of India have availed

DNA fingerprinting services from CDFD during this period. Telangana forwarded the highest number of cases (27) followed by Bihar (24), Goa (14), Chhattisgarh (12), Tamil Nadu (9), Andhra Pradesh (6), Maharashtra (6), Puducherry (4), Uttar Pradesh (4), Punjab (3), Karnataka (2), Kerala (2), Assam (1), Delhi (1), Uttarakhand (1) and West Bengal (1) (Fig.1).



The cases involving deceased identity (37%), maternity/paternity (36%) and biological relationship (23 %) constituted the bulk of the cases received (Fig.2).



Revenues generated:

During this reporting period, an amount of ₹ 36,25,215/- (Rupees thirty six lakhs twenty five thousand two hundred and fifteen only) has been

received towards DNA fingerprinting analysis charges, which is inclusive of GST (18% at present) as levied by the Govt. of India.

DIAGNOSTICS DIVISION

Faculty	Ashwin Dalal	Staff Scientist
Adjunct Faculty	Prajnya Ranganath Shagun Aggarwal Dhanya Lakshmi N	Associate Professor, NIMS Associate Professor, NIMS Assistant Professor, NIMS
PhD Students	Anjana Kar Dipti Deshpande A Sandeep Arijita Mitra	Senior Research Fellow Senior Research Fellow Junior Research Fellow (since July 2017) Junior Research Fellow (since July 2017)
Other Members	Aneek Das Bhowmik Maria Celestina Vanaja L Samyuktha Vineeth VS Amrita Bhattacharjee Ramya Padmaja T P Divya M Chitra Sravani Laxmi Priyanka P. Rajitha Angalena R Usha Rani Dutta M Muthulakshmi A SobhanBabu Jamal Md Nurul Jain Vasantha Rani C. Krishna Prasad R. Sudheer Kumar	Research Associate Research Associate (till October 2017) Research Associate (till February 2018) Research Associate Research Associate SIAMG Fellow (till February 2018) SIAMG Fellow (till February 2018) Project Assistant Project Assistant (till January 2018) Project Assistant Project Assistant (since January 2018) Technical Officer Senior Technical Officer Technical Officer Technical Officer Technical Officer Technical Officer Technical Officer Technician Technician

Objectives

1. To conduct genetic evaluation for patients/families with genetic disorders
2. To develop new methods and assays for genetic analysis and engage in research on chromosomal and single gene disorders
3. To act as national referral center for analysis and quality control of genetic tests for few genetic diseases
4. To impart training in genetic evaluation of patients with genetic disorders

I. Services provided and Training programs during the year 2017-2018

Clinical Genetics

A total of 4332 patient samples were analyzed for genetic testing, during the year 2017-18. These consisted of patients with chromosomal

disorders, monogenic disorders, mental retardation, congenital malformations, inborn errors of metabolism, and other familial disorders.

The Department of Medical Genetics established at Nizam's Institute of Medical Sciences, Hyderabad is functioning successfully. A total of 3207 patients were examined and counseled in the unit during 2017-18. In addition antenatal ultrasonograms were done in 547 cases, antenatal invasive procedures (chorionic villus sampling and amniocentesis) in 281 cases and foetal autopsies were conducted in 134 fetuses. A 3 year training program for Diplomate of National Board (DNB) in Medical Genetics initiated with affiliation to National Board of Examinations, New Delhi is running successfully; three batches of students (total 5 students) have joined so far and the fourth batch is due to join in May 2018.

Genetic investigations done during 2017-18

Investigation	Total cases	Positives
Cytogenetics	1305	109 (8.4%)
Proband	1116	104 (9.3%)
Prenatal	189	5 (2.6%)
Molecular Genetics	2504	710 (28.4 %)
Proband	2364	679 (28.7 %)
Prenatal	140	31 (22.1 %)
Biochemical Genetics	523	177 (33.8%)
Proband	498	171 (34.3%)
Prenatal	25	6 (24%)

Cytogenetics

Disease	Abnormality	No of cases
Down Syndrome	47,XY,+21	26
	47,XX,+21	17
	46,XX,rob(21;21) +21	1
	46,XY,rob(21;21) +21	1
	46,XX,rob(14;21) +21	2
	47,XY,+marker	2
	47,XY,+21,inv(9)	1
	47,SC,+21,21s+	1
	47,SC,+21	1
Turner syndrome	Monosomy X (45,X)	4
	mos 45,X/ 46,XY	2
	mos 45,X/46,X,i(X)	3
	mos 45,X/46,X,+marker	1
	Mos 45,X/46,X,r(X)	1
	mos 45X/47,XXX	1
Klinefelter Syndrome	47,XXY	1
	mos47,XXY/46,XY	1
	48,XXYY	1
Sex reversal	46,XX	1
	46,XY	2

Quantitative Fluorescent PCR (QF-PCR)

MLPA/QF PCR	Patients	Positives
Prenatal (Aneuploidy)	66	4
Postnatal (Microdeletion)	152	24

Disease/translocation	Probe	No of tests
Di-George Syndrome	TUPLE(22q11.2)/ARSA(22q13)	6
Marker chromosome	WCP-12, WCP-1, 11, 13,9, 3 SE(X)/(Y), Acro-p-arm	10
Spectral karyotyping		4

Structural chromosomal abnormalities	
Inversions	
46,X,inv(Y)	2
46,XY,inv(9)	1
46,XX,inv(9)	3
46,XY,inv(9)(p12q13),15ps+, 15ps+	1
Deletions	
46,XX,del(11)(q23)	1
47,SC,+Xp-	1
Duplications	
46,XX,7q	1
Translocations	
46,XX,t(3;12)(q27;q13)	1
46,XX,t(2;14)(p25;q23)	1
46,XX,t(17;22)(q22;p11.2)	1
46,XY,t(1;10)	1
46,XX,der(15;22)(q10;q10)+21	1
45,XX,rob(15;22)(q10;q10)	1
46,XY,t(4;8)(q31.1;q24.2),inv(9))	1
46,SC,rob(14;21)(q10;q10)+21	1
Polymorphic variants	21

Biochemical Genetics

Disease/Test	Positives
Urine & Blood Metabolic Screening tests (N=144)	34
Amino acid disorders (N=90)	36
Non Ketotic Hyperglycinemia	8
Hyperornithinemia	2
Phenylketonuria	3
MSUD	2
Increased plasma Glutamic acid	8
Other amino acid disorders	7
Hyperhomocysteinemia	6

Disease/Test	Positive
Lysosomal storage disorders (N=264)	101
Hurler syndrome	5
Hunter syndrome	8
Sanfilippo B	3
Morquio A disease	32
Arylsulphatase B	2
GM1-Gangliosidosis	14
Gaucher disease	9
Krabbe disease	1
Pompe disease	1
Nieman Pick disease	3
Mucopolidosis	7
Metachromatic Leukodystrophy	7
Fabry disease	4
Hexosaminidase A/B	
Tay Sachs disease	3
Sandhoff disease	1
Multiple sulfatase	1

Prenatal diagnosis (25)	
	6
Pompe's disease	1
Krabbe's disease	0
Metachromatic Leukodystrophy	1
Gaucher's disease	1
Hurler syndrome	1
Hunter	0
Morquio A disease	2
Arylsulphatase B	0
Niemann Pick disease	0
Hexosaminidase A/B	0

Molecular genetics

Name of Disorders	No of Cases	Positive	Negative		
DMD/BMD	226	153	73		
DMD Carrier Analysis	46	18	28		
Spinal Muscular Atrophy	123	44	79		
SMA Carrier Analysis	78	27	51		
Hemophilia	12	2	10		
		Normal	Homozygous	Heterozygous	Compound Heterozygous
β thalassemia and Sickle cell anemia	233	20	147	9	57
β thalassemia carriers	89	5	-	84	-
Factor V Leiden	275	261	-	14	-
Factor II mutation	158	158	-	-	-
Cystic Fibrosis	138	125	5	8	-
Pancreatitis	43	31	-	12	-
Connexin 26	18	13	5	-	-
Achondroplasia	10	5	-	5	-
Alpha thalassemia	31	23	3-Triplication 5-het deletion		
Gilbert Syndrome	81	7	65	9	-
Maternal contamination	412	0	0		
Apert Syndrome	5	1	-	4	-
MTHFR	7	2	-	5	-

Cpd Heterozygous= Compound Heterozygous, NA- Not applicable

Molecular genetics—prenatal diagnosis

Name of the Disorders	No of Cases	Positive	Negative		
DMD	7	0	7	-	-
Spinal Muscular atrophy	24	7	17	-	-
Myotonic dystrophy	1	1	0		
Fragile X Syndrome	1	-	1		
Hemophilia	-	-	-		
		Normal	Homozygous	Heterozygous	Compound Heterozygous
β thalassemia	91	23	11	48	9
Connexin	2	1	1		
Cystic Fibrosis	13	10	2	1	
Achondroplasia	1	-	-	1	

II. Diagnostics Research

Project 1: Human exome sequencing for identification of novel genes in rare mendelian disorders

Summary of work done until the beginning of this reporting year (April 1, 2016 – March 31, 2017)

Single gene disorders are rare health conditions that affect a small number of people as compared to other diseases in population. But collectively they account for important cause of morbidity and mortality. To date ~ 7000 distinct rare diseases have been documented and new rare diseases are being reported regularly. The classical methods of gene identification include chromosomal mapping, linkage analysis and homozygosity mapping. Although these methods are persuasive, there are certain limitations, which have been overcome by new sequencing technology: Massively parallel sequencing or Next generation sequencing. Next generation sequencing has made it possible to identify candidate gene using just a few affected individuals or parent child trio.

The identification of candidate gene for single gene disorders has importance, not only in prenatal diagnosis and genetic counseling of affected families, but also in basic research towards understanding of gene function and pathophysiology of disease. Over the past years of providing services in clinical genetics, we have identified several interesting novel disorders and syndromes with a single gene pattern of Mendelian inheritance. We have employed exome sequencing to identify novel genes in such families.

Details of work done in the current reporting year (April 1, 2017 – March 31, 2018)

We have studied a family (Family 1) wherein two siblings were similarly affected with a progressive neurodevelopmental disorder with microcephaly seizures and spastic quadriplegia and born out of consanguineous marriage. Whole exome sequencing in both the siblings identified a novel homozygous nonsense variant

NM_006303.3:c.105C>A, present in *AIMP2* gene, creating a stop codon at 35th position in exon 1 (p.Y35X). Sanger sequencing confirmed the presence of variant in the patients and carrier status in parents (Fig. 1A-D). Absence of the variant was also confirmed in 2 different normal control samples (by Sanger sequencing) and our in-house database. Pathogenicity testing using online tools predicted the variant as likely disease causing (CADD PHRED score- 34). Using online tool GeneMatcher (<https://genematcher.org/>) a second Indian family (Family 2) with single proband has been ascertained with similar phenotype and having the same variant (by different research group, Fig. 1E-H). A joint collaboration with the group and homozygosity mapping analysis was performed with the exome data of the 3 affected individuals which showed that the variant in *AIMP2* is located in a common shared ROH (regions of homozygosity) of 940 Kb observed in chromosome 7. It is likely that the variant was inherited from a common ancestor. We have also performed reverse transcriptase quantitative PCR (RT-qPCR) analysis using the patient cDNA which revealed reduced level of *AIMP2* mRNA as compared to controls although the difference was not statistically significant (P-values for younger and elder sibling were 0.1694 and 0.1240, respectively). This suggests low level of NMD of *AIMP2* mRNA in peripheral leukocytes (Fig. 1I). *AIMP2* gene encodes an auxiliary protein p38 of human multi-ARS (Aminoacyl-tRNA synthetase) complex. *AIMP2*/p38 is a key component of this complex and is crucial to maintain the stability of the complex. Several neurological disorders including neurodegeneration, microcephaly, ID, leukodystrophy, seizures, spasticity, cerebral and cerebellar atrophy have been recognized with defects in various components of mammalian tRNA synthetase complex and their associated proteins (Fig. 1J). Hence, this deleterious mutation (p.Y35X) which likely results in a truncated protein, likely to undergo nonsense mediated decay, could be the cause of the phenotype observed in these patients. This is the first report of a disease causing variant in *AIMP2* gene (MIM:600859).

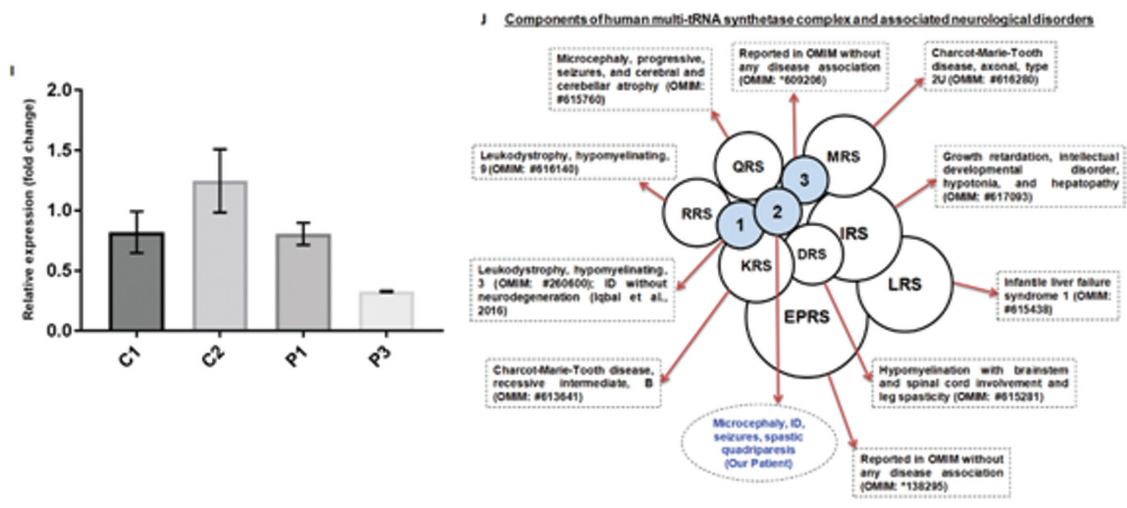
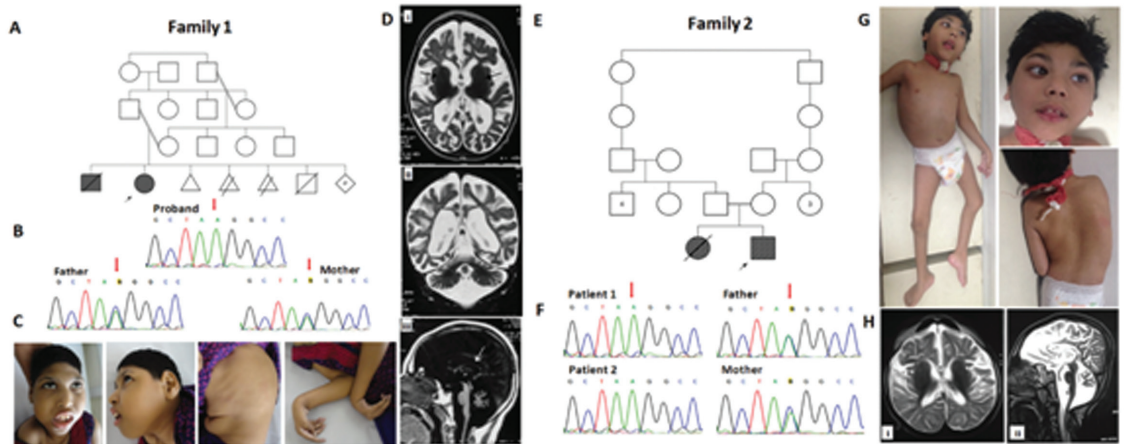


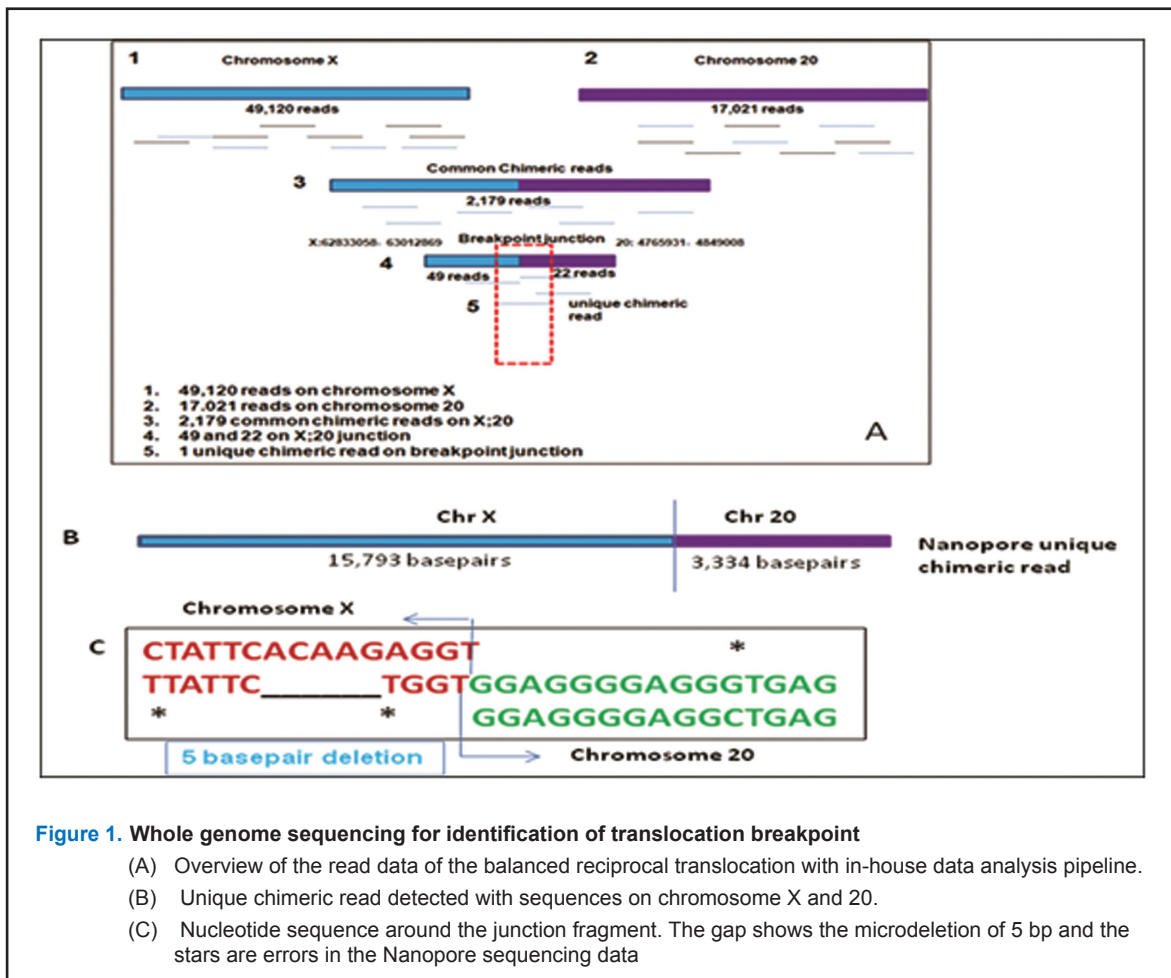
Figure 1.

- (A) Pedigree of family 1.
- (B) Sanger validation of the *AIMP2* variant in family 1. The pathogenic variation c.105C>A of *AIMP2* is found in homozygous state in proband and is heterozygous in her parents.
- (C) Proband (P1) at 7 years of age shows microcephaly, kyphoscoliosis and contractures at wrist joints.
- (D) T2 weighted magnetic resonance imaging of brain on postnatal day 10 revealed significant cerebral atrophy, cerebellar atrophy, prominent cisterna magna, hypo-intensities in the bilateral basal ganglia
- (E) Pedigree of family 2.
- (F) Sanger validation of the *AIMP2* variant in family 2. The variation c.105C>A of *AIMP2* is found in homozygous state in probands and is heterozygous in their parents.
- (G) P3 at age 6 years shows microcephaly, contractures at knees and ankles and kypho-scoliosis.
- (H) T2 weighted magnetic resonance imaging of brain done at age 4 years shows cerebral atrophy, cerebellar atrophy, hypo-intensities in the bilateral basal ganglia
- (I) Reverse transcription quantitative PCR (RT-qPCR) analysis showing the expression of *AIMP2* mRNA (\pm SD) in the probands (P1 & P3) and two unrelated controls (C1 & C2). Comparative $\Delta\Delta$ Ct method was used to calculate gene expression, and *HERC1* was used as reference gene.
- (J) The components of the multi-tRNA synthetase complex and associated neurological disorders. Three AIMPs (1, 2, and 3) are multiply linked to most of the enzyme components. RRS- Arginyl-tRNA synthetase, QRS- Glutaminyl-tRNA synthetase, MRS- Methionyl-tRNA synthetase, IRS- Isoleucyl-tRNA synthetase, DRS- Aspartyl-tRNA synthetase, KRS- Lysyl-tRNA synthetase, EPRS- Glutamyl-prolyl-tRNA synthetase, LRS- Leucyl-tRNA synthetase.

Project II: Whole Genome Sequencing for characterization of novel genes and de novo balanced chromosomal rearrangements in human genetic disorders (This is a new activity)

Single gene disorders and balanced chromosomal rearrangements (BCRs) associated with diseases are important cause of human genetic diseases leading to morbidity and mortality. Over the past twenty years, we, along with other clinical geneticist colleagues have identified many interesting and novel genetic disorders and syndromes with a single gene pattern of Mendelian inheritance. Handigodu syndrome, a rare and painful osteoarthritic disorder was identified in few districts of South India still having

unidentified cause of disease although a genetic etiology has been suspected in previous studies. De novo balanced chromosomal rearrangements in patients with disease phenotype is a unique opportunity to identify gene responsible for the condition by characterizing the breakpoint. However existing genetic tools like targeted gene sequencing, array comparative genomic hybridization, and exome sequencing cannot detect all types of genetic variations in a single test. Whole genome sequencing can characterize all types of genetic variants in all parts of the genome (Figure 2). Such completeness can lead to the identification of pathogenic variants and hence influence diagnosis, genetic counselling and treatment.



Details of work done in the current reporting year (April 1, 2017 – March 31, 2018)

Next generation sequencing based whole genome sequencing is now becoming a powerful tool for characterizing and mapping translocations

to base pair resolution level. However the bioinformatics analysis and interpretation is still challenging. We have done whole genome sequencing using 1D long read sequencing by Oxford Nanopore sequencing technology

(Minlon R9.4, Oxford Nanopore) for a patient with developmental delay, seizures who was also found to have a de-novo balanced translocation between chromosome X and 20. Nanopore sequencing provides unbiased data as there is no PCR amplification during library preparation and the longest reads amongst technologies currently available. The 1D libraries yielded total of 1,332,335 reads with 1,104,065 quality passed reads and an average read length of 9 kb. About 79.65% of the reads mapped to hg19 reference genome. Of these, 18,135(2.70%) reads mapped to chromosome 20 and 44,900 (6.68%) reads mapped to Chromosome X, for an average depth of about 1.46X. The standard pipeline using BWA -MEM was used to align the data to reference genome. Shell script was used to extract the chimeric reads from the SAM file by using the sub chromosomal coordinates as reference sequence and we identified 2,179 chimeric reads mapping to chromosome X and 20. Out of these, 49 and 22 unique reads mapped to Xq11.1 and 20p13 region targeting the sub chromosomal location. These unique reads were visualized in Integrative Genomic Browser (IGV) which showed one chimeric breakpoint anchoring read of 18 kb (Fig. 2A). This single read showed approximately, 15,793 bp, homology to chromosome X, and 3,334 bp homology to chromosome 20 (Fig. 3B). On alignment of chimeric sequence to the reference genome, the Xq11.1 breakpoint was found to disrupt the second intron of the ARHGEF9 gene (62,976,819) and the 20p13 region breakpoint was found to be between the RASSF2 and

SLC23A2 genes (4,816,380). Further the breakpoint junction on X showed a microdeletion of 5 base pairs whereas the chromosome 20 region showed no deletion except the presence of several repeats (Fig. 3C). We have previously done Fluorescent In Situ Hybridization based mapping of this breakpoint. After 2 years of laborious experiments, we could narrow down the breakpoint to somewhere between exon 1 and 2 of ARHGEF. On the other hand the same breakpoint was mapped to base pair level in just a month using the whole genome sequencing. This illustrates the power of NGS based sequencing in identification of various types of mutations/rearrangements in human genome.

Project III: Clinical, biochemical and molecular analysis of lysosomal storage disorders

Summary of work done until the beginning of this reporting year (April 1, 2016 – March 31, 2017)

Lysosomal storage disorders are a heterogeneous group of disorders associated with specific lysosomal enzyme deficiency. The diagnosis in most of these disorders is based on enzyme assay. There is a large amount of overlap in enzyme levels among carriers and normal people; hence it is very difficult to detect carriers by enzyme assay. Mutation detection is helpful for carrier detection and accurate prenatal diagnosis. Our study aims to characterize the clinical features, biochemical parameters and molecular defects in various lysosomal storage disorders.

Table I: Data sheet showing mutation analysis for LSDs over last eight years

Lysosomal Storage Disorder	Gene	Number of cases	Total mutations	Novel mutations
Niemann-Pick disease types A & B	SMPD1	138	81	46
Niemann- Pick disease type C	NPC1	14	8	3
Niemann- Pick disease type C	NPC2	1	1	1
Metachromatic leukodystrophy	ARSA	88	61	25
Mucopolysaccharidosis I	IDUA	31	22	15
Mucopolysaccharidosis II	IDS	33	20	7
Mucopolysaccharidosis VI	ARSB	38	24	18
Sialidosis	NEU1	5	3	3
Mucopolysaccharidosis II/II	GNPTAB	59	37	24
Total		407	257	142

Details of work done in the current reporting year (April 1, 2017– March 31, 2018)

Over last eight years we have been able to identify mutations in more than 400 patients with different lysosomal storage diseases (LSDs) (Table 1). This was done as part of a National Task Force on Lysosomal Storage Diseases funded by Indian Council of Medical Research and Department of Health Research. This study has revealed the mutation spectrum in patients with LSDs in the Indian population.

Publications

Research papers published in 2017:

1. Alber M, Kalscheuer VM, Marco E, Sherr E, Lesca G, Till M, Gradek G, Wiesener A, Korenke C, Mercier S, Becker F, Yamamoto T, Scherer SW, Marshall CR, Walker S, Dutta UR, Dalal AB, Suckow V, Jamali P, Kahrizi K, Najmabadi H, Minassian BA (2017). ARHGEF9 disease: Phenotype clarification and genotype-phenotype correlation. **Neurology Genetics** 26;3(3):e148.
2. Harms FL, Girisha KM, Hardigan AA, Kortüm F, Shukla A, Alawi M, Dalal A, Brady L, Tarnopolsky M, Bird LM, Ceulemans S, Bebin M, Bowling KM, Hiatt SM, Lose EJ, Primiano M, Chung WK, Juusola J, Akdemir ZC, Bainbridge M, Charng WL, Drummond-Borg M, Eldomery MK, El-Hattab AW, Saleh MA, Bézieau S, Cogné B, Isidor B, Küry S, Lupski JR, Myers RM, Cooper GM, Kutsche K. (2017). Mutations in EBF3 Disturb Transcriptional Profiles and Cause Intellectual Disability, Ataxia, and Facial Dysmorphism. **American Journal of Human Genetics** 100(1):117-127.
3. Uttarilli A, Pasumarthi D, Ranganath P, Dalal AB (2017). Functional characterization of arylsulfatase B mutations in Indian patients with Maroteaux-Lamy syndrome (mucopolysaccharidosis type VI). **Gene** 599:19-27.
4. Das Bhowmik A, Gupta N, Dalal A, Kabra M (2017). Whole exome sequencing identifies a homozygous nonsense variation in ALMS1 gene in a patient with syndromic obesity. **Obesity Research in Clinical Practice** 11(2):241-246.
5. Francis F, Bhat V, Balachander B, Khare C, Bethou A, Dalal A, Ponnala R (2017). Look Up to Diagnose Down! **Indian Journal of Pediatrics** 84(12):961-962.
6. Tallapaka KB, Ranganath P, Dalal A (2017). Variable Expressivity and Response to Bisphosphonate Therapy in a Family with Osteoporosis Pseudoglioma Syndrome. **Indian Pediatrics** 54(8):681-683.

Publications (until 31st March 2018):

1. Nagarajan K, Swamiappan E, Anbazhagan S, Dalal A, Adithan S, Krings T (2018). "Twig-like" cerebral vessels are not pathognomonic for ACTA A2 mutations: A case report. **Interventional Neuroradiology**: 1591019918765239.
2. Gaucher Disease Task Force, Puri RD, Kapoor S, Kishnani PS, Dalal A, Gupta N, Muranjan M, Phadke SR, Sachdeva A, Verma IC, Mistry PK (2018). Diagnosis and Management of Gaucher Disease in India - Consensus Guidelines of the Gaucher Disease Task Force of the Society for Indian Academy of Medical Genetics and the Indian Academy of Pediatrics. **Indian Pediatrics** 55(2):143-153.
2. Shukla A, Das Bhowmik A, Hebbar M, Rajagopal KV, Girisha KM, Gupta N, Dalal A (2018). Homozygosity for a nonsense variant in AIMP2 is associated with a progressive neurodevelopmental disorder with microcephaly, seizures, and spastic quadriparesis. **Journal of Human Genetics** 63(1):19-25.
3. Aggarwal S, Tandon A, Bhowmik AD, Dalal A (2018). Autopsy findings in EPG5-related Vici syndrome with antenatal onset: Additional report of Focal cortical microdysgenesis in a second trimester fetus. **American Journal of Medical Genetics** 176(2):499-501.
5. Kar A, Phadke SR, Das Bhowmik A, Dalal A (2018). Whole exome sequencing reveals a mutation in ARMC9 as a cause of mental retardation, ptosis, and polydactyly. **American Journal of Medical Genetics** 176(1):34-40.
6. Das Bhowmik A, Salem Ramakumaran V, Dalal A (2018). Tarsal-carpal coalition syndrome: Report of a novel missense mutation in NOG gene and phenotypic delineation. **American Journal of Medical Genetics** 176(1):219-224.
7. Narayanan DL, Deshpande D, Das Bhowmik A, Varma Dr, Dalal A (2018). Familial choreoathetosis due to novel heterozygous mutation in PDE10A. **American Journal of**

Medical Genetics 176(1):146-150.

8. Aggarwal S, Tandon A, Das Bhowmik A, Safarulla JM, Dalal A (2018) A Dysmorphology Based Systematic Approach Toward Perinatal Genetic Diagnosis in a Fetal Autopsy Series. **Fetal and Pediatric Pathology** 37(1):49-68.

Research papers in press (as on 31st March 2018):

1. Aggarwal S, Das Bhowmik A, Tandon A, Dalal A. Exome sequencing reveals blended phenotype of double heterozygous FBN1 and FBN2 variants in a fetus. **European Journal of Medical Genetics**.
2. Tallapaka K, Venugopal V, Dalal A, Aggarwal S. Novel RSPO1 mutation causing 46,XX testicular disorder of sex development with palmoplantar keratoderma: A review of literature and expansion of clinical phenotype. **American Journal of Medical Genetics**.
3. Das Bhowmik A, Patil SJ, Deshpande DV, Bhat V, Dalal A. Novel splice-site variant of UCHL1 in an Indian family with autosomal recessive spastic paraplegia-79. **Journal of Human Genetics**.
4. Patil SJ, Das Bhowmik A, Bhat V, Satidevi Vineeth V, Vasudevamurthy R, Dalal A.

Autosomal recessive otofaciocervical syndrome type 2 with novel homozygous small insertion in PAX1 gene. **American Journal of Medical Genetics**.

5. Godbole KG, Ramachandran A, Karkamkar AS, Dalal AB (2018). Compound Heterozygosity for Hb Alpertion (HBB: c.407C>T) and IVS-I-5 (G>C) (HBB: c.92+5G>C) Mutations Presenting as a Moderate Anemia in an Indian Family. **Hemoglobin**.

Other publications like patents, Book chapters, etc.(01.04.2017 to 31.03.2018)

1. Usha R. Dutta, Ashish Bahal, V.S. Vineeth, Vasantha Sarvade, Prajnya Ranganath, Ashwin Dalal (2017). A novel mosaic complex supernumerary marker chromosome in a girl with seizures: systematic characterization of the complex marker. **Gene Reports** 8:128–133.
2. *Aggarwal S (2017). Counseling for Fetal Central Nervous System Defects. **Journal of Fetal Medicine** 4(2): 65–73.
3. *Aggarwal S (2017). Fetal Dysmorphology: An Indispensable Tool for Synthesis of Perinatal Diagnosis. **Genetic clinics** 10(2):11-19.

*Work done elsewhere

PLANT DNA FINGERPRINTING SERVICES

Chairperson	Dr. Subhadeep Chatterjee	Staff Scientist
Members	Dr. K. Anupama	Staff Scientist
	Lakshmi Vaishna	Technical Assistant
	Chandrashekar	Technician
	G. Shivaram	Office supporting staff

Objectives

1. Testing the purity of Basmati samples received from Export Inspection Council (EIC), Ministry of Commerce, Government of India, Basmati rice exporters from India, and other countries; and
2. To assess the genetic purity of rice hybrids and cytoplasmic male sterile lines used in rice hybrid seed production.

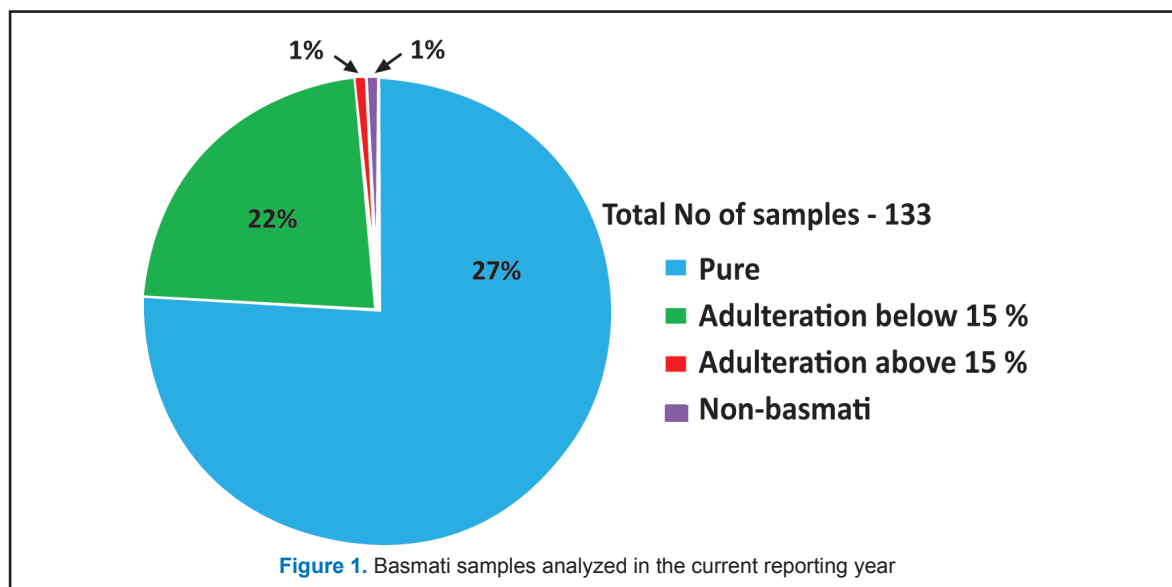
Summary of the work done until the beginning of this reporting year (upto March 31, 2017)

A total of 153 Basmati samples obtained from EIC were analyzed. For those samples that were complex in nature (more than two Basmati and adulterant varieties), single grain analysis

India, Basmati rice exporters from India and other countries.

During the current reporting year, a total of 133 samples were analyzed and the number of samples indicating the percentage of adulteration with non-basmati rice is shown in Figure 1.

The protocol for DNA testing of Basmati rice was initially developed for detection of adulteration using eight Simple Sequence Repeats (SSRs) marker assay with eleven notified Basmati varieties. In view of the complexities and challenges arising in adulteration testing, efforts are being made to further expand as well as fine tune the present protocol as mentioned below:



was followed for identification of rice varieties. Developed three co-dominant markers that differentiates CMS and maintainer lines of rice.

Details of progress made in the current reporting year (April 1, 2017 - March 31, 2018)

Objective 1: Testing the purity of Basmati samples received from Export Inspection Council (EIC), Ministry of Commerce, Govt. of

i) Updating the database of Basmati varieties.

At present thirty varieties of Basmati rice have been notified by Department of Agriculture and Cooperation (DAC) under Seeds Act, 1966. The profiles of all the new notified Basmati varieties using the eight marker panel were generated to develop a comprehensive database that can be used while analyzing the test samples.

ii) Generate new panels of markers for accurate detection of adulteration and varietal identification.

With increase in the number of notified Basmati varieties, the profiles of few of the Basmati varieties are overlapping with the adulterant profiles. In addition, some of the new notified varieties have the same marker profiles. Therefore, it is important to identify new panel of markers that will enable accurate detection of adulteration and clear varietal identification. So

and alkali spreading value or Gelatinization temperature is available. Currently, genotyping all the Basmati varieties and adulterants for InDel marker and SNPs is in progress.

Objective 2: To assess the genetic purity of rice hybrids and cytoplasmic male sterile lines used in rice hybrid seed production.

Rice hybrids are produced by three-line system that includes A-line (Cytoplasmic Male-sterile line), B-line (Maintainer line), and R-line (Restorer line). According to Indian seed act, purity of

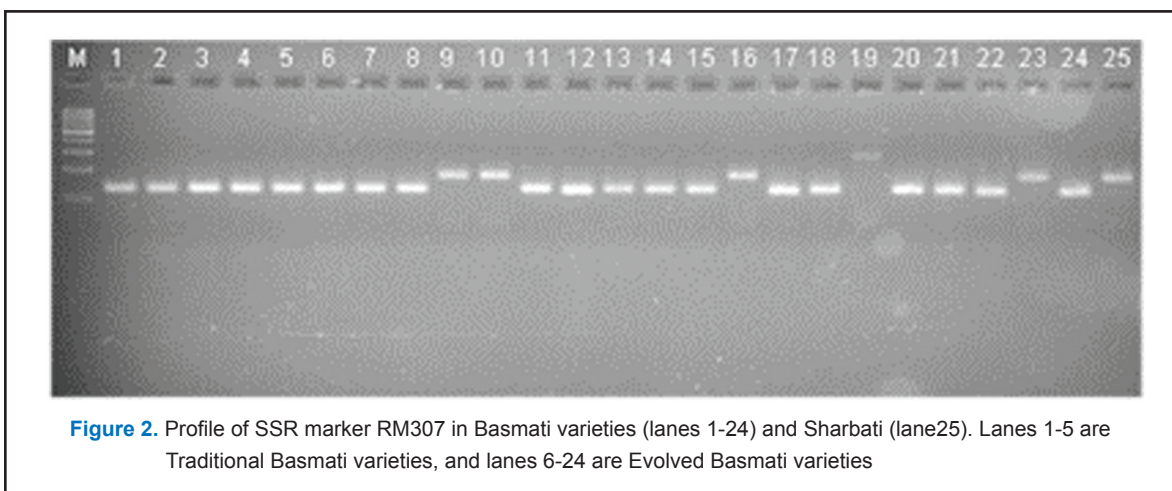


Figure 2. Profile of SSR marker RM307 in Basmati varieties (lanes 1-24) and Sharbati (lane 25). Lanes 1-5 are Traditional Basmati varieties, and lanes 6-24 are Evolved Basmati varieties

far, 35 SSR markers that are reported to have high polymorphic information content (PIC) are screened on Basmati and non-Basmati (Sharbati) varieties. Nine markers are monomorphic across all tested varieties and nineteen SSR markers are monomorphic in Traditional Basmati varieties and exhibited polymorphism in Evolved Basmati varieties (Figure 2). None of the markers presented a unique allele in Sharbati. These results suggest that a very high number of markers have to be tested to develop a new panel.

To circumvent the problems associated with ambiguous scoring of bands in agarose gels, in future experiments PCR products will be fluorescently labeled with Tetramethyl-Rhodamine dUTP and will be electrophoresed by capillary electrophoresis in ABI3730 Genotyper and allele sizes scored with Genemapper software version 4.

Development of a marker panel based on SNPs/Indels in genes conferring unique features to Basmati rice may help in clear differentiation of Basmati from non-basmati varieties. An eight base pair deletion in *badh2* gene is known to be responsible for fragrance. Information on SNPs in genes determining grain length, amylose content

hybrid rice should be of 98% and that of the cytoplasmic male sterile line should be of 99%. It is estimated that even 1% impurity in hybrid seed reduces the yield by 100Kg/hectare. A-line and B-line are iso-nuclear lines and differ only in the cytoplasmic DNA content, particularly in the mitochondrial DNA. Three co-dominant markers that differentiate CMS and maintainer lines were developed using reported mitochondrial DNA sequence of A-line and B-line. The objective of this work is to develop an assay system to find the admixture of B-line in A-line on bulked seed lot as an alternative to testing on individual seeds.

The forward primer of markers were tagged with fluorescent dye and PCR was performed on the genomic DNA isolated from A and B-lines and the PCR product was run by capillary electrophoresis using ABI3730. Along with A-line specific band, B-line specific band was also present in the A-line and the composition of B-line specific fragment was around 3% of the A-line specific fragment. This is because around 60% of mitochondrial DNA is present in the nuclear genome, thus making it difficult to estimate the B-line admixture in A-line accurately. To overcome this problem, such polymorphic regions should be selected

where B-line specific regions are not present on chromosome or the polymorphic region (both A-line and B-line specific region) is completely absent on the chromosome. However, such regions are not available. On the other hand,

A-line specific sequence of the polymorphic mitochondrial genome is present on the chromosome. The primer of this marker will be tagged with fluorescent dye and studied for its suitability in the development of the assay.

शोध
Research

LABORATORY OF BACTERIAL GENETICS

Studies on gene regulation, transcription termination, (p)ppGpp metabolism and amino acid and ion-transport in *Escherichia coli*

Faculty	Abhijit A Sardesai R Harinarayanan	Staff Scientist Staff Scientist
INSA Senior Scientist	J Gowrishankar	
Ph D Students	Rajvardhan M Kapshikar Suchitra Upreti Nalini Raghunathan Rajeshree Sanyal Nida Ali Ravish Sharma J Mallikarjun Sayantan Goswami Swati Dubey Vani Singh Neeraj Kumar Yogesh Patidar Vishweshwar	Senior Research Fellow Senior Research Fellow Senior Research Fellow Senior Research Fellow Senior Research Fellow Senior Research Fellow Junior Research Fellow Junior Research Fellow Junior Research Fellow Junior Research Fellow Junior Research Fellow Junior Research Fellow Junior Research Fellow (since July 2017)
Other Members	J Krishna Leela T S Shaffiqu Vimala Allada P Hima Bindu	Technical Officer Technical Officer Research Associate Research Associate (till Oct. 2017)

The Laboratory of Bacterial Genetics comprises three research groups engaged in investigations on several aspects of the physiology and genetics of *Escherichia coli*, and is majorly supported by the Department of Biotechnology as a Centre of Excellence for Microbial Biology. The work undertaken in this reporting year is described below under the following objectives:

Objectives

1. Occurrence of pathological R-loops and their consequences;
2. Essentiality and oligomerization features of RNase E;
3. The PtsP-PtsO-PtsN phosphorelay and potassium (K⁺) metabolism;
4. Studies on basic amino acid export;
5. Understanding the role of (p)ppGpp in the growth rate dependent modulation of cell division;
6. Genetic and molecular characterization of the glycerol induced growth stasis in the *glpD* mutant;

7. Studies on the role of SpoT and GppA in the amplification of stringent response;

Summary of work done until the beginning of this reporting year (upto March 31, 2017)

The work undertaken in earlier years on each of the objectives has been summarized in the first parts of the corresponding description below.

Details of progress made in the current reporting year (April 1, 2017 – March 31, 2018)

Occurrence of pathological R-loops and their consequences

Transcription-translation coupling is one of the fundamental attributes of the bacterial way of life. Over the past several years our laboratory has been working on the hypothesis that when bacterial transcription is uncoupled from translation, the nascent transcript is prone to re-annealing with the upstream DNA to generate RNA-DNA hybrids (R-loops) that are toxic; and that an important function for the Rho and NusG proteins in *E. coli* – which target nascent RNAs that are not being simultaneously translated for transcription termination – is to prevent such

R-loop formation. Transcription-associated R-loops in the genome are believed to confer toxicity, in both prokaryotes and eukaryotes, by impeding the progression of replication forks as well as by serving as sites for aberrant initiation of DNA replication; in bacteria, the latter is also referred to as constitutive stable DNA replication (cSDR).

The evidence obtained so far in support of the hypothesis have included the following: (i) Complete knock-out of Rho or NusG function in wild-type *E.coli* is lethal, and this lethality can be overcome by ectopic expression of a phage T4-derived R-loop helicase UvsW. (ii) The genome-wide sites of R-loop occurrence have been mapped, and we have shown that the prevalence of R-loops is increased when Rho or NusG function is compromised. (iii) Since antisense transcripts are not translated, their synthesis is also terminated by action of Rho and NusG, and we had reported last year that in Rho-deficient strains antisense RNA from genomic regions form R-loops that are revealed in RNA-Seq experiments upon concomitant expression of UvsW.

In the current year, we had completed the analysis of the RNA-Seq data to determine the genome-wide relationship between R-loop formation and antisense transcription in *E. coli*. The evidence suggests that in Rho-deficient cells, R-loop formation blocks subsequent rounds of antisense transcription at more than 500 chromosomal loci. Hence these antisense transcripts, which can extend beyond 10 kb in their length, are only detected when Rho function is absent or compromised and the UvsW helicase is concurrently expressed. Thus the potential for antisense transcription in bacteria is much greater than hitherto recognized; and the cells are able to retain viability even when nearly one-quarter of their total non-rRNA abundance is accounted for by antisense transcripts, provided that R-loop formation from them is curtailed. We have also shown that *E. coli* mutants deficient for RNase H (which degrades RNA in R-loops) display heightened sensitivity to the Rho inhibitor bicyclomycin, once again supporting the notion that Rho inhibition is associated with increased occurrence of R-loops.

Since, as mentioned above, R-loops can provoke aberrant DNA replication called as cSDR, we have been investigating the mechanisms of cSDR in *E. coli*. The genetic hallmark of cSDR is its ability to confer viability to mutants that are defective for DnaA-mediated replication

initiation at *oriC* (eg, to *dnaA* deletion strains). In work completed this year, we have identified a novel cSDR mechanism in *dam* mutants of *E. coli* but which is not R-loop mediated, as briefly described below.

The Dam methylase in *E. coli* catalyzes adenine methylation at palindromic GATC sites in DNA, by which it participates in MutHLS-directed mismatch repair, DNA replication initiation from *oriC*, sister chromatid cohesion, and regulation of gene expression. *dam* mutants suffer increased double strand breaks (DSBs) through aberrant mismatch processing, and such DSBs can therefore be prevented by additional mutation in the *mutH/L/S* genes.

We have found that the *dam* mutant is viable even in absence of DnaA and therefore can perform cSDR (in the presence of two additional mutations that are permissive for non-*oriC*-initiated replication forks to traverse the full circular chromosome); this cSDR was abolished by the *mutH/L/S* mutations, implying that DSBs are necessary for cSDR. However, DSBs alone did not appear to be sufficient for cSDR, since the radiomimetic agent phleomycin (at sublethal concentrations) did not rescue the *dnaA* deletion lethality.

This led us to postulate that Dam deficiency has a second effect in promoting cSDR (in addition to generating DSBs), and indeed this was shown to be the case since the *dam mutH/L/S* mutants (that do not suffer DSBs) now exhibited cSDR when exposed to phleomycin. We suggest that this second role brought about Dam methylation of GATC sites may be ensure that replication forks that are assembled during DSB repair progress in the appropriate direction, and that their abnormal retrograde progression in *dam* mutants is responsible for cSDR.

As an offshoot from our work on Rho-dependent transcription termination, we have also been studying variants of the nucleoid proteins H-NS (which is known to silence gene transcription) since we had shown earlier that certain dominant-negative H-NS mutants can alleviate the effects of Rho deficiency. In the present studies, we have purified wild-type H-NS and the dominant-negative variants (L26P, $\Delta 64$, $\Delta 93$, I119T) and used them in in vitro-transcription assays. Although our experiments were able to demonstrate, for the first time, dominant-negativity of the H-NS mutants in vitro, their modulatory effect on Rho-mediated termination was not observed, suggesting that

perhaps additional factors may be required for this effect. Furthermore, an unexpected observation was that the $\Delta 64$ variant, but not the $\Delta 93$ variant, directly and completely inhibited RNA polymerase in vitro, and we suggest that a hitherto unidentified auto-inhibitory domain may exist in H-NS between amino acid residues 64 and 93 whose physiological relevance remains to be determined.

Essentiality and oligomerization features of RNase E

RNase E is an endoribonuclease in *E. coli* which is essential for viability. It is the major determinant of mRNA stability and is also involved in processing and maturation of ribosomal RNAs and transfer RNAs. The enzyme, which acts preferentially on RNA substrates with a 5'-monophosphate end, is a homotetramer of a 1061-amino acid long polypeptide that can be said to be comprised of a catalytic N-terminal half (NTH, residues 1-530) and a non-catalytic C-terminal half (CTH, residues 531-1061). The CTH is dispensable for viability (although it does appear to aid in substrate recruitment), and it represents an intrinsically unstructured region which forms the scaffold for assembly of a multi-protein complex called the degradosome. The crystal structure of the NTH tetramer has been solved both as of itself and in complex with RNA, from which it can be deduced that 5'-end sensing of the RNA molecule is carried out in the "sensor pocket" of one subunit (which includes residues R169 and T170) while the endonucleolytic scission is performed in the active site of the adjacent subunit (which includes residues D303 and D346).

In work reported last year, we had provided genetic evidence for inter-subunit complementation in the NTH of RNase E. Thus, NTH polypeptides bearing either an R169Q mutation (abolishing 5'-end sensing) or a D303A or D346A mutations (abolishing endonucleolytic activity) were individually lethal, but when co-expressed were viable. The model is that the mutant subunits can assemble as hetero-oligomers such that RNA 5'-end sensing and cleavage can be done in a "cross-over" manner between the two mutant subunits. In the present year, we have been attempting to re-constitute the inter-subunit complementation phenotype in vitro, for which we have so far overexpressed and purified the N-terminally His-tagged polypeptides and have standardized the endonuclease assay for RNase E using a 5'-radiolabelled RNA oligonucleotide substrate. Further experiments towards the

reconstitution are in progress.

In last year's report, we had also provided preliminary evidence, based on over expression toxicity of RNase E polypeptides with or without the CTH, that the CTH region confers toxicity and had speculated that this intrinsically unstructured segment undergoes toxic aggregation in the bacterial cytoplasm, akin perhaps to that described for amyloidogenic or prionogenic proteins in eukaryotic cells. In the current year, we have confirmed that overexpression of the isolated CTH polypeptide region indeed is lethal in *E. coli*, but that this lethality is alleviated in mutants bearing Δ CTH mutation of RNase E on the chromosome. These results suggest that the toxicity is consequent to loss of RNase E activity in cells following aggregation of all the CTH regions, and we are in the process of undertaking further experiments to test this notion.

The PtsP-PtsO-PtsN phosphorelay and potassium (K^+) metabolism

In *E. coli* and other bacteria uptake of some sugars occurs via specific uptake systems referred to as phosphotransferase systems (PTS). Typically, the PTS comprises a multiprotein phosphorelay that co-opts a phosphate moiety from phosphoenolpyruvate (PEP) that is transferred onto the incoming sugar. *E. coli* possesses a PTS comprising PtsP, PtsO and PtsN, with PEP-dependent phosphorelay operating in the same sequence. This PTS is thought to be paralogous to the classical glucose PTS. However, unlike the glucose PTS neither any membranous components of the PtsP-PtsO-PtsN phosphorelay, nor the phospho-acceptor substrate of PtsN are known.

We have earlier delineated a physiological link between the PtsP-PtsO-PtsN phosphorelay and cellular K^+ (potassium) ion metabolism. Our studies implicate the involvement of dephospho-PtsN as a negative regulator of the YcgO K^+ efflux transporter. This is based on the observation that a Δ ptsN mutant of a strain bearing all K^+ uptake systems was growth inhibited in media of high external K^+ concentrations ($[K^+]_{es}$), a phenotype referred to as the K^S . Furthermore, the K^S was paradoxically associated with measurable K^+ limitation that was alleviated by overexpression of a K^+ uptake protein such as Kup. A similar K^S was also elicited in the wild type strain upon overexpression of YcgO that also was associated with K^+ limitation and was suppressed by over expression of Kup. Additional studies implicated the absence of dephospho-PtsN as being causal to K^+ limitation of the Δ ptsN mutant. The

aforementioned observations are consistent with the notion that dephospho-PtsN is a negative regulator of the YcgO K⁺ efflux protein.

Recently we noted the involvement of dephospho-PtsN as a stimulator of the Trk K⁺ uptake system comprising TrkA, TrkH, SapD and SapF proteins. Accordingly, the $\Delta ptsN$ derivative of a strain bearing Trk as the only K⁺ uptake system displayed K⁺ limited growth in low (1 mM) and high (115 mM) [K⁺]_e (that is K^S) containing media. Absence of YcgO suppressed the K^S but did not affect the K⁺ limited growth in low [K⁺]_e medium. Chromosomal lesions that led to overproduction of the K⁺ uptake protein Kup or of the ATP binding subunits of the Trk system, namely SapD and SapF, alleviated the dual K⁺ limitation of the aforementioned strain. The effect of overproduced SapD/F required a functional TrkA that constitutes the regulatory gating ring of the TrkH K⁺ transporter and a functional TrkH. Levels of SapD/F or of TrkA were not impaired in the $\Delta ptsN$ mutant and chromosomal expression exclusively of dephospho-PtsN did not lead to K⁺ limited growth both at low and high [K⁺]_e. Our studies are supportive of the notion that dephospho-PtsN may function as a regulator of ATP binding by TrkA, the gating ring of the TrkH K⁺ transporter, and stimulate the K⁺ uptake through the Trk system. The stimulatory effect of dephospho-PtsN on K⁺ uptake via the Trk and Kdp (identified by another laboratory) transporters on the one hand and its inhibitory effect on K⁺ efflux via YcgO on the other is indicative of a central role for dephospho-PtsN in co-ordination of transmembrane K⁺ flux under certain conditions. Current studies are directed towards testing the notion that dephospho-PtsN may interact with TrkA, and this is being tested by two-hybrid analyses and co-purification studies.

Recently we have obtained evidence that the YajC SecD SecF triumvirate that mediates protein secretion in *E. coli* is also involved in K⁺ metabolism in *E. coli* on the basis that genetic lesions that disable their activity suppress the K^S of the $\Delta ptsN$ mutant. Additional genetic studies indicate that impaired YajC-SecD-SecF activity confers upon a cell an as yet unidentified means to cope with K⁺ limitation. This finding provides a basis to explain alleviation of the K^S of the $\Delta ptsN$ mutant by lesions that perturb YajC-SecD-SecF activity. Lastly, we have noted that perturbations in SecD/F activity additionally appear to impair the functioning of two K⁺ uptake systems Kdp and TrkG/H presumably by affecting their membrane biogenesis. The aforementioned findings are being pursued further.

Studies on basic amino acid export

In this component of research we have reported genetic and physiological studies on the L-arginine (Arg) and L-lysine (Lys) exporters ArgO and LysO. Furthermore, we have constructed a detailed membrane topology map of the L-arginine exporter ArgO employing the substituted cysteine accessibility method and alkaline phosphatase fusion analyses and have proposed a model for the disposition of its trans-membrane helices in the cytoplasmic membrane of *E. coli*. Lastly we have studied the genetic basis of resistance of *E. coli* to the L-arginylalanine (Arg-Ala) dipeptide and have noted that to a large extent the resistance is correlated with the presence of a wild type copy of *ydhE* whose product encodes an inner membrane protein belonging to the multidrug and toxin extrusion (MATE) family. The contribution of ArgO in mediating resistance to Arg-Ala is less. Our genetic and physiological studies on this phenomenon are consistent with a scenario in which the presence of the Arg-Ala dipeptide in the medium causes an as yet unknown physiological defect(s) that may not be related to excessive levels of Arg in the cytoplasm and that ArgO and YdhE mitigate the defect(s) presumably by exporting Arg-Ala. Furthermore, it is speculated that Arg-Ala may serve as a proxy for an as yet unknown, naturally occurring substrate for YdhE (and ArgO), probably an antimicrobial compound.

Towards obtaining structure function relationships of LysO, we have initiated studies to delineate the membrane topology of LysO. In this regard we have employed compartment specific reporter fusion technology to probe the topology of LysO. We have constructed a set of LysO-PhoA protein hybrids in which LysO protein segments of differing lengths, originating from the N-terminus of LysO are fused to PhoA (alkaline phosphatase) lacking its signal sequence. Analyses of these LysO-PhoA hybrids has indicated that the C-terminus of LysO is located in the periplasm and the topology of LysO is consistent with the notion that LysO may bear a transmembrane domain comprising eight transmembrane segments. Currently we are engaged in increasing the resolution of LysO topology using the substituted cysteine accessibility method.

Understanding the role of (p)ppGpp in the growth rate dependent modulation of cell division

Previous work from this laboratory has shown that basal (p)ppGpp contributes to the regulation of cell division by positively regulating the

level of FtsZ, the structural protein involved in septum formation. This regulation, which is not essential for the maintenance of cell division under normal growth conditions, is required for septum formation in absence of the Lon protease. The latter synthetic phenotype arises consequent to increased activity of the SulA protein which is an inhibitor of FtsZ function and is normally degraded by the Lon protease. In a related study, it was observed that null mutation in the (p)ppGpp synthase gene *relA* conferred synthetic growth defect in the presence of the hypomorphic *ftsZ84* allele. Based on these phenotypes, a genetic study was initiated to decipher the role of (p)ppGpp in the modulation of cell division by identifying genetic suppressors of the *relA ftsZ84* growth defect by transposon mediated mutagenesis or by using a plasmid over-expression library.

In work undertaken in the reporting period we obtained genetic suppressors by transposon mutagenesis, knock out of candidate genes and as well as gene over-expression using a multi-copy plasmid library and mapped them. We showed that each of the suppressor mutation and the over-expression suppressors obtained was able to suppress the *ftsZ84*, *relA ftsZ84* and the *relA lon* growth defect suggesting that there was a common molecular basis for the growth defect observed in the different genetic backgrounds. The data obtained from this study suggested the existence of cross-talk between the processes of cell division and fatty acid/phospholipid synthesis. Specifically, the data supported a model, wherein a decrease in the cell-division capacity in the cell could be compensated by the reduced rate of fatty acid biosynthesis.

Genetic and molecular characterization of the glycerol induced growth stasis in the *glpD* mutant

It has been reported that the addition of glycerol or glycerol-3-P induced growth arrest in the *glpD* mutant of *E. coli* with a concomitant decrease in the levels of nucleotides; the molecular basis of this effect remains unclear. Based on our results we had proposed that the growth stasis induced by glycerol is caused by the inhibition of PRPP synthesis and the consequent decrease in the nucleotide pool. We also proposed that the inhibition of PRS (PRPP synthase) activity could be due to the depletion of ATP and the accumulation of ADP from the unfettered GlpK activity. The same cannot be said for the glycerol-3-P induced stasis, as the decrease in PRPP pool is concomitant with that of the nucleotides unlike

in the case of glycerol where PRPP depletion is seen before that of the nucleotides.

In this reporting period we have tested if the depletion of the nucleotides is causal to the growth inhibition conferred by glycerol/glycerol-3-P in the *glpD* mutant. In order to do this, we asked if elevating the intracellular concentration of the nucleotides, particularly that of the purines, whose concentration decreased, could support growth of the *glpD* mutant in the presence of glycerol or glycerol-3-P. It has been reported that the intracellular concentration of the purine nucleotides can be elevated in the presence of the *gsk3* mutation by supplementing the growth medium with guanosine. *gsk3* is a gain of function mutation that renders the guanosine kinase protein resistant to feedback inhibition by GTP, leading to the de-repressed synthesis of GMP and leading to increase in the ATP and GTP levels. In the *gsk3* mutant, following guanosine supplementation, along with an increase in the purine nucleotide pool, there is growth arrest from depletion of the UTP pool, an increase in the ppGpp pool and starvation for histidine and tryptophan (Petersen C, 1999, *JBiolChem*, 274: 5348-5356). Since *prs-1* was identified as a genetic suppressor of the guanosine induced growth defect, it was proposed that these above mentioned changes arose from the reduction in the PRPP pool, following the inhibition of the Prs activity (Petersen C, 1999). Based on these findings, in order to increase the cellular purine nucleotide concentration without compromising the growth, we constructed the *gsk3 deoD udp* strain and cultured it following supplementation of guanosine, uridine, histidine and tryptophan to the growth medium.

To ask if the increase in purine nucleotide pool alleviated the glycerol/glycerol-3-P induced stasis in the *glpD* mutant, we constructed the *glpD gsk3 deoD udp* quadruple mutant and studied its growth in the presence of glycerol/glycerol-3-P. We observed that the glycerol-3-P induced stasis was significantly alleviated but the glycerol induced stasis was unaffected. Interestingly, a rapid depletion in the UTP pool (despite the presence of uridine in the growth medium) was observed following glycerol supplementation but not glycerol-3-P suggesting that the depletion of the UTP pool could be responsible for the growth arrest. These results show, in the *glpD* mutant background, the effect of glycerol on the cellular nucleotide pool varies as the intracellular pool of the nucleotides are altered, that is, UTP level that is not significantly perturbed in an otherwise wild type background decreases rapidly when

the purine nucleotide pools are elevated. The molecular basis of this effect is being examined.

Studies on the role of SpoT and GppA in the amplification of stringent response

We had noted an unusual pattern in the accumulation of the stringent nucleotides, (p)ppGpp, following the depletion of SpoT or when the stringent response is provoked by amino acid starvation following SpoT depletion or in the hydrolase defective SpoT mutants or in the complete absence of SpoT function. Under each of these conditions, there was accumulation of ppGpp but not pppGpp. When we sought to increase the pppGpp pool under these conditions by the inactivation of the pppGpp hydrolase GppA, we observed synthetic growth inhibition associated with RelA-dependent synthesis of (p)ppGpp. We also observed that, despite the absence of SpoT hydrolase activity, following the reversal of amino acid starvation, growth resumed after a lag and was associated with the degradation of ppGpp.

Our study has revealed, for the first time, the potential of the stringent nucleotide pppGpp, to activate the RelA-dependent stringent response in the cell in the absence of amino acid starvation. This activation is evident under two conditions as the basal pppGpp pool is elevated through the inactivation of GppA. One, when there is wild type RelA activity and the SpoT hydrolase activity is reduced, and two, when there is reduced RelA activity in the complete absence of SpoT functions (both synthase and hydrolase). It is unclear if the pppGpp driven activation of stringent response could be physiological, that is, contribute to the stringent response under some conditions in *E.*

coli. However, given that the stringent response is conserved across bacteria and that the basal level of (p)ppGpp and the capacity for synthesis and hydrolysis of (p)ppGpp varies between bacteria and within a bacterium depending on the environmental signal, it is conceivable that RelA mediated synthesis of (p)ppGpp could be activated by pppGpp under some physiological states.

We identified the *mutT* and *nudG* genes to be multi-copy suppressors that rescued the growth defect of the $\Delta spoT$ and $\Delta spoT \Delta gppA$ strains. MutT is a nudix hydrolases whose primary cellular function is to prevent the misincorporation of the mutagenic nucleotide 8-oxo-dGTP in the DNA. However, *in vitro*, it was found to hydrolyze all 8 nucleotides. Our results show that MutT is capable of hydrolyzing the stringent nucleotides *in vivo*. NudG is also a member of the Nudix hydrolase family that shows high specificity for hydrolysis of pyrimidine (deoxy)nucleoside triphosphates. Its preferred substrate appears to be 5-hydroxy-CTP. Our results show that NudG is capable of hydrolyzing the stringent nucleotides *in vivo*. Interestingly, both these proteins, in the absence of their over-expression supported the accumulation of pppGpp during stringent response in the $\Delta spoT$ strain.

Publications in 2017

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Publications in 2018 (till March 31, 2018)

1. Raghunathan, N., R. M. Kapshikar, J. K. Leela, J. Mallikarjun, P. Bouloc, and J. Gowrishankar. 2018. Genome-wide relationship between R-loop formation and antisense transcription in *Escherichia coli*. ***Nucleic Acids Research*** (in press).

LABORATORY OF CELL CYCLE REGULATION

Elucidating the role of chromatin modifying proteins in cell cycle regulation

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Objectives

1. Study of non-canonical roles of H3K4 HMTs in cell cycle regulation.
2. Role of H3K4 HMTs in regulation of repetitive non-coding regions.

Project 1: Study of non-canonical roles of H3K4 HMTs in cell cycle regulation.

Activities of Histone 3 lysine 4 histone methyltransferase (H3K4 HMTs) are linked to active gene expression, but their precise role in cell cycle regulation is now being uncovered. In this project, we are looking at 'other' roles of H3K4 HMT by studying one of the members of this chromatin-modifying complex –MLL– that influence cell cycle regulation.

Summary of work done until the beginning of this reporting year (up to March 31, 2017)

We showed that loss of MLL/WDR5 complex delays progression through mitosis and compromises chromosome alignment as well as spindle formation in mitosis.

Details of the progress made in the current reporting year (April 1, 2017 –March 31, 2018)

We previously observed prolonged pro-metaphase in MLL- and WDR5-depleted cells which indicates a defect in chromosome congression and/ or in attachment of chromosomes to mitotic-spindle microtubules (MTs). In order to identify the proteins interacting with MLL/WDR5 complex, we performed tandem affinity purification from HeLa cells stably

expressing triple-epitope (S-protein, Flag and streptavidin-binding peptide; SFB)-tagged WDR5 using streptavidin-agarose beads and S-protein-agarose beads followed by mass spectrometry analysis. Our mass spectrometry studies identified kinesin 2A, 5A and dynein heavy chain as the putative binding partners of WDR5. Particularly, kinesin 13 family protein, Kif2A was highly enriched in our SFB-WDR5 pull-down. However, as all three members of this family—Kif2A, Kif2B, and Kif2C—are involved in mitosis, particularly spindle assembly and chromosome congression, we decided to perform interaction study with all three. Similarly, we identified Kif5A—one of three members of Kinesin-1 family— as interacting partner. However, as Kif5A is specifically expressed in neurons we included the more ubiquitously expressed Kif5B, with reported role in chromosome alignment, in our interaction analysis. Finally, we found dynein in our mass spectrometry analysis. Dynein cytoplasmic heavy chain (DYNC1H1) occurs in a multimeric complex of many dynein and dynactin subunits, and is also known to play a role in several mitotic processes including spindle assembly and chromosome congression. To determine if our proteins interact with the dynein motor complex, we used other subunits like dynein intermediate chain (DYNC1I2), and dynactin subunit p150 (DCTN1) in our interaction studies.

In order to validate the specificity of interaction with identified (and other candidate) proteins, we used mitotically synchronized HeLa cells stably

expressing Localization and Affinity Purification (LAP)-tagged bacterial artificial chromosome transgenes under the influence of their native promoters (Kind gift from A. Hyman; Poser et al., *Nat. Methods*, 2008). These proteins have C-terminus fusion with S-protein tag for affinity purification and GFP for localization (Poser et al., *Nat. Methods*, 2008). All the above mentioned LAP-tagged proteins were pull-down using S-protein agarose beads and checked for their interaction with the subunits of MLL complex. As WDR5, but not RbBP5 or Ash2L, has been found in other protein complexes (van Nuland et al., *Mol. Cell Biol.* 2013), we used RbBP5 to confirm if the interaction was indeed with MLL complex. In all LAP tagged proteins, except Kif2B and Kif5A, the GFP fused kinesin or dynein protein was enriched in the pull downs with S-protein (Figure 1A, see anti-GFP antibody immunoblot). When checked for interaction, Kinesin 13-family members, Kif2A, and Kif2C, pulled down substantial amounts of endogenous WDR5 and RbBP5. Similarly, Kif2B, Kif5A, Kif5B, and all dynein subunits (DYNC1H1, DYNC1I2 and DCTN1) showed interaction with WDR5 and RbBP5 (Figure 1A). HeLa cells stably expressing SFB-tagged GFP showed no interaction with WDR5 and RbBP5 proteins. We also checked Kif11 (Kif11-LAP), another kinesin active in mitosis, for interaction with WDR5 and RbBP5, and found no significant interaction among these proteins (Figure 1A). Our results indicate that MLL complex is able to interact with some members of kinesin and dynein motor proteins.

In order to advance our studies, we focused on Kinesin-13 family proteins which regulate MT dynamics to control spindle assembly and chromosome congression. Out of these, Kif2A showed strongest association with WDR5 and Kif2B weakest in Glutathione-S-transferase (GST) affinity protein interaction study (data not shown). Hence, we decided to study Kif2A further. Moreover, Kif2A has been identified as WDR5 interacting partner previously as well (van Nuland et al., *Mol. Cell Biol.* 2013).

We started by immunoprecipitating endogenous WDR5, RbBP5 and MLL_C subunits using specific antibodies (Figure 1B). All subunits of the MLL complex were able to pull down endogenous Kif2A. As a control, we used MLL2, a MLL family member that did not show any mitotic defects in our previous assays. While we were able to immunoprecipitate MLL2 in quantities comparable to MLL_C, Kif2A band was not apparent in MLL2 IP (Figure 1B) indicating that

Kif2A interaction was specific to MLL1. We also probed for endogenous Kif2A in the SFB-WDR5 and SFB-MLL_C pull-downs. We found that SFB-MLL_C, but not SFB-GFP, was able to pull-down endogenous Kif2A as robustly as SFB-WDR5 (Figure 1C).

Next, we performed pull-down experiments using GST or GST-WDR5 fusion proteins. While GST-WDR5 interacted robustly with endogenous Kif2A, no interaction was detected with another spindle associated kinesin—Kif11 (Kif11-LAP, detected using anti-GFP) indicating that the interaction between Kif2A and WDR5 was highly specific (Figure 1D). Our mutational analysis had demonstrated that MLL_C subunit regulates proper chromosome alignment and also interacts with Kif2A (Figure 1B, C). In order to map the domain of MLL_C, which interacted with Kif2A, we created three truncations of MLL_C protein as shown in Figure 1E (top) and expressed them as N-terminal GST fusions. Using U2OS cells stably expressing GFP-Kif2A, we found that GFP-Kif2A interacted with GST-MLLC D2 and D3 truncations but not with GST-MLLC D1 (Figure 1E). These experiment show that both WDR5 and MLLC can interact with Kif2A and these three proteins most likely occur as a complex.

MLL complex regulates Kif2A recruitment to the spindle poles and spindle microtubules.

To explore mitosis-specific regulation of Kif2A, we depleted Kif2A by RNAi. siRNA treatment removed Kif2A from the poles and spindle MTs (Figure 2A). A majority of Kif2A-deficient cells display bipolar spindles if they enter mitosis in the presence of low doses of nocodazole (Ganem and Compton, *J. Cell Biol.* 2004). Out of these 16.26% cells displayed elongated spindle similar to what we observed upon MLL or WDR5 siRNA knockdown previously. Further, analogous to MLL and WDR5 RNAi, and consistent with previous report, we observed that Kif2A knockdown leads to significant chromosome misalignment (Figure 2C a) and increased pole-to-pole distance (Figure 2B b). This indicated that Kif2A and MLL may act in the same pathway to regulate spindle assembly and chromosome congression.

To investigate how MLL/WDR5 may functionally regulate Kif2A, we explored the localization of endogenous Kif2A on the spindle pole and MTs upon MLL or WDR5 knockdown. We observed that endogenous Kif2A staining at the spindle poles and MTs diminished considerably upon depletion of MLL or WDR5 during mitosis (Figure 2C) even though the levels of Kif2A seemed

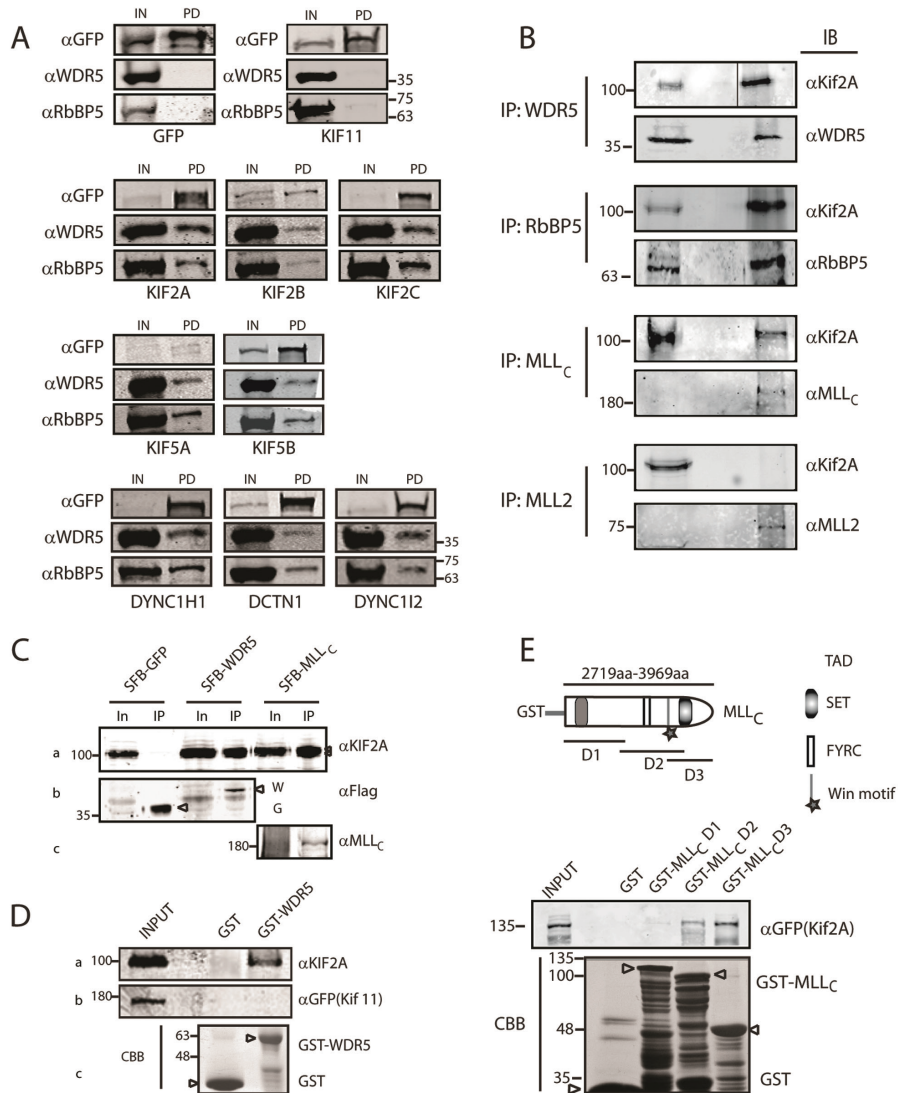
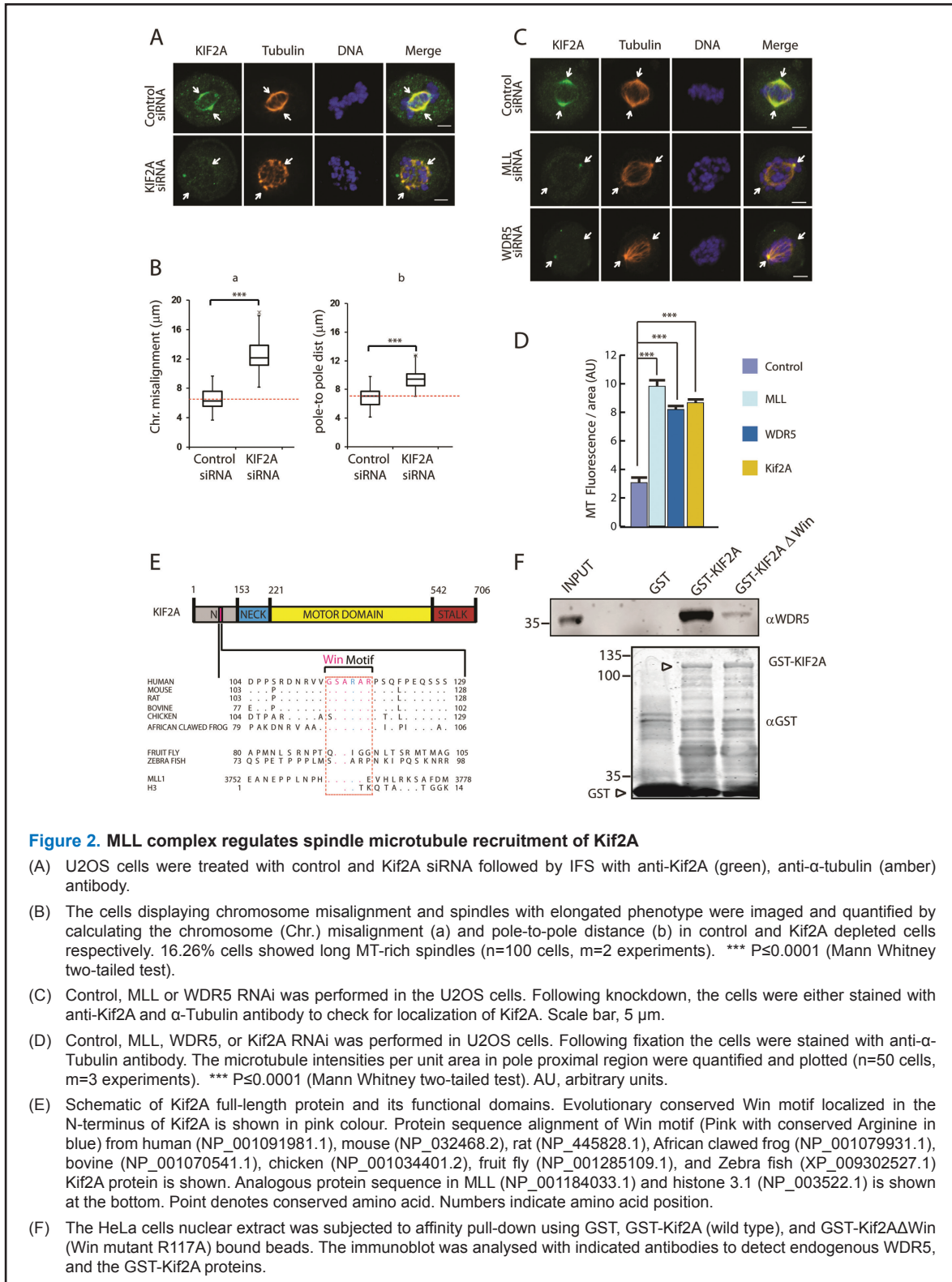


Figure 1. MLL complex interacts with kinesin and dynein motor proteins.

- (A) The HeLa cells expressing transgenes with LAP-tag fusion at C-terminus were used. LAP tagged-Kif2A, Kif2B, Kif2C, Kif5A, Kif5B, dynein heavy chain (DYNC1H1), Dynein Intermediate chain (DYNC1I2), and Dynactin subunit (DCTN1) expressing cells were synchronized in mitosis, lysed and subjected to affinity pull-down using S-protein agarose beads. The HeLa cells expressing SFB-GFP or Kif11-LAP were used as controls. The recovered bead-bound proteins were analysed by immunoblot using antibodies indicated on left. Position of molecular weight marker (in kDa) is shown on the right. IN, input; PD, pull down.
- (B) HeLa spinner cells were lysed and subjected to endogenous immunoprecipitation (IP) using antibodies against endogenous WDR5, RbBP5, MLL_C, and MLL2. The anti-IgG antibody was used as control. The immunoblots (IB) were probed with antibodies indicated on right. Black line indicates that intervening lanes have been spliced out.
- (C) HeLa cells stably expressing SFB-GFP, SFB-WDR5 and SFB-MLL_C were subjected to pull-downs using S protein-agarose beads. The immunoblots were probed with indicated antibodies to detect endogenous Kif2A and exogenous GFP, WDR5 (using anti-Flag) and MLL_C respectively. G, SFB-GFP; W, SFB-WDR5.
- (D) HeLa cells stably expressing Kif11-LAP were synchronized in mitosis, lysed and subjected to affinity pull-down using GST and GST-WDR5-bound beads. The immunoblots were probed with anti-Kif2A (panel a) and anti-GFP (panel b) antibodies to detect endogenous Kif2A and transgenic Kif11-LAP proteins respectively. Panel c shows the bead-bound GST or GST-WDR5 proteins stained with Coomassie Brilliant Blue (CBB).
- (E) The top figure shows schematic representation of N-terminal GST fused fragments of MLL_C subunit (D1 to D3; indicated in bold lines) used for interaction study shown below. Numbers indicate amino acid (aa) residues. TAD, transcriptional activation domain; FYRC, "FY-rich" domain C-terminal (FYRC); SET, SET domain; line with star denotes the WDR5 interacting (Win) motif. Bottom figure shows mitotically synchronized U2OS cells stably expressing GFP-Kif2A and subjected to affinity pull-down using GST, and deletions of MLL_C. Immunoblot was probed using anti-GFP. Proteins stained with CBB are shown at the bottom. (B-E) Position of molecular weight marker (in kDa) is shown on the left.

unaffected in interphase cells (data not shown). MLL is a transcriptional co-activator and affects a large number of genes. However, no significant changes in either the Kif2A transcript or H3K4

trimethylation levels at Kif2A promoter have been reported upon loss of MLL (Wang et al., Mol. Cell Biol. 2009).



Kif2A is a microtubule depolymerizing motor. Consistent with the reduced levels of spindle-pole-localized Kif2A in MLL and WDR5 siRNA treated cells, we observed increase in the levels of polymerized tubulin at the spindle poles, similar to that observed upon Kif2A knockdown (Figure 2D). Taken together our results demonstrate that MLL complex specifically regulates recruitment of Kif2A to spindle microtubules during mitosis, and thus its spindle-associated depolymerase function.

Kif2A interacts with WDR5 via a conserved 'Win' motif.

An arginine containing Win motif in MLL interacts with WDR5. Interestingly, we have discovered a similar arginine containing motif in the N-terminus region of Kif2A (Figure 2E). As shown, this evolutionary conserved motif shows considerable similarity with MLL Win motif (Figure 2E). Even though, we observed interaction of WDR5 with all Kinesin 13 motors (Figure 1A) the Win motif was only present in Kif2A. In order to address if this motif in Kif2A was responsible for its association with WDR5 subunit of MLL complex, we mutated the arginine in this motif to alanine (R117A) and performed protein interaction studies using GST fusion of Kif2A protein. We observed that while GST-Kif2A interacted robustly with WDR5 protein (Figure 2F), the association of GST-Kif2A Δ Win with WDR5 was pronouncedly reduced indicating that the Kif2A-WDR5 interaction was dependent on the Win motif of Kif2A.

To conclude, we have identified a crucial role of MLL complex in regulation of spindle microtubule localization of Kif2A. Kif2A interacts with MLL and WDR5 via its 'Win' motif and MLL/WDR5 complex recruits it to spindle poles in mitosis. This activity of MLL complex ensures proper chromosome segregation and spindle formation during mitosis.

Project 2: Role of H3K4 HMTs in regulation of repetitive non-coding regions.

Although MLL and family members are best studied for their role as HMT in transcription, many aspects of even this role are not well understood. Here we are specifically referring to non-coding repeats regions like centromere, and loci transcribed by RNA Polymerase I (RNAPI)—

ribosomal DNA (rDNA), which have been shown to bear H3K4 methylation marks. However, the identity of the methyltransferase, depositing these marks to facilitate transcription on these regions, is not known. In this new project we will address the role of H3K4 HMTs in these regions and try to understand if H3K4 is involved in regulating non-coding RNA transcription as well.

Summary of work done until the beginning of this reporting year (up to March 31, 2017)

This is a new project.

Details of the progress made in the current reporting year (April 1, 2017 –March 31, 2018)

For addressing the role of H3K4 HMTs in non-coding repeat region transcription, we choose to look at the role of MLL at centromeres first. The centromere is a chromosomal locus where kinetochore; the macromolecular proteinaceous structure is assembled to facilitate chromosome segregation. Human centromeres consist of highly repetitive and homogenous higher-order repeats (HORs) of 171 bp alpha satellite monomers extending upto 5Mb in size. At alpha satellite domains, CENP-A containing nucleosomes are interspersed with H3 containing nucleosomes. Centromeric chromatin was believed to be heterochromatic in nature until marks such as H3K4Me2 and H3K36Me2 were discovered on the centromeres. Specific targeting of histone demethylase LSD1 to centromeric regions of human artificial chromosome lead to abrogation of centromere function followed by chromosome mis-segregation suggesting that H3K4 methylation actively participates in chromosome segregation. RNA polymerase II (RNAPII) transcribes the alpha-satellite repeats during mitosis and early G1 phase. Non-coding transcripts resulting from alpha-satellite repeats recruit CENP-A chaperon HJURP to load CENP-A on centromeres in human. Both H3K4Me2 and H3K36Me2 maintain the open state of chromatin that is favorable for transcription. The epigenetic players of repressive H3K9Me3 at pericentric region are well characterized while knowledge about the complexes that impart activating H3K4Me2 at centromere is completely lacking.

We will undertake systematic experiments to uncover the identity of methyltransferase active at these regions.

Publications:

1. Zafar Ullah Zargar, Mallikharjuna Kimidi and Shweta Tyagi (2018). Dynamic site-specific recruitment of RBP2 by pocket protein p130 modulates H3K4 methylation on E2F-responsive promoters. *Nucleic Acids Res.* 46(1): 174-188.
2. Ali A, Veeranki SN, Chinchole A and Tyagi S (2017). MLL/WDR5 Complex Regulates Kif2A Localization to Ensure Chromosome Congression and Proper Spindle Assembly during Mitosis. *Developmental Cell*, 41 (6). 605-622.e7.

LABORATORY OF CELL DEATH & CELL SURVIVAL

Functional protein networks controlling cellular pathways

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	Maniharika Vemula	Research Associate
	Sunu Joseph	Project-SRF
	Kiranmayee V	BITS trainee
	Nanci Rani	Technical Assistant

Objective of the lab

The broad objective of the group is to identify and characterize new players in different cellular processes by mapping the phosphorylation and ubiquitination networks.

Summary of work done until the beginning of this reporting year (upto March 31, 2017)

In the theme of phosphatase biology, we initiated our studies on functional phosphatase network with the identification of interacting partners of every phosphatase in human cell. Phosphatases play a critical role in nearly every cellular process, including metabolism, gene transcription, translation, cell cycle progression, protein stability, signal transduction, and apoptosis. We cloned 143 human protein phosphatases in a gateway compatible triple tagged (SBP-Flag-S protein) vector and each of them was individually expressed in cells. Protein complexes were isolated by tandem affinity purification and interacting proteins were identified by using LC-MS/MS analysis. A total of 76773 interactions were obtained from 143 phosphatase purifications. Depending on the substrate residue they act on, protein phosphatases are broadly classified into two classes such as (A) Tyrosine phosphatases and (B) Serine/Threonine phosphatases. Firstly, we started to analyse the interactome of human tyrosine phosphatases. By utilizing proteomic approach, a total of 41872 interactions were obtained from 81 tyrosine phosphatase

purifications. By using a SAINT score cut off of 0.8, empirical fold change score $FCA > 3$, $FCB > 2.5$, $IS > 1$ and WD score > 1 , we identified high confident interactions (HCIs) mediated by 916 proteins (HCIPs). Finally, we presented an interaction network of 81 human tyrosine phosphatases built on 1884 high-confidence interactions of which 85% are unreported. During the course of this work, we have already identified and characterized WWP2, PNU2S and TOPK as novel functional partners of PTEN. Recently, we identified a new cellular function for PTEN where we have shown that PTEN via interacting with Rab7 functions in endosome maturation. In addition to PTEN interactome, we also started developing networks of other cellular phosphatases. We identified PPM1G as a molecular switch that controls differential cellular role of E3 ligase WWP2, by controlling the transition between monomeric WWP2 and WWP2/WWP1 heterodimer. In another example, we demonstrated an important role of non-receptor tyrosine phosphatase PTPN5 in cytokinetic abscission.

Details of progress in the current reporting year (April 1, 2017 - March 31, 2018)

Theme 1. Functional studies on phosphatase networks

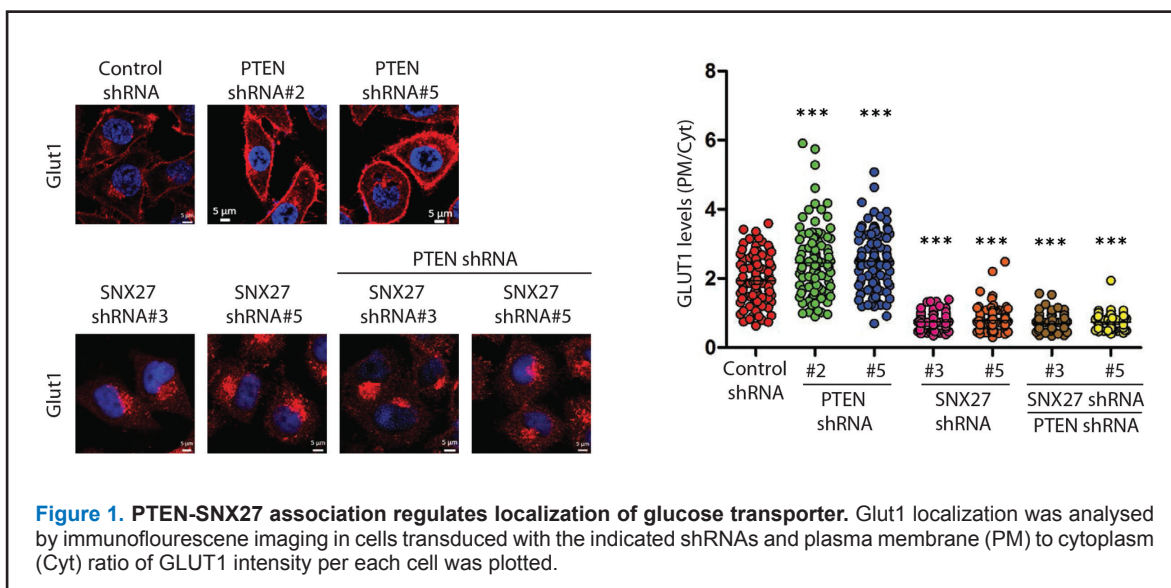
Currently, we are focused on actively expanding the functional network of different families of

phosphatases in the cell. In order to further understand the functional role of these interactions, we annotated them to KEGG pathways. Importantly, several key cellular signaling pathways such as PI3-K, Hippo-YAP, Wnt, Hedgehog, HIF-1, mTOR, Ras-MAPK, AMPK, RAP1 and VEGF were highly enriched for HCIPs of different phosphatases. We found several known as well as novel phosphatase associations enriched among these cellular signaling pathways. For example, CDC25 phosphatases were found to interact with cell cycle proteins, dual specific phosphatases (DUSPs) interact with proteins in MAPK signaling pathway. But, at the same time other phosphatases such as DUSP family were found to be associated with DNA replication proteins, implicating new functions for these phosphatases in this process. Interestingly, we also found several phosphatases associated with RAB/VPS/SNX family of proteins, possibly indicating new functions for these phosphatases in vesicular trafficking. Given that roles of phosphatases in regulation of vesicular trafficking were limited so far, this data may be used to further explore roles of individual phosphatases in this critical cellular process.

In addition to mapping the phosphatase network, we simultaneously started to characterize several of putative functional interactions of these purified phosphatases. To this end, we made significant progress in understanding multiple novel phosphatase interactions in the lab. The data generated from some of these exciting interactions has been presented below.

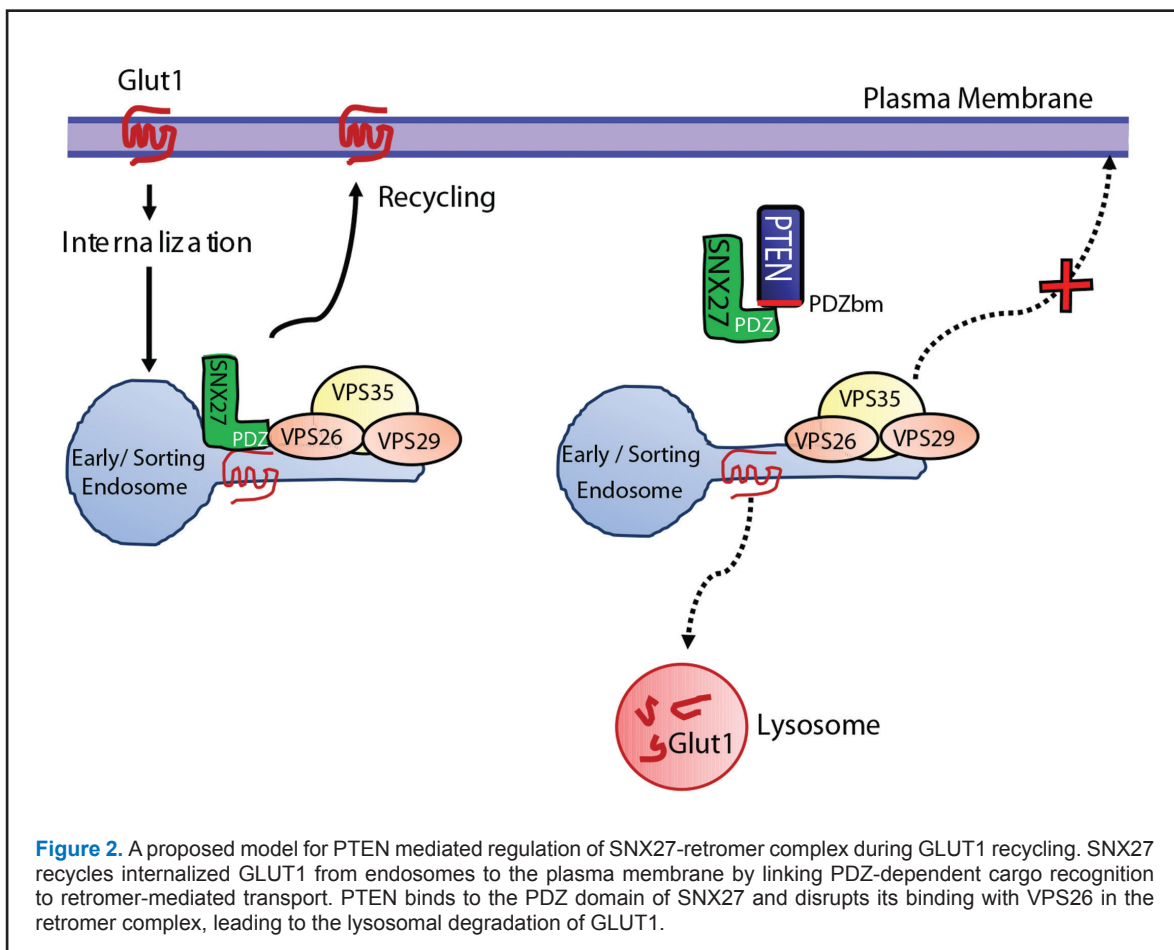
1.1 PTEN controls recycling of glucose transporters by modulating retromer assembly

PTEN is a well-known tumor suppressor that acts to down-regulate cell proliferation, survival and metabolic signaling pathways, majorly through its lipid phosphatase activity. Recently, we have demonstrated a new role for PTEN during late endosome maturation, where it controls trafficking of EGFR by regulating Rab7 localization to endosomes. In addition to its role in endosome maturation, now we identified a critical regulatory role of PTEN in endosomal recycling of GLUT1 and glucose transport in a phosphatase independent manner. In our interactome analysis, we found SNX27 as one of the novel PTEN associated proteins. SNX27 is sorting nexin protein that associates with the retromer complex (a heterotrimer of VPS26-VPS29-VPS35) and promote the endosome-to-membrane trafficking of plasma membrane receptors such as glucose transporter. We confirmed the *in vitro* as well as *in vivo* association of PTEN with SNX27. We found that PDZ binding motif (TKV) PTEN and PDZ domain of SNX27 are required for their interaction. Interestingly, our search for PTEN somatic mutations outside its phosphatase domain using COSMIC database revealed a pathogenic PTEN mutation at 401 position in the PDZ binding motif where threonine is mutated to isoleucine in soft tissue sarcoma. Importantly, while full length PTEN efficiently associates with SNX27, the T401I mutant is severely defective in binding to SNX27 further supporting the importance of intact PDZ binding motif in PTEN for their interaction.



Functionally, we found that PTEN-SNX27 interaction is critical for regulation of recycling of glucose transporter GLUT1 to the plasma membrane (Figure 1). Depletion of PTEN in cells resulted in increased GLUT1 levels at the plasma membrane, which were lowered upon co-depletion of SNX27 suggesting that PTEN suppresses GLUT1 levels at plasma membrane by controlling SNX27. Further, we found that depletion of PTEN significantly enhanced cellular uptake of glucose. Interestingly, we observed increased glucose uptake upon PTEN depletion even in the presence of an active Akt inhibitor (MK-2206), suggesting that PTEN may control glucose transport independent of its phosphatase function and classical Akt

pathway. Mechanistically, we have shown that PTEN competes with the binding of SNX27 with its retromer components, specifically with VPS26, and thus PTEN prevents the assembly of SNX27-retromer complex. Our analysis with various deletion and point mutants of PTEN and SNX27 suggested that PTEN binds with SNX27 adjacent to VPS26 binding site and therefore restricts access to VPS26 leading to defective retromer assembly. In conclusion, we demonstrated that PTEN binds with SNX27 and hinders its access to VPS26 retromer complex and thereby prevents the recycling of GLUT1 to plasma membrane followed by impaired glucose uptake (Figure 2).



1.2. PHLPP1-SGT1 interaction is essential for kinetochore assembly

PHLPP is a tumor suppressor phosphatase that plays critical roles in cell survival. In this study, we identified PHLPP1 as an essential protein required for proper assembly of kinetochores in cells. By analyzing our interactome data, we

found SGT1 as one of the potential interacting partners of PHLPP1. Since SGT1 is critical for proper kinetochore assembly during mitotic cycle, we tested if loss of PHLPP1 phenocopies SGT1 loss from cells. Time-lapse imaging revealed that silencing of PHLPP1 in HeLa cells lead to delayed progression of cells in mitosis.

Delayed progression of cells in mitosis upon PHLPP1 depletion is accompanied with multiple severe mitotic defects such as misaligned chromosomes, multipolar spindles and abnormal centrosomes. We found that outer kinetochore proteins such as HEC1 and CENP-E failed to localize to kinetochores in PHLPP1 depleted cells. Mechanistically, we found that PHLPP1 dephosphorylates SGT1 at 4 conserved residues (S17, S249, S289, T233). Phosphorylation of these residues on SGT1 acts as a signal for recognition by ubiquitin machinery, which is subsequently sent for proteasomal degradation. Thus, loss of PHLPP1 from cells resulted in accumulation of phosphorylated SGT1 there by promotes SGT1 degradation and thereby causes defective assembly of kinetochores. We found RNF41 as a novel E3 ligase that ubiquitinate and degrade SGT1 in a phosphorylation dependent manner. Interaction of SGT1 with RNF41 is dramatically enhanced in the absence of PHLPP1 and conversely exogenous expression of PHLPP1 lead to loss of SGT1 interaction with its E3 ligase. Thus, PHLPP1 protects SGT1 from polyubiquitination and degradation by interfering with SGT1 interaction with its E3 ligase RNF41. Importantly, either depletion of RNF41 or expression of non-phosphorylatable SGT1 mutant rescued the kinetochore defects caused due to PHLPP1 loss. To further test if the existence of interplay between PHLPP1 and RNF41 is functionally relevant for kinetochore assembly, we performed double depletion experiments. We hypothesized that SGT1 loss and kinetochore defects caused by PHLPP1 depletion would be rescued by simultaneous depletion of RNF41. In support of this hypothesis co-depletion of RNF41 and PHLPP1 rescued SGT1 loss as well as recruitment of other proteins such as HEC1 and CEPN-E at kinetochores. In conclusion, we demonstrated the existence of a dynamic interplay between PHLPP1 and RNF41 in regulation SGT1 stability, which is critical for proper kinetochore assembly.

Theme 2: Roles of canonical and non-canonical ubiquitination in cells

Ubiquitination is an ATP-dependent, highly ordered multistep enzymatic process, which results in covalent attachment of ubiquitin to the substrate. Ubiquitin linked to the substrates serves as a molecular tag that marks proteins for either degradation by proteasome dependent pathway or to function in wide variety of processes in a proteasome independent manner. In this theme we are interested in studying both the canonical

and non-canonical functions of ubiquitination in cells. During previous years, we have reported that an oncogenic E3 ligase WWP2 ubiquitinates PTEN and p73 in a canonical K48 linkage that leads to their degradation through proteasome. On the other hand, we have demonstrated a novel degradation independent functional role for non-canonical ubiquitin linkages (K27 chains) in mediating protein secretion. We have shown that K27 ubiquitin linkage on YB-1 by HECT-E3 ligase HACE1 is necessary for its interaction with Tumor Susceptibility Gene 101 (TSG101), a component of the Multi Vesicular Body (MVB) pathway, which facilitates its secretion. Currently, we are expanding the functional diversity of ubiquitination by finding new components in the ubiquitin system.

2.1. Identification of new functional substrates of oncogenic E3 ligases

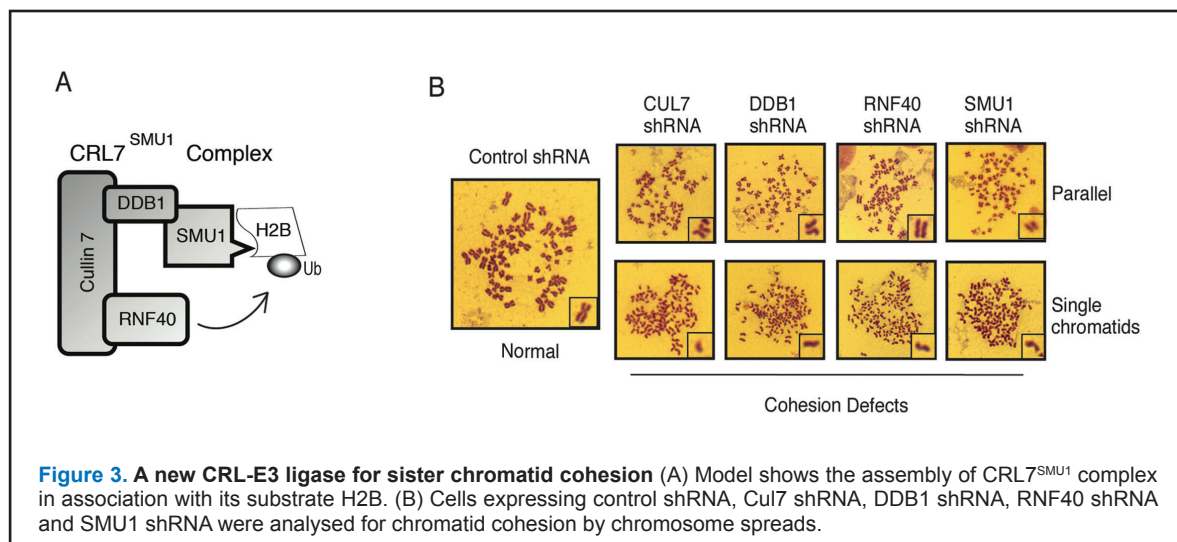
We identified a HECT-type E3 ligase WWP2 as a potential oncogene in our previous studies. Currently, we are searching for the functionally relevant substrates for this E3 ligase in cells. By using interaction proteomics approach, we found that WWP2 binds to DVL2, a critical component of Wnt signaling pathway. We found that WWP2 ubiquitinates Dvl2 but, interestingly does not lead to its degradation. In our functional experiments, knock out of WWP2 from cells using CRISPR resulted in defective activation of Wnt pathway. Interestingly, we found several ubiquitin-binding domain containing proteins in the interacting list of DVL2 upon Wnt stimulation. It is possible that non-canonically ubiquitinated DVL2 might specifically interact with UBA containing proteins, which is critical for its translocation to the sites of Wnt induced signalosomes. We are currently probing the interactions of various UBA domain proteins with ubiquitinated DVL2, which will help us to mechanistically understand the basis of Wnt induced signalosome formation. We already tested DVL2 association with some of these ubiquitin-binding proteins such as MARK2 and RAP80 but however we did not find any evidence for their association to be regulated by WWP2. We continue to look for the other K63 ubiquitin chain binding proteins in the DVL2 list. On the other hand, we aim to assign new cellular functions for the HECT E3 ligases by performing substrate profiling using human protoarrays. ProtoArray human protein microarray version 5.0 contains over 9,000 unique human proteins individually purified and arrayed under native conditions. We performed

an *in vitro* ubiquitination assay on the array using WWP2 as an E3 ligase. Our preliminary analysis has revealed several new substrates for WWP2 that were associated with distinct functions. We are currently in the process of functionally characterizing these novel substrate-WWP2 associations.

2.2. Identification of new functional E3 ligase complexes and their substrates

E3 ligases are critical proteins in the final step of the ubiquitination process where they recruit ubiquitin charged E2 enzymes along with specific substrates. In this work, we aim to identify new complexes for E3 ligases by using proteomics approach and further characterize their substrates by using human protoarrays. In one example we identified a new CRL type E3 ligase (designated as CRL7^{SMU1} complex) that has an essential role in maintenance of chromatid cohesion (Figure 3). Previous studies have demonstrated that DCAF1/VprBP, a protein with WD repeat region and LisH domain acts as a substrate recognition component of HECT-type as well as RING-based E3 ubiquitin ligase complexes. To identify proteins with a similar combination of LisH and WD repeat organization that may assemble E3 ligase complexes, we performed a global search for LisH domain (ID: PS50896) using UniProt database. We retrieved 28 human proteins that contain LisH domain of which nine of them exhibit a combination of LisH domain with WD repeats in their architecture.

To test the possibility if these proteins assemble E3 ligase complexes, we isolated protein complexes associated with SMU1, one of the listed proteins with such a domain organization. We identified that SMU1 assembles CRL type of E3 ligase that contains DDB1, CUL7 and RNF40 E3 ligase as core components. We also identified Histone H2B as a bonafide substrate for this new E3 ligase complex. SMU1 acts as a substrate recognition component in the E3 ligase complex. siRNA mediated depletion of SMU1 lead to loss of H2B interaction with E3 ligase components and there by resulted in diminished substrate ubiquitination. Functionally, we found that knock down of individual components of CRL7^{SMU1} complex led to accumulation of mitotic cells with multiple mitotic defects such as lagging chromosomes, anaphase/nuclear bridges, multipolar spindles and defective chromatid cohesion. Mechanistically, we have shown that depletion of CRL7^{SMU1} leads to loss of H2B ubiquitination at SMC1a locus and thus subsequently compromised SMC1a expression in cells. Knock down of CRL7^{SMU1} components or loss of H2B ubiquitination led to defective sister chromatid cohesion, which is rescued by restoration of SMC1a expression. In conclusion, we identified a new CRL type E3 ligase complex that promotes monoubiquitination of H2B to drive the expression of SMC1a, which is essential for maintenance of sister chromatid cohesion during mitosis.



Publications

Research papers published in 2017

1. Shinde SR, Maddika S (2017). PTEN regulates glucose transporter recycling by impairing SNX27 retromer assembly. **Cell Rep.** 21(6): 1655-1666.
2. Gangula NR, Maddika S (2017). Interplay between the phosphatase PHLPP1 and E3 ligase RNF41 stimulates proper kinetochore assembly via the outer-kinetochore protein SGT1. **J Biol Chem.** 292(34): 13947-13958.
3. Kumar P, Munnangi P, Chowdary KR, Shah VJ, Shinde SR, Kolli NR, Halehalli RR, Nagarajaram HA, Maddika S (2017). A human tyrosine phosphatase interactome mapped by proteomic profiling. **J Proteome Res.** 16(8): 2789-2801.

4. Shinde SR, Maddika S (2018). Post-translational modifications of Rab GTPases. **Small GTPases.** 9(1-2): 49-56.

Research papers in press as on 31st March 2018

1. Shah VJ, Maddika S. CRL7^{SMU1} E3 ligase complex-driven H2B ubiquitylation functions in sister chromatid cohesion by regulating SMC1 expression. **J Cell Sci.**
2. Behera S, Kapadia B, Kain V, Alamuru-Yellapragada NP, Murunikkara V, Kumar ST, Babu PP, Seshadri S, Shivaridraiah P, Hiriyan J, Gangula NR, Maddika S, Misra P, Parsa KVL. ERK1/2 activated PHLPP1 induces skeletal muscle ER stress through the inhibition of a novel substrate AMPK. **Biochim Biophys Acta.**

LABORATORY OF CELL SIGNALLING

Investigating the functions of phosphate-rich biomolecules in eukaryotic cells

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	Sitalakshmi Thampatty R	Senior Research Fellow
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	Arpita Singh	Junior Research Fellow (since Feb. 2018)
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	Ravichand Palakurti	Research Associate
	Manasa Chanduri	Research Associate
	Prathyusha Munnangi	Project JRF (till Dec. 2017)
	Vineesha Oddi	Project Technical Assistant
	Aditya Rane	Project JRF (since Sept. 2017)
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	Paul Wender	Stanford University, USA
	Dorothea Fiedler	FMP, Berlin, Germany
	Kana M. Sureshan	IISER, Thiruvananthapuram

Objectives

Our laboratory studies the biochemical, cellular and physiological functions of two phosphate-rich biomolecules: (i) the inositol pyrophosphate, IP_7 (5PP- IP_6), and (ii) inorganic polyphosphate (polyP). Our broad objectives are (a) to understand the cellular processes by which the levels of these small molecules are regulated, and (b) investigate the cellular and physiological processes that these phosphate-rich molecules influence.

Summary of work done until the beginning of this reporting year (upto March 31, 2017)

IP_7 is synthesised from IP_6 and ATP by a family of enzymes known as inositol hexakisphosphate (IP_6) kinases, of which there is one isoform in budding yeast, and three isoforms in mammals. We utilise *S. cerevisiae*, mammalian cell lines, and knockout mouse strains as model systems to investigate the signalling and metabolic pathways that are altered when IP_7 levels are perturbed.

Protein pyrophosphorylation is a unique attribute of inositol pyrophosphates such as IP_7 , wherein the β -phosphate moiety can be transferred from IP_7 to a pre-phosphorylated serine residue in a protein to generate pyrophosphoserine. We have previously demonstrated that IP_7 -mediated pyrophosphorylation regulates RNA polymerase I activity in budding yeast (Thota et al., Biochem J, 2015), and dynein motor function in mammalian cells (Chanduri et al., Biochem J, 2016). We also presented preliminary data demonstrating that the oncoprotein c-Myc can undergo pyrophosphorylation in vitro, and that c-Myc shows a longer half-life and lower ubiquitylation levels in cells lacking IP6K1.

We have earlier reported that male mice lacking IP6K1 are infertile. IP6K1 expression was observed predominantly in pachytene spermatocytes and round spermatids. Mice lacking IP6K1 complete meiosis to generate round spermatids, but these fail to mature properly, instead forming irregularly shaped abnormal

elongated spermatids that undergo apoptosis. Therefore, *Ip6k1^{-/-}* mice display spermiogenesis failure and consequent azoospermia, the absence of mature spermatozoa in the epididymides.

Polyphosphate (polyP) is a biopolymer that consists of phosphate units of varying number linked by phosphoanhydride bonds. PolyP of chain length 60-100 phosphate units is present in dense granules of mammalian platelets, and regulates the blood clotting cascade at multiple stages. Other critical functions have been assigned to polyP in mammals, including cell signalling, membrane transport, and energy metabolism. PolyP research has lagged behind the exploration of other biopolymers, largely because uniform chain length polyP or nonhydrolysable analogs of polyP are not available. While yeast and bacterial polyP synthases have been identified, they have no mammalian homologs based on sequence similarity. We have earlier demonstrated that mice lacking IP6K1, which have reduced levels of IP₇, also have low levels of platelet polyP, and consequently display defects in blood clotting (Ghosh et al., 2013).

Details of progress made in the current reporting year (April 1, 2017 - March 31, 2018)

Project 1: Inositol pyrophosphates regulate stability of the oncoprotein c-Myc

To examine whether the oncoprotein c-Myc undergoes endogenous pyrophosphorylation, we conducted a 'back pyrophosphorylation' assay, and observed significantly reduced 5[$\beta^{32}\text{P}$]IP₇ mediated pyrophosphorylation of native c-Myc from *Ip6k1^{+/+}* MEFs compared with *Ip6k1^{-/-}* MEFs (Figure 1A), suggesting that this protein is heavily pyrophosphorylated *in vivo*. Human and mouse c-Myc possess a PEST domain, which has been shown to signal protein degradation. This sequence, rich in Ser, Asp and Glu residues is likely to be a site for pyrophosphorylation by IP₇ (Figure 1B). Incubation of radiolabeled IP₇ with the c-Myc

PEST domain (amino acid residues 201-268 in human c-Myc) revealed that the PEST domain is indeed the region of pyrophosphorylation (Fig 1C). By subjecting shorter fragments of the c-Myc PEST sequence to phosphorylation by CK2, followed by IP₇-mediated pyrophosphorylation, we mapped the sites of pyrophosphorylation to the fragment containing residues 238 to 265 (Figure 1B). Mass spectrometry analysis revealed that CK2 phosphorylates two residues in the sequence ranging from Thr244 to Ser252. We generated two mutant versions of the c-Myc PEST sequence (aa 201 to 268): (i) 3S/A, in which Ser 249, 250 and 252 are replaced by Ala, and (ii) 3S/T in which Ser 249, 250 and 252 are replaced by Thr. As expected, IP₇ mediated pyrophosphorylation was abolished in the 3S/A mutant (Figure 1C). Interestingly, the 3S/T mutant underwent CK2 phosphorylation, but pyrophosphorylation was substantially reduced (Figure 1C). This data reveals for the first time that phosphorylated Thr residues do not accept the β phosphate from IP₇ with the same efficiency as phosphorylated Ser residues. To determine the effect of phosphorylation or pyrophosphorylation of the target Ser residues on the stability of full length c-Myc, we replaced Ser 249, 250 and 252 with (i) Ala to prevent phosphorylation and pyrophosphorylation, (ii) Thr to allow phosphorylation but disallow pyrophosphorylation, or (iii) Asp, to mimic phosphorylation but not pyrophosphorylation. All three mutant versions of c-Myc showed lower levels of pyrophosphorylation by IP₇ (Figure 1D). When expressed in HEK293 cells, all three mutants displayed a longer half-life (Figure 1E), and reduced ubiquitylation (Figure 1F), compared with the native protein. Therefore, the loss of Ser pyrophosphorylation on c-Myc mirrors the situation in *Ip6k1^{-/-}* MEFs, where c-Myc cannot be pyrophosphorylated and shows less ubiquitylation and greater stability. We are currently attempting to identify the E3 ligase that binds to the PEST motif in c-Myc, and determine whether this binding is regulated by pyrophosphorylation of the target site.

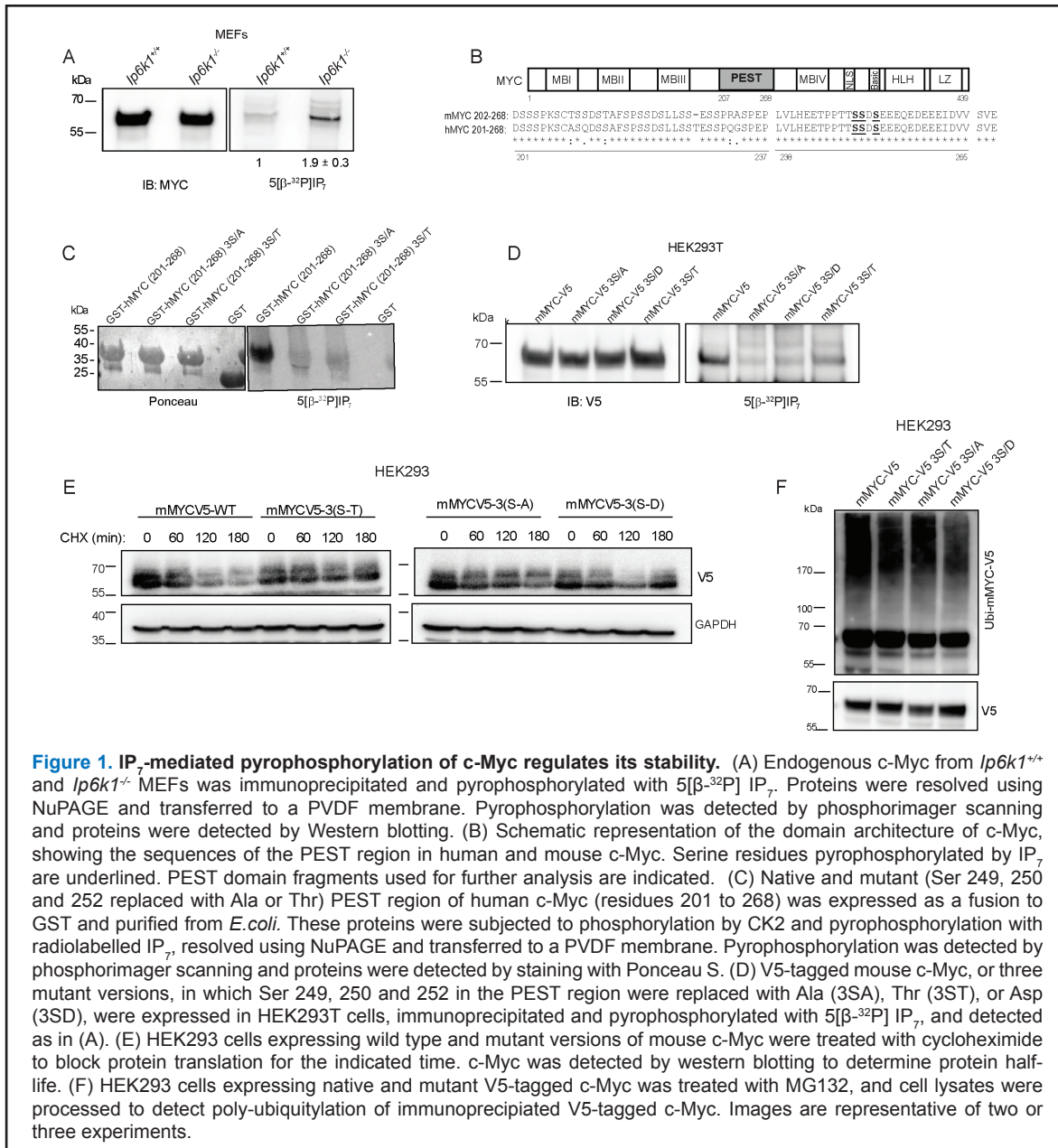


Figure 1. IP₇-mediated pyrophosphorylation of c-Myc regulates its stability. (A) Endogenous c-Myc from *Ip6k1^{+/+}* and *Ip6k1^{-/-}* MEFs was immunoprecipitated and pyrophosphorylated with 5[β-³²P] IP₇. Proteins were resolved using NuPAGE and transferred to a PVDF membrane. Pyrophosphorylation was detected by phosphorimager scanning and proteins were detected by Western blotting. (B) Schematic representation of the domain architecture of c-Myc, showing the sequences of the PEST region in human and mouse c-Myc. Serine residues pyrophosphorylated by IP₇ are underlined. PEST domain fragments used for further analysis are indicated. (C) Native and mutant (Ser 249, 250 and 252 replaced with Ala or Thr) PEST region of human c-Myc (residues 201 to 268) was expressed as a fusion to GST and purified from *E. coli*. These proteins were subjected to phosphorylation by CK2 and pyrophosphorylation with radiolabelled IP₇, resolved using NuPAGE and transferred to a PVDF membrane. Pyrophosphorylation was detected by phosphorimager scanning and proteins were detected by staining with Ponceau S. (D) V5-tagged mouse c-Myc, or three mutant versions, in which Ser 249, 250 and 252 in the PEST region were replaced with Ala (3SA), Thr (3ST), or Asp (3SD), were expressed in HEK293T cells, immunoprecipitated and pyrophosphorylated with 5[β-³²P] IP₇, and detected as in (A). (E) HEK293 cells expressing wild type and mutant versions of mouse c-Myc were treated with cycloheximide to block protein translation for the indicated time. c-Myc was detected by western blotting to determine protein half-life. (F) HEK293 cells expressing native and mutant V5-tagged c-Myc was treated with MG132, and cell lysates were processed to detect poly-ubiquitylation of immunoprecipitated V5-tagged c-Myc. Images are representative of two or three experiments.

Project 2. Role of IP6K1 in mouse spermatogenesis

Immunofluorescence analysis of squash preparations of wild type testes revealed enrichment of IP6K1 in perinuclear granules in round spermatids (Figure 2A). These granules are reminiscent of chromatoid bodies, which are ribonucleoprotein complexes found in round spermatids, and are involved in mRNA translational control, mRNA decay and small RNA-mediated gene regulation. Co-staining with the chromatoid body marker protein MIWI revealed that IP6K1 is indeed localised to the

chromatoid body in round spermatids, in addition to being present in the cytoplasm (Figure 2A). The ultrastructure of the chromatoid body was further examined by transmission electron microscopy. In contrast to massive sponge-like perinuclear aggregates observed in *Ip6k1^{+/+}* spermatids, chromatoid bodies were either fragmented or absent in *Ip6k1^{-/-}* spermatids (Figure 2B). Spermiogenesis is accompanied by extensive chromatin reorganization during which most of the nucleosomal histones are initially replaced by transition proteins (TNP1 and TNP2) and subsequently by protamines

(PRM1 and PRM2). The mRNAs encoding these proteins are transcribed in round spermatids, but are stored in a translationally repressed state to be translated during spermatid elongation. To investigate whether the loss of the chromatoid body in *Ip6k1*^{-/-} spermatids leads to changes in TNP2 and PRM2 expression, we examined the levels of these proteins in 28 and 35 day old juvenile testes. At 28dpp, neither protein was detected in *Ip6k1*^{+/+} testes whereas *Ip6k1*^{-/-} testes showed premature expression of both these proteins (Figs. 2C, D). TNP2 and PRM2 expression was detected in both *Ip6k1*^{+/+} and *Ip6k1*^{-/-} 35dpp testes, but protein levels were higher in knockout mice. In contrast to protein levels, there was either a decrease or no change

in the levels of *Tnp2* and *Prm2* transcript in 28 and 35dpp *Ip6k1*^{-/-} compared with *Ip6k1*^{+/+} testes. This suggests that the premature expression of these proteins is not attributable to an increase in mRNA abundance, but instead is a result of derepression of their translational silencing. Testicular sections of 28 dpp mice revealed that TNP2 is highly expressed in the nuclei of a few abnormally condensed *Ip6k1*^{-/-} spermatids (Figure 2E), and PRM2 is prematurely expressed in the nuclei of most of the *Ip6k1*^{-/-} round spermatids (Figure 2F). Taken together, our data suggest that IP6K1 is essential for chromatoid body formation and for the temporal regulation of *Tnp2* and *Prm2* mRNA expression in round spermatids. This work was published recently (Malla and Bhandari, 2017).

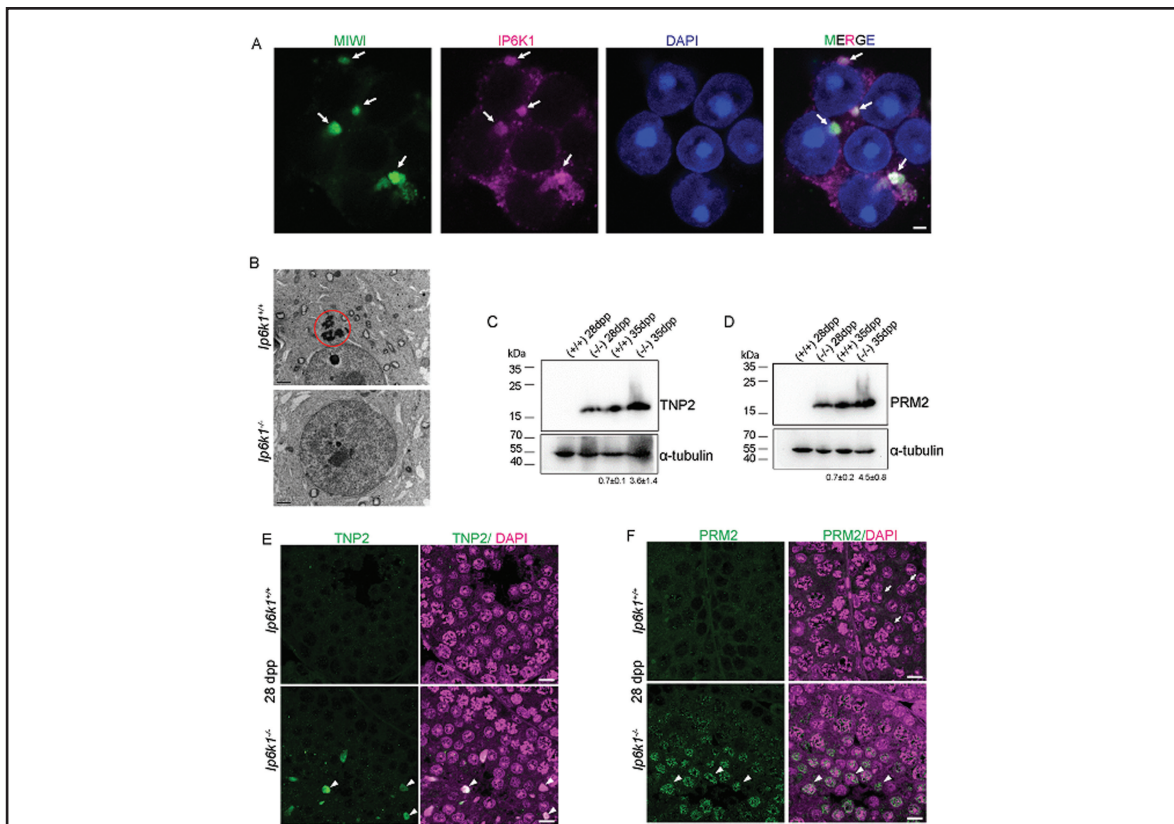
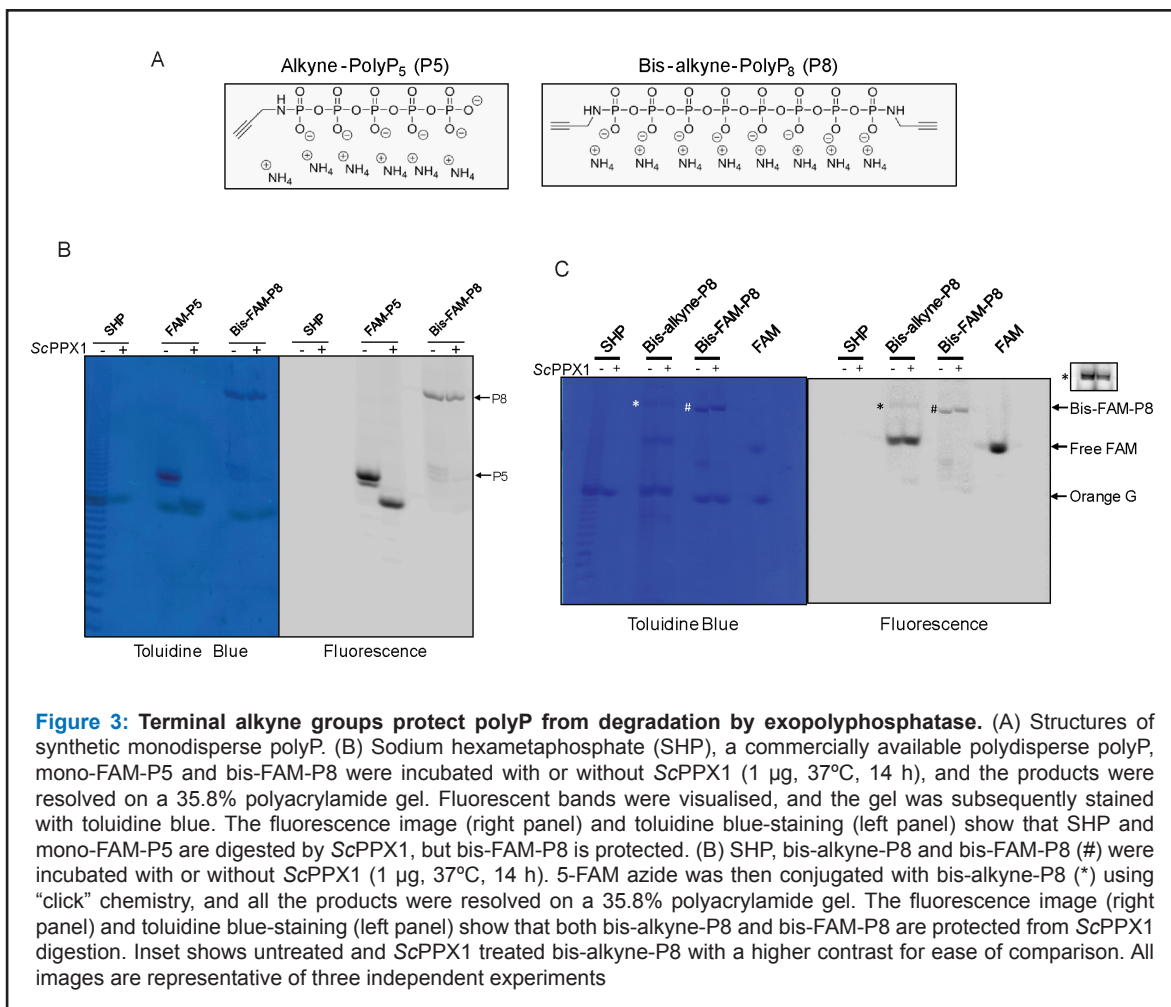


Figure 2. IP6K1 is essential for formation of chromatoid body (CB) and prevents the premature translation of key spermiogenic genes. (A) Immunostaining of round spermatid squash preparations shows colocalisation (white) of IP6K1 (magenta) with the chromatoid body marker MIWI (green) indicating that IP6K1 is present in the chromatoid body (arrows). Spermatid nuclei were counterstained with DAPI (blue). Scale bar: 2 µm. (B) Representative TEM images of round spermatids showing amorphous lobulated electron-dense chromatoid bodies in *Ip6k1*^{+/+} spermatids (encircled) and no chromatoid bodies in *Ip6k1*^{-/-} spermatids. Scale bar: 1 µm. (C,D) Western blot analysis of TNP2 (C) and PRM2 (D) in 28 dpp (days postpartum) and 35 dpp testes of *Ip6k1*^{+/+} (+/+) and *Ip6k1*^{-/-} (-/-) mice reveals premature expression of these proteins in 28 dpp *Ip6k1*^{-/-} testes. The levels of TNP2 or PRM2 in 35 dpp testes, normalised to the levels of the loading control (α-tubulin) are indicated as the mean±s.e.m. from three independent experiments. (E, F) Immunostaining (green) of *Ip6k1*^{+/+} and *Ip6k1*^{-/-} 28 dpp testis cross-sections to detect TNP2 (E) or PRM2 (F). Nuclei are counterstained with DAPI (magenta). *Ip6k1*^{+/+} 28 dpp testes show no detectable TNP2 staining, whereas *Ip6k1*^{-/-} testes show premature translation of TNP2 in some abnormally shaped spermatids (arrowheads). PRM2 is prematurely translated in round spermatids of 28 dpp *Ip6k1*^{-/-} mice (arrowheads) but not present in *Ip6k1*^{+/+} round spermatids (arrows). Scale bar: 10 µm.

Project 3. Function and metabolism of polyphosphate in mammals

Our collaborator, Dr. Henning Jessen, from the University of Freiburg, Germany, has developed a procedure to chemically synthesize monodisperse (i.e. fixed chain length) polyP and tag it at either one or both ends with an alkyne group, which can then be used to conjugate the polyP to any chemical group, including the fluorophore FAM, using "click" chemistry (Figure 3A). We examined whether the alkyne group present at the ends of synthetic monodisperse polyP is able to protect against degradation by exopolyphosphatase, which is likely to be present in all cell extracts. *S. cerevisiae* exopolyphosphatase (PPX1) was able to completely digest a 5-mer polyP that

was coupled with a fluorophore (FAM) at only one end, whereas polyP₈, that was coupled with FAM at both ends, was resistant to cleavage by PPX1 (Figure 3B). To test whether protection from PPX1 was afforded by the presence of the bulky fluorophore, or whether an alkyne group alone is sufficient to prevent degradation of synthetic polyP₈ by exopolyphosphatases, we incubated bis-alkyne polyP₈ with PPX1, and subsequently coupled it to FAM for visualisation on a gel (Figure 3C). We noted that the small alkyne group is able to protect against PPX1 activity, suggesting that a terminal alkyne would confer resistance to polyP from degradation in a cell extract. We are currently attempting to use this synthetic monodisperse polyP to identify mammalian proteins that interact with polyP.



Publications

Research papers published in the calendar year 2017 (in print with final page numbers)

1. Malla AB and Bhandari R (2017). IP6K1 is essential for chromatoid body formation and temporal regulation of TNP2 and PRM2 expression in mouse spermatids. **Journal of Cell Science** 130: 2854-2866.
2. Shah A, Ganguli S, Sen J and Bhandari R (2017). Inositol pyrophosphates: energetic, omnipresent and versatile signalling molecules. **Journal of the Indian Institute of Science** 97: 23-40.

Research papers in press as on 31st March 2018 (Note: Advance online publication prior to appearing in print will be considered 'in press')

1. Ansari MZ, Kumar A, Ahari D, Priyadarshi A, Padmavathi L, Bhandari R and Swaminathan R (2018). Protein charge transfer absorption spectra: an intrinsic probe to monitor structural and oligomeric transitions in proteins. **Faraday Discussions** in press.

Other publications:

1. Malla AB and Bhandari R (2017). IP6K1 is indispensable for the temporal regulation of mouse spermiogenic proteins. **Cell Biology Newsletter, published by Indian Society of Cell Biology** 36: 38-39.

LABORATORY OF CHROMATIN BIOLOGY AND EPIGENETICS

Understanding functions and regulation of Sirtuin family protein deacetylases

Principal Investigator	Devyani Haldar	Staff Scientist
PhD Students	Lahari Konada Raghavendra Vadla Amrita Sengupta Shalini Aricthota	Senior Research Fellow (till Oct. 2017) Senior Research Fellow (till Dec. 2017) Senior Research Fellow (till Nov. 2017) Senior Research Fellow
Other Members	Nirupama Chatterjee Vasavi Bhogadi Nabila Khan	Technical Officer (till Aug. 2017) Project JRF (since Jan. 2018) BITS trainee (since Jan. 2018)
Collaborators	Manojit Pal	DRILS, Hyderabad

Objectives

Reversible acetylation/deacetylation of proteins regulates numerous important cellular processes. The Sirtuin family NAD⁺ dependent protein/histone deacetylases (HDAC) are conserved from yeast to mammals and carry out a broad range of crucial cellular functions ranging from transcriptional silencing to DNA damage response, cell cycle regulation, metabolism and aging etc. Their molecular functions in DNA metabolic processes such as DNA replication and repair has not been studied extensively. During some of these processes, the expression level of specific sirtuins are known to alter, indicating conditional regulation of these proteins. However, the molecular functions and mechanism of regulation of sirtuins under many of these conditions remain elusive.

Our aim is to understand the molecular functions and mechanism of regulation of sirtuins during DNA damage response and repair. We use yeast and human cell lines as model systems. Based on our findings in yeast, we would like to extend our working hypothesis to mammalian cells. There are seven sirtuins (SIRT1-7) in mammals. The mammalian sirtuins have different sub cellular localization for e.g. SIRT1, SIRT6 and SIRT7 localizes to nucleus, SIRT2 to cytoplasm while SIRT3, SIRT4 and SIRT5 to mitochondria. Besides, a few sirtuins exhibit shuttling between different subcellular compartments and this distinct sub-cellular localization decides their function. Since fission yeast, *S. pombe* is more closely related to higher eukaryotes and sirtuins are conserved from yeast to mammals, we use fission yeast, *S. Pombe* as a model system to study sirtuin biology. Fission yeast, *S. pombe* has three sirtuins, Sir2, Hst2 and Hst4. Deletion

analysis and other studies have shown that all these genes function in transcriptional silencing. However, deletion of only *hst4* gene, not *sir2* and *hst2* genes, show interesting phenotypes such as slow growth, elongated morphology, fragmented DNA and DNA damage sensitivity indicating it could have additional functions. These phenotypes are very useful tools to uncover novel signaling pathways where Hst4 could be functioning. Also, it has been shown to function in maintenance of genome stability. Interestingly, the level of Hst4 decrease when cells are exposed to DNA damage.

We focused on the following objectives:

- 1) Understanding the molecular functions and mechanism of regulation of fission yeast sirtuin Hst4 during DNA damage response.
- 2) Investigation of nuclear localisation and function of human sirtuin 3 (SIRT3).

Project 1: Understanding the molecular functions and regulation of sirtuin family NAD⁺ dependent histone deacetylase Hst4 of fission yeast, *Schizosaccharomyces pombe*.

The expression of Hst4 decreases during the S phase of the cell cycle as well as when cells are exposed to DNA damage. The timely regulation of Hst4 is important for maintenance of genomic integrity. However, the implication of Hst4 degradation, signaling mechanism and the molecular machinery for its degradation on exposure to specific DNA damaging agents such as MMS (Methy methane sulphonate) are not known. This project is aimed at investigating mechanism of regulation of Hst4 during DNA damage stress and also, to gain further insights into the replication stress associated DNA damage pathway in fission yeast.

Summary of work done until the beginning of this reporting year (upto March 31, 2017)

HDACs are known to be regulated in different ways depending on their function. Fission yeast sirtuins regulate genome stability. Our earlier work shows that the level of Hst4 decrease in S phase and in response to DNA damage. Therefore, we checked whether the protein turnover is due to transcriptional or translational regulation and observed little reduction in transcript level. Since the reduction was less than 2 folds, we hypothesized the role of post-translational regulation such as ubiquitination in the degradation of Hst4. In order to check the role of proteasome in the regulation of Hst4, half life of Hst4 was determined in the wildtype and proteasome mutant (*mts2-1*) strain on cycloheximide treatment. The levels of Hst4 were stabilized in proteasome mutant (*mts2-1* strain) significantly as compared to wild type. Further, the levels of Hst4 on DNA damage was checked in the mutant strain. Stabilization of Hst4 was observed in *mts2-1* strain during MMS treatment as compared to wild-type strains. Thus, these results show that Hst4 is regulated by ubiquitin mediated proteosomal degradation. E3 ligases are very important component of the ubiquitination machinery as they specify the substrates targeted for ubiquitination. The SCF ubiquitin ligase is a conserved E3 ligase which regulates the expression of many cell cycle proteins which in turn regulates the G1/S switch. To study the role of SCF ubiquitin ligase in the regulation of hst4, stability of Hst4 protein was determined in SCF mutant strain. Hst4 was stabilized in SCF mutant significantly as compared to wild type. This was comparable to the stability of Hst4 observed in proteosomal mutants. Further, Hst4 is known to be down regulated when cells are exposed to DNA damaging agent MMS. To examine if decrease in level of Hst4 on DNA damage is also mediated through SCF ubiquitin ligase, Hst4 levels were determined in SCF mutant by Western blot. The level of Hst4 did not decrease on MMS treatment in SCF mutant. Further, the degradation of Hst4 was rescued by the plasmid complementation of SCF component back in the null background. Collectively, these results show that Hst4 is regulated by ubiquitin ligase SCF mediated proteolysis in response to DNA damage.

The above result showed the stabilization of Hst4 in the proteasome mutant, therefore, next we wanted to determine whether Hst4 is directly modified by Ubiquitination and targeted

for degradation via proteasome. For this, we used a His-Ubiquitin pull down by Nickel affinity strategy. We overexpressed His-tagged Ubiquitin in the proteasome mutant strain and looked for ubiquitinated Hst4 by western blot after pulling down the His-Ubiquitin using Nickel NTA beads. The experiment was performed with both untreated as well as MMS treatment cells. The higher mobility modified bands of Hst4 being visible in the proteasome mutant strain. Further, we found the bands were enhanced on MMS treatment. This result proves that Hst4 is modified by ubiquitination and thus confirming, its targeted degradation via 26S proteasome. Covalent modification of proteins with ubiquitin plays an important role in a wide array of cellular processes. The E3 ubiquitin ligases are central to determining the timing and specificity of substrate proteolysis. There are two conserved ubiquitin ligases that regulate cell cycle progression: anaphase promoting complex/cyclosome (APC/C) and Skp1-Cdc53/Cullin-1-F-box (SCF). APC/C helps in regulation of G2/M progression and SCF in G1/S transition. Since, Hst4 is highly abundant in G2/M phase and its levels go down in S phase and on treatment with DNA damaging agents that cause replication stress, such as MMS, we hypothesized the role of SCF ubiquitin ligase complex in the regulation of Hst4. SCF ligases are multi-subunit E3 ligases and F box protein component of the complex dictates the specificity by interacting with the phosphorylated substrate. Our results show that Hst4 is stabilized on MMS treatment in both *skp1*(*skp1-94*) and F- box protein mutant (SCF mutant) strains where the components of SCF ligase complex were inactivated. Work is underway to determine whether degradation of Hst4 on DNA damage is phosphorylation dependent as SCF complex recognize phosphorylated substrate proteins and if the degradation of Hst4 is mediated by DNA damage checkpoint proteins.

Details of progress made in the current reporting year (April 1, 2017 - March 31, 2018)

A functional checkpoint is very important for activation of downstream signalling in response to DNA damage to repair the damage. Also, in our previous work, it has been shown that *hst4* and checkpoint kinase *rad3* double mutants are synthetic lethal. (Halder et al., 2008). Rad3 (ATR) is the major checkpoint sensor in *S. pombe*. Therefore, we wanted to check whether Rad3 has any role in the degradation of Hst4 in response to DNA damage. We determined the levels of Hst4 protein in the *rad3* mutant

strain (ROP265) after treating the cells with DNA damage (MMS). The figure 1A shows, there is no change in the levels of Hst4 on MMS treatment in *rad3* mutant confirming that a functional checkpoint is important for mediating the degradation of Hst4. The checkpoint sensor Rad3 acts through phosphorylation of downstream effector kinases and thereby exerts its checkpoint functions. Cds1 (Rad53 homolog) and Chk1 are the DNA replication and DNA damage checkpoint effector proteins respectively in *S pombe*. Since Hst4 is degraded on MMS and HU treatment and that was dependent on Rad3 we wanted to check the role of Cds1 and Chk1 in the degradation of Hst4. The treatments were done as discussed previously and protein levels were detected by Western blot. As shown in figure. 1B and 1C, Hst4 getting degraded on

MMS and HU treatment in *cds1* and *chk1* mutant strain similarly as wild type. We further did a bioinformatics analysis on the Hst4 protein to look for putative kinase sites. We found putative serine/threonine ddk motifs in the C-terminus of Hst4. To confirm the bioinformatics analysis, we checked the levels of Hst4 in DDK deficient strain in *S. pombe*. DDK are major regulators of DNA replication and also mediate intra-S phase checkpoint in response to DNA replication stress. As shown in figure 1D, we found potential stability of Hst4 in *ddk* mutant strain as compared to wild type. These results indicated that degradation of Hst4 on DNA damage is mediated by DNA damage checkpoint proteins and could be phosphorylation dependent. Work is underway to determine whether degradation of Hst4 on DNA damage is phosphorylation dependent.

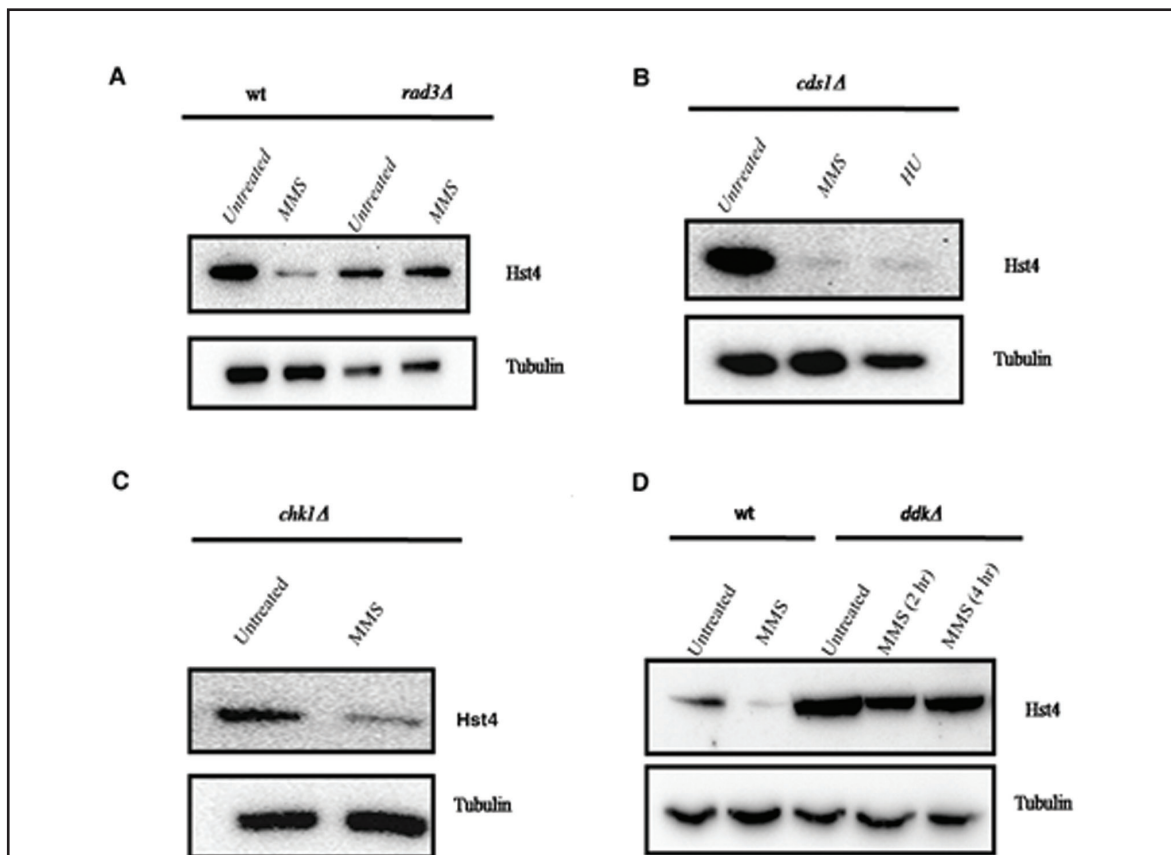


Figure 1. Checkpoint sensor ATR/Rad3 and Dbf4 dependent kinase mediated Hst4 downregulation on DNA damage (A) wild-type and *rad3* mutant strain were grown in rich medium till mid log phase and treated with 0.015% MMS and whole cell extracts were immunoblotted with indicated antibodies. **(B)** *cds1* mutant strains were grown in rich medium till mid log phase and treated with 0.015% MMS and 10 mM HU and whole cell extracts were immunoblotted with indicated antibodies. **(C)** *chk1* mutant strains were grown in rich medium till mid log phase and treated with 0.015% MMS and whole cell extracts were immunoblotted with indicated antibodies. **(D)** wild-type and *ddk* mutant strain were grown in rich medium till mid log phase and treated with 0.015% MMS for indicated time and whole cell extracts were immunoblotted with indicated antibodies.

Project 2: Investigation of nuclear localisation and function of human sirtuin 3 (SIRT3).

Mammalian sirtuins have a conserved HDAC domain and flanking N and C terminal domain. The subcellular localization is regulated by the presence of NES or NLS at either of the N or C-terminal domains, for example, the entry and exit of SIRT1 and SIRT2 into the nucleus is dependent on nuclear localization sequence (NLS) and nuclear export sequence (NES). For instance, SIRT1 on phosphorylation by JNK-1 enters the nucleus, inside the nucleus it has important histone and non-histone substrates, like NF- κ B subunits and histone marks H3K56ac, H3K9ac, H4K16ac etc., while in cytoplasm, it deacetylates acetyl-CoA synthase 1 and hydroxy-3-methylglutaryl CoA synthase 1. Similarly, SIRT2 which is primarily cytoplasmic, moves to the nucleus during mitosis and deacetylates H4K16ac. Human SIRT3 (hSIRT3) is a major mitochondrial deacetylase that deacetylates acetyl-CoA-synthetase (AceCS), glutamate dehydrogenase (GDH), succinate dehydrogenase and complex I functioning in mitochondria. Few reports have shown that the full-length SIRT3 (FL-SIRT3) also localizes to nucleus and functions as transcriptional regulators of nuclear genes regulating metabolic processes in mitochondria. It deacetylates Ku70 and abrogates Ku70-Bax interaction and regulate the transcription of stress related genes as well. In an earlier study, we observed that overexpression of human SIRT3 in HEK cells resulted in reduction of H3K56ac levels, which is a known core domain histone H3 modification. SIRT3, however, was reported to reside mostly in mitochondria but few studies had indicated it could have nuclear functions. Thus, we propose to investigate and decipher novel human SIRT3 interacting proteins in the nucleus and determine its nuclear functions.

Summary of work done until the beginning of this reporting year (upto March 31, 2017)

During an earlier study, we observed that the overexpression of SIRT3 resulted in reduction of H3K56ac levels indicating, it could be a potential substrate for SIRT3. Thus, to confirm the nuclear localization of SIRT3, HeLa cells were treated with Leptomycin B (LMB), which specifically inhibits CRM1 dependent nuclear export, and IF was performed using antibody against SIRT3 at different time points to observe its localization. The increased levels and retention of SIRT3 in the nucleus was observed in a time dependent manner, 120mins showed the maximum

retention inside the nucleus. The NES containing proteins are exported to the cytoplasm in a CRM-1 dependent manner and this export is inhibited by treatment with LMB. Since, SIRT3 retained inside the nucleus on LMB treatment, therefore, we checked the presence of NES sequence in the SIRT3 protein using NES prediction software (Net NES1.1 Server). The predicted NES with a score above 0.5 were selected and aligned with previously known similar NES containing proteins. The NES was predicted to be present between the amino acids 314 to 324 of SIRT3 and contains a cluster of hydrophobic amino acids. To map the SIRT3 NES, a GFP-tagged SIRT3 deletion construct lacking the C-terminal region (amino acid 314-399) was generated. The wild type SIRT3 and the deletion constructs were overexpressed in HeLa cells by transient transfection and the percentage of transfected cells with nuclear SIRT3 were counted. Nearly 94% of cells overexpressing (NES Δ 314-399) showed nuclear retention. Next, to identify the hydrophobic residues crucial for NES function, the first three leucine residues in the predicted NES were mutated to alanine [(L315A), (L315, 316A) and (L315, 316, 318A)] using site-directed mutagenesis. The GFP tagged mutation constructs were generated and the GFP expression was quantified as; percentage of cells expressing mutant SIRT3 in cytoplasm alone (%C), in nucleus alone (%N) and both in cytoplasm and nucleus (%C+N). The SIRT3 mutants (L315A) and (L315, 316A) exhibited similar localization with ~ 60 % of cells showing both cytoplasmic and nuclear localization. However, 94% of SIRT3 mutant (L315, 316, 318A) was detected in nucleus alone, indicating amino acids 315-324 contains the NES. These results confirm presence of NES in SIRT3, disruption of which restrict it in the nucleus. Overall, these results demonstrate that a novel NES dependent shuttling of SIRT3 from the nucleus to cytoplasm.

Details of progress made in the current reporting year (April 1, 2017 - March 31, 2018)

Previously, we have observed that overexpression of SIRT3 in HEK 293 cells decreased the level of H3K56ac, indicating acetylated histone H3K56 could be a substrate of this deacetylase. Therefore, to investigate whether SIRT3 deacetylates H3K56, SIRT3 wild-type (SIRT3WT) and catalytic mutant (SIRT3H248Y) constructs were overexpressed in 293 cells and Western blot was performed using H3K56ac antibody. The SIRT3WT deacetylated H3K56, however, SIRT3H248Y mutant failed to

deacetylate. Additionally, the H3K56ac levels were determined using immunofluorescence (IF) studies on overexpression of GFP-SIRT3WT and SIRT3H248Y constructs in 293 cells. The GFP-SIRT3WT was able to deacetylate H3K56, while SIRT3 catalytic mutant (SIRT3H248Y) could not. To further confirm the deacetylation of H3K56 by SIRT3, siRNA mediated knock down of SIRT3 was performed in HEK293 cells and the levels of the acetylation was detected by Western blot. Knocking down SIRT3 showed an increase in the levels of H3K56ac (Sengupta A and Haldar D, 2018). These results show that H3K56ac is a novel substrate of SIRT3 *in vivo*.

The acetylation of H3K56 is required for cell survival on DNA damage and SIRT3 overexpression protects cells from cell death on genotoxic stress. Therefore, we next investigated whether SIRT3 functions in DNA damage response pathway via deacetylating H3K56. We treated U2OS cells with methyl methanesulphonate (MMS) in a concentration dependent manner to induce DNA damage via alkylation. Immunofluorescence studies using antibodies against both SIRT3 and H3K56ac, showed a significant concentration dependent increase in levels of SIRT3 on MMS treatment. Concurrently, we observed a significant decrease in the levels of H3K56ac, which was represented by measuring the nuclear intensity of both H3K56ac and SIRT3 using Zen software (Figure 2A,2B). Significantly, we observed that SIRT3 formed nuclear foci in response to DNA damage, indicating it may function in DNA repair and promote cell survival in MMS. The treatment of U2OS cells with MMS induced formation of SIRT3 foci in the nucleus. To test whether these foci colocalize with repair foci, we monitored the localization of SIRT3 with DNA damage marker, γ H2AX on MMS treatment by immunofluorescence. We observed colocalization of γ H2AX with SIRT3 in presence of MMS (Figure 2C). The colocalization coefficient was measured from the scatter plots using ZEN software (Figure 2D). To test whether SIRT3 is required for cell survival

on DNA damage, siRNA mediated knockdown of SIRT3 was carried out and the viability of the U2OS cells was determined by colony formation assay in the presence and absence of MMS. SIRT3 knock down cells formed 2-fold less colonies as compared to the control, indicating it is required for cell survival on DNA damage, and represented by plotting absolute number of colonies formed on SIRT3 knockdown in presence of absence of MMS (Figure 2E,2F). To further confirm the effect of absence of SIRT3 on cell viability, we determined the percentage of cell survival on siRNA mediated knock down of SIRT3 using Annexin V and PI staining. The reduction in cell survival indicates, in absence of SIRT3 around 1.5-fold increase in apoptotic cells population, which was represented by quantifying the number of apoptotic cells using FACS on SIRT3 knockdown in presence of absence of MMS (Figure 2G, 2H). Overall, these results indicated SIRT3 localizes to DNA repair foci in response to DNA damage and promotes cell survival.

Publications

Research paper

1. Amrita Sengupta and **Devyani Haldar** (2018) Human Sirtuin 3 (SIRT3) deacetylates histone H3 lysine 56 to promote Nonhomologous end joining repair. **DNA Repair** 61; 1-16.
2. Raghavendra Vadla and **Devyani Haldar** (2018) Mammalian target of rapamycin complex 2 (mTORC2) controls glycolytic gene expression by regulating Histone H3 Lysine 56 acetylation. **Cell cycle** 17:110-123.
3. A novel SIRT1 inhibitor, 4bb induces apoptosis in HCT116 human colon carcinoma cells partially by activating p53. Ghosh A, Sengupta A, Seerapu GPK, Nakhi A, Shivaji Ramarao EVV, Bung N, Bulusu G, Pal M, Haldar D. **Biochem Biophys Res Commun**. 2017 Jul 1;488(3):562-569.

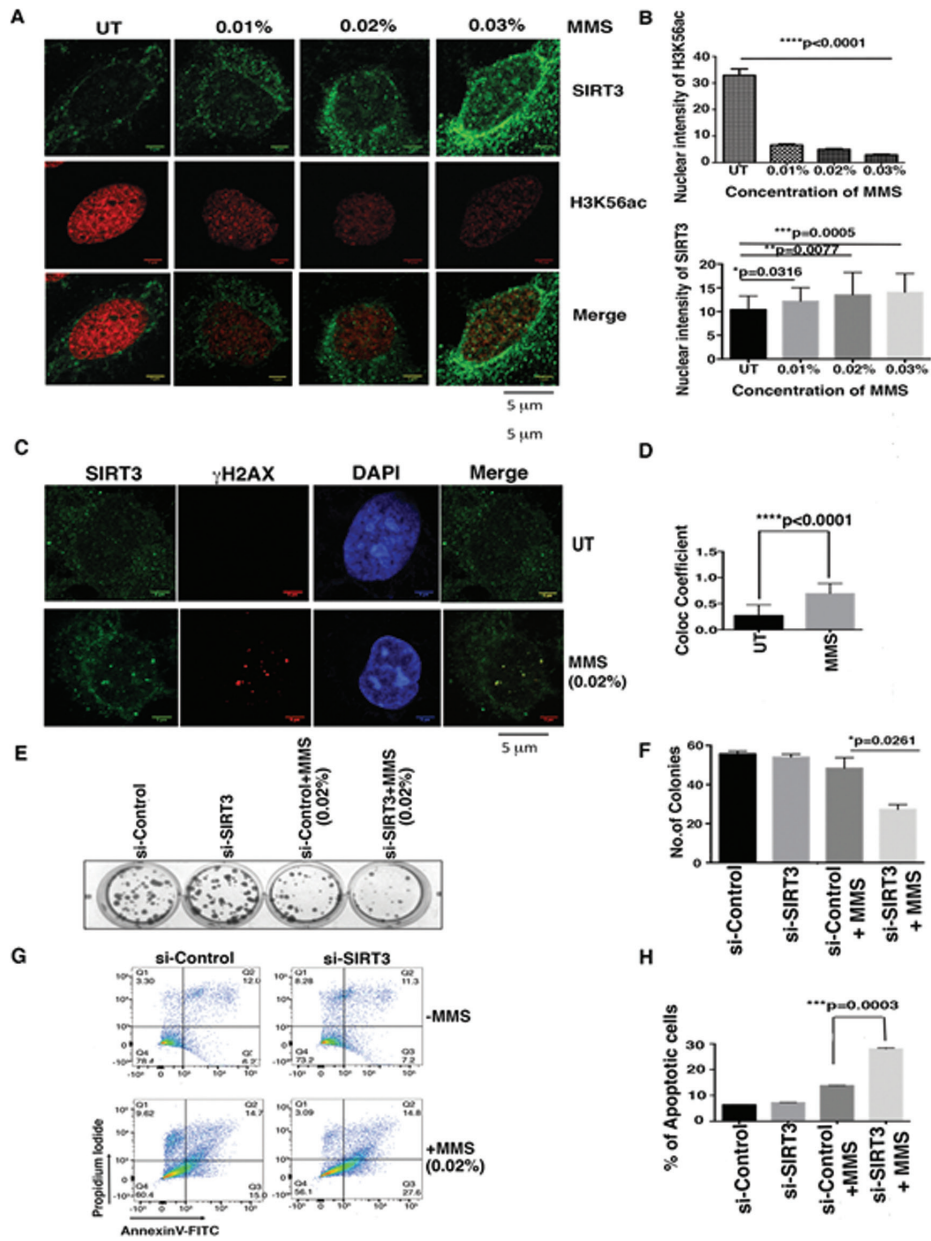


Figure 2. SIRT3 promotes cell survival on DNA damage. (A) Representative IF image of SIRT3 deacetylating H3K56ac, the cells were treated with MMS for 2hrs with concentrations as indicated, and stained with H3K56ac (red) and SIRT3 (green) antibody, and imaged using a confocal microscope. (B) Nuclear intensity per cell was calculated using ZEN imaging software and plotted using Graph pad prism. Approximately 150 cells were calculated, n=3, unpaired t-test was performed to find out the statistical significance. (C) Representative IF image of SIRT3 colocalization with γH2AX, the cells were treated with MMS for 2hrs as indicated concentration, fixed and stained with γH2AX (red), SIRT3 (green) antibody and counterstained with DAPI (blue), and observed under confocal microscope. (D) Plot representing quantification of colocalization coefficient between SIRT3 and γH2AX of 200 cells, n=3. (E) Representative image of colony formation of si-Control and si-RNA knock down U2OS cells on MMS treatment. (F) Quantification of number of colonies formed in si-Control and si-RNA mediated knock down of SIRT3 in U2OS cells in absence and presence of MMS treatment, Unpaired t-test was performed to find out the significance between Scrambled and si-RNA knock down SIRT3 up on MMS treatment n=3. (G) Representative image of dot plot of si-Control and si-RNA knock down U2OS cells on MMS treatment. (H) Quantification of percentage of apoptotic cells in si-Control and si-RNA mediated knock down of SIRT3 in U2OS cells in presence and absence of MMS. Unpaired t-test was performed to find out the statistical significance between si-Control and si-RNA knock down SIRT3 up on MMS treatment n=3.

LABORATORY OF COMPUTATIONAL AND FUNCTIONAL GENOMICS

Computational and functional genomics of biological organisms

Principal Investigator	Akash Ranjan	Staff Scientist
Ph D Students	Mr. Rohan Misra	Senior Research Fellow
	Mr. Ajit Roy	Senior Research Fellow (till June 2017)
	Mr. Rajendra Kumar Angara	Senior Research Fellow
	Mr. Abhishek Kumar	Senior Research Fellow
	Mr. Debasish Kumar Ghosh	Senior Research Fellow
	Mr. Shailesh Kumar Gupta	Senior Research Fellow
	Mr. S. Akshaykumar Nanaji	Junior Research Fellow
Other Members	Dr. U S Raghavender	SERB-DST Young Scientist (till June 2017)
Collaborators	Anthony Adlagatta	CSIR-IICT Hyderabad, India
	V. Vindal	University of Hyderabad, Hyderabad, India.
	V. K. Mishra	CDFD, Hyderabad, India.

Objectives

The primary research objective of our group is to understand structure and function of regulatory genes, encoded in various genomes, and their products that are required to coordinate important cellular functions. We use a combination of computational and experimental approach to achieve our goal.

Project 1

Characterization and functional studies of transcription regulators

Summary of work done until the beginning of this reporting year (upto March 31, 2017)

There are five proteins (Rv0043c, Rv0165c, Rv0494, Rv0586 and Rv3060c) annotated as Fatty acid degradation regulator (FadR) family of proteins in *M. tuberculosis*. We have characterised the transcriptional regulatory role of Rv0494 together with its promoter organisation and binding sites. Further, we demonstrated Rv0494 as lipid responsive, and starvation inducible auto-regulator.

Details of the work done in the current reporting period (April 1, 2017 - March 31, 2018)

In the current study, we have tested which one of the five FadR proteins from *M. tuberculosis* is the functional homologue of *E. coli* FadR? *E. coli* with a functional copy of *fadR* is unable to utilize medium chain fatty acids (< 12 carbon chain-length) such as decanoic acid as a sole source of carbon due to the inability of such lipids to de-repress the fatty acid regulon. However,

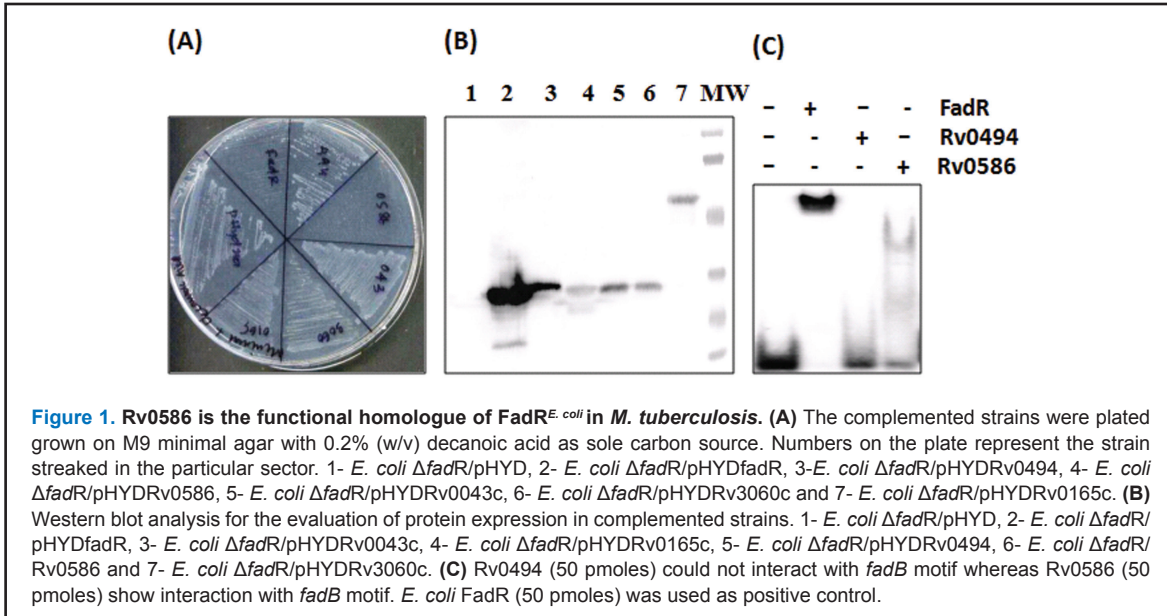
the $\Delta fadR$ strains are able to utilize such fatty acids because of the constitutive expression of *fad* regulon genes required for fatty acid catabolism. This strategy was applied to identify the functional homolog of *E. coli fadR* among the five *fadR* paralogs encoded by *M. tuberculosis* genome. The derivatives of *E. coli* $\Delta fadR$ with various *M. tuberculosis* FadR homologues were tested for their ability to grow on M9 minimal media containing decanoic acid as a sole carbon source. *E. coli* $\Delta fadR/pHYDfadR$ and *E. coli* $\Delta fadR/pHYDRv0586$ strains have failed to show any significant growth while other strains show significant growth (Figure 1A). The construct pHYD*fadR* serves as a positive control, where *fadR* gene from *E. coli* strain MC4100 was cloned in pHYD3025 and used to transform *E. coli* $\Delta fadR$. We further confirmed the expression of proteins in all strains using western blot analysis (Figure 1B). The results of this experiment indicated that among all the FadR paralogs present in *M. tuberculosis*, only Rv0586 complemented *E. coli* $\Delta fadR$ and is presumably repressed the *fad* regulon, due to which decanoic acid was not utilized as a carbon source.

FadR^{*E. coli*} is known to interact with *fadB* gene operator site, which play an important role in the fatty acid degradation (Dirusso et al, 1992). In order to test the ability of Rv0494 and Rv0586 to interact with *fadB* gene upstream sequence, we tested the binding capacity to the two proteins to *fadB* motif (5' TCTGGTACGACCAGA 3'; recognized by FadR^{*E. coli*}). As expected, the recombinant FadR^{*E. coli*} showed strong interaction to the *fadB* motif (Figure 1C); Rv0494 did not

show any interaction with *fadB* motif, while Rv0586 showed significant interaction with *fadB* motif (Figure 1C). The interaction of Rv0586 with *fadB* motif supports the possible repression

of the fatty acid degradation pathway and complementation ability in *E. coli* Δ *fadR* similar to FadR^{*E. coli*}.

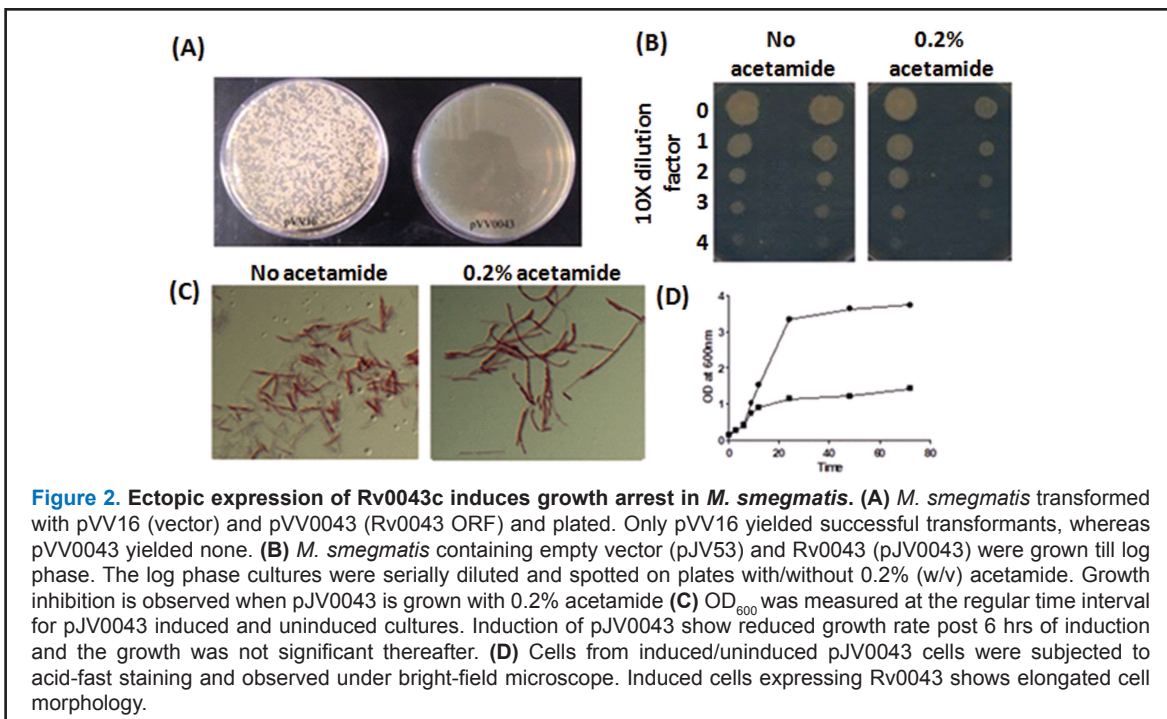
Characterisation of *M. tuberculosis* Rv0043c



protein

Rv0043c is a protein belonging to the FadR family of proteins. In the current reporting year, we have shown that Rv0043c protein's expression has growth inhibitory effect on *M. smegmatis*. The constitutive expression constructs of Rv0043c

by pVVRv0043 vector produced no viable transformants of *M. smegmatis* (Figure 2A). Induced expression of Rv0043c, by acetamide inducible system (pJV0043), was also found to be growth inhibitory on solid medium (Figure 2B). Further, the growth curve analysis



suggested that the Rv0043c expression caused slower growth rate from 6 hrs of post-induction, finally leading to total growth arrest after 24hrs of induction (Figure 2C). The maintenance of steady absorbance suggested that the growth-arrested cells were intact/alive without any lysis. Observation of uninduced and induced cells in bright-field microscopy for morphological differences, it was observed that the growth-arrested cells with Rv0043c expression were elongated compared to the uninduced cells (Figure 2D). However, the Rv0043c expression did not change the acid-fastness of growth-arrested *M. smegmatis* cells (Figure 2D).

Project 2

Molecular characterization of *Plasmodium falciparum* acyl-CoAs binding proteins

Summary of work done until the beginning of this reporting year (upto March 31, 2017)

In our previous studies, we had characterized the biophysical and the lipid binding properties of the three *Plasmodium falciparum* acyl-CoAs binding proteins (*pf*ACBPs), naming ACBP16, ACBP99 and ACBP749. We had shown that the *pf*ACBPs are globular proteins that are composed of mainly α -helical structures. These

*pf*ACBPs could bind to long-chain fatty acyl-CoAs, like myristoyl-CoA, and conjugated lipids, like phosphatidylcholine. However, the *pf*ACBPs could not bind the fatty acids (like palmitic acid) and phosphatidic acid. All the *pf*ACBPs showed high sequence conservation and two specific tyrosine residues (Y30 and Y33) were identified as the critical residues that determined the interaction stability with the fatty acyl-CoAs.

Details of progress made in the current reporting year (April 1, 2017 - March 31, 2018)

In the current reporting year, we have conducted studies to find the small molecule modulators of *pf*ACBPs. To identify the potential binding molecules, we used high-through virtual screening of the chemicals of Pubchem and Tres Cantos antimalarial compound set (TCAMS) against the modelled structures of ACBP16, ACBP99 and ACBP749. We identified several small molecules that had the ability to interact with *pf*ACBPs with affinity. The FDA approved drug, mefloquine, appeared as one promising candidate. We confirmed the high-affinity binding of mefloquine with *pf*ACBPs by isothermal titration calorimetry experiments (Figure 3A). Mefloquine appeared to be a competitive inhibitor for *pf*ACBPs against their substrates, like myristoyl-CoA. Mefloquine

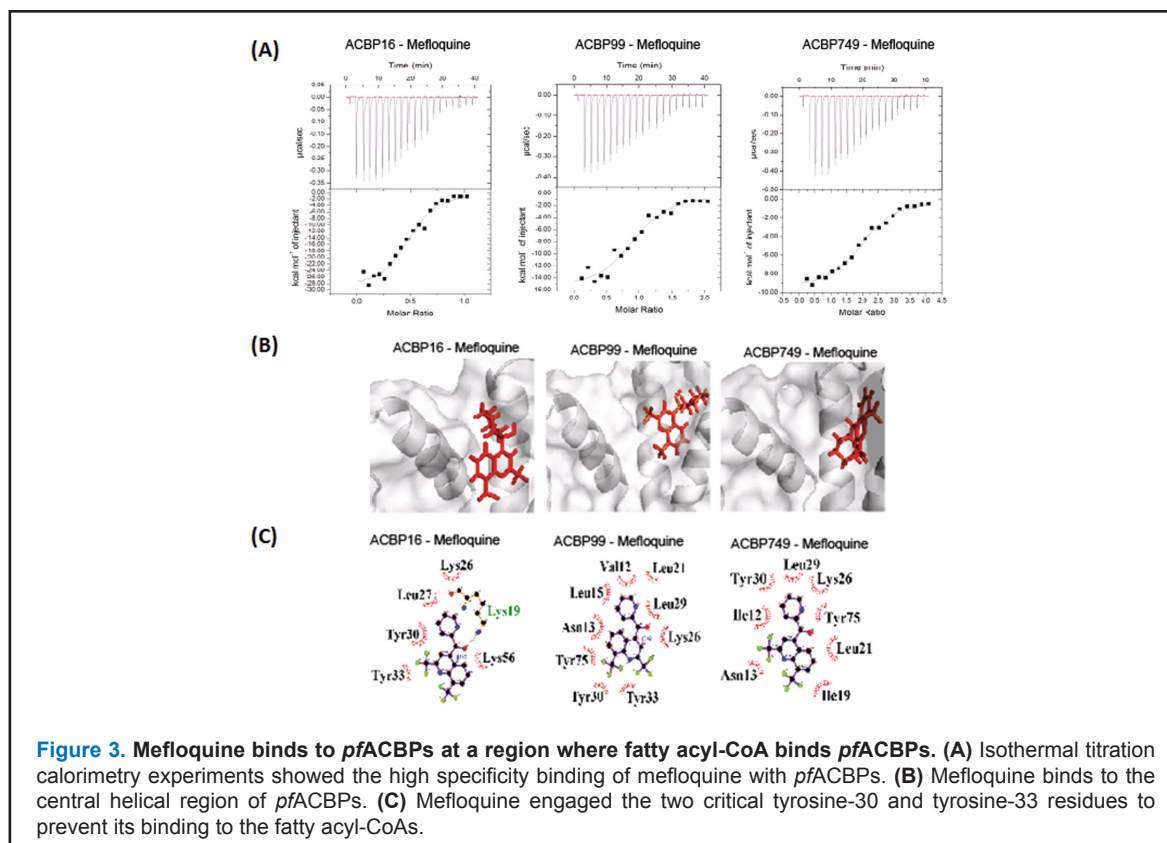


Figure 3. Mefloquine binds to *pf*ACBPs at a region where fatty acyl-CoA binds *pf*ACBPs. (A) Isothermal titration calorimetry experiments showed the high specificity binding of mefloquine with *pf*ACBPs. **(B)** Mefloquine binds to the central helical region of *pf*ACBPs. **(C)** Mefloquine engaged the two critical tyrosine-30 and tyrosine-33 residues to prevent its binding to the fatty acyl-CoAs.

engaged and blocked the essential Y30 and Y33 to prevent the fatty acyl-CoA binding to *pf*ACBPs (Figure 3B, 3C). Mefloquine showed high toxicity against the *P. falciparum*, resulting in slower multiplication and increased death of the organism in presence of mefloquine.

Project 3

Functional studies on the homeostasis circuits of aggregation-prone proteins

Summary of work done until the beginning of this reporting year (upto March 31, 2017)

In the previous studies, we have identified as Huntingtin interacting protein K (HYPK) as a global sensor and regulator of aggregation-prone proteins, like poly-glutamine expanded Huntingtin-exon1 (Htt97Qexon1), α -Synuclein-A53T and SOD1-G93A. HYPK can specifically sequester aggregation-prone proteins, but not the non-aggregating proteins, both *in vitro* and in neuron cells. HYPK itself is an aggregating protein and it can form different kinds of supra-molecular structures, like amorphous and annular aggregates. The self-aggregation of

HYPK follows prion-like seed nucleation type oligomerization. We also established the fact that HYPK was involved in the neddylation dependent autophagy of proteinaceous aggregates. We determined the complete mechanism of neddylation dependent autophagy. We identified that ectopic expression of Nedd8 induced and maintained the autophagy. Poly-neddylation of Htt-exon1 could deliver the aggregates to autophagy vacuoles. Lysine-60 type poly-neddylation linkage of the lysine-15 residue of Htt-exon1 led to the autophagic degradation of Htt-exon1.

Details of progress made in the current reporting year (April 1, 2017 - March 31, 2018)

In the current period of study, we have identified the process by which HYPK enhances the autophagic degradation of poly-neddyated huntingtin exon 1 (Htt exon1) protein aggregates (Figure 4). HYPK acted as a scaffolding protein which tethered to the poly-neddyated proteins by its UBA domain, while also interacted with LC3 with its tyrosine-type (Y-type) LC3 interacting region. HYPK bridged the localization of LC3

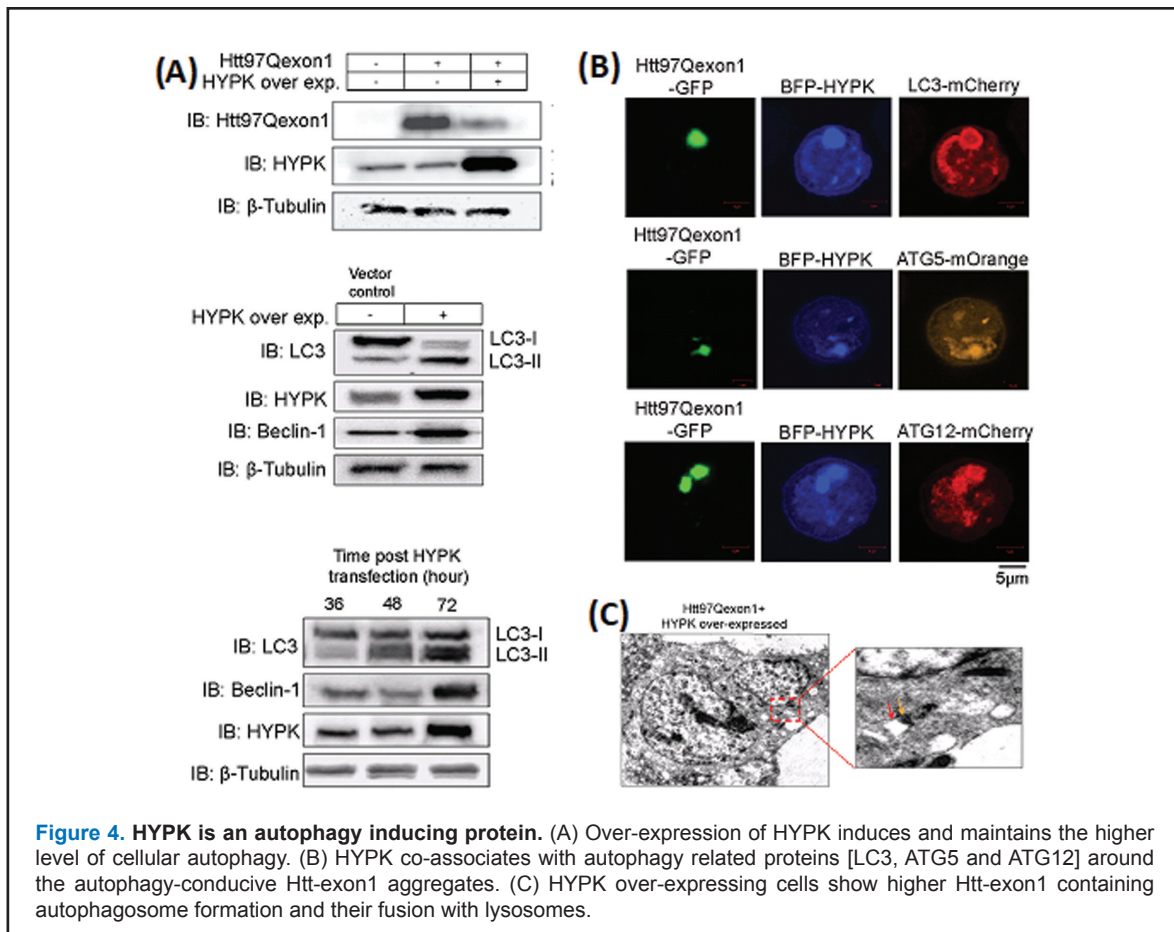


Figure 4. HYPK is an autophagy inducing protein. (A) Over-expression of HYPK induces and maintains the higher level of cellular autophagy. (B) HYPK co-associates with autophagy related proteins [LC3, ATG5 and ATG12] around the autophagy-conducive Htt-exon1 aggregates. (C) HYPK over-expressing cells show higher Htt-exon1 containing autophagosome formation and their fusion with lysosomes.

to the poly-ubiquitinated Htt-exon1 aggregates to initiate the formation of pre-autophagosome complex. Over-expression of HYPK could lead to increased basal level of cellular autophagy. HYPK showed the ability to reduce the toxicity exerted by the huntingtin exon 1 protein aggregates and helped in cell survival/maintenance of cell physiology.

Publications

Research papers published in 2017-2018

1. Angara RK, Yousuf S, Gupta SK, Ranjan A (2018). An IclR like protein from mycobacteria regulates leuCD operon and induces dormancy-like growth arrest in

Mycobacterium smegmatis. *Tuberculosis*, 108: 83-92.

2. Ghosh DK, Roy A, Ranjan A (2018). Aggregation-prone regions in HYPK help it to form sequestration complex for toxic protein aggregates. *Journal of Molecular Biology*, 430: 963-986.
3. Ghosh, DK, Roy A, Ranjan A (2018). Disordered nanostructure in Huntingtin Interacting Protein K acts as a stabilizing switch to prevent protein aggregation. *Biochemistry*, 53(13): 2009-2023.

LABORATORY OF *DROSOPHILA* NEURAL DEVELOPMENT

Understanding patterning and development of Central Nervous System using
Drosophila melanogaster

Faculty	Rohit Joshi	Staff Scientist
Ph D Students	Risha Khandelwal	Senior Research Fellow
	Neha Ghosh	Senior Research Fellow
	Ravi Ranjan	Senior Research Fellow
	Rashmi Sipani	Senior Research Fellow
	Asif Ahmad Bakshi	Senior Research Fellow
	Yamini Rawal	Junior Research Fellow (Since Feb. 2018)
Other Members	Chandra Shekhar Singh	Technical Assistant
	Bijaylaxmi Swain	Project Assistant (till Sep. 2017)

Objective

The key objective of the lab is to understand how neural progenitor cells generate a variety of different cell types and cell numbers in developing Central Nervous System (**CNS**) of an organism. Hox family of transcription factors are known to play an important role in execution of these features along the Anterior-Posterior (**AP**) axis of the CNS during development. Our lab is using *Drosophila melanogaster* as a model organism, to understand these phenomena by focusing mainly on early embryonic and larval stages of development. To this end, the specific aims of our lab are as follows:

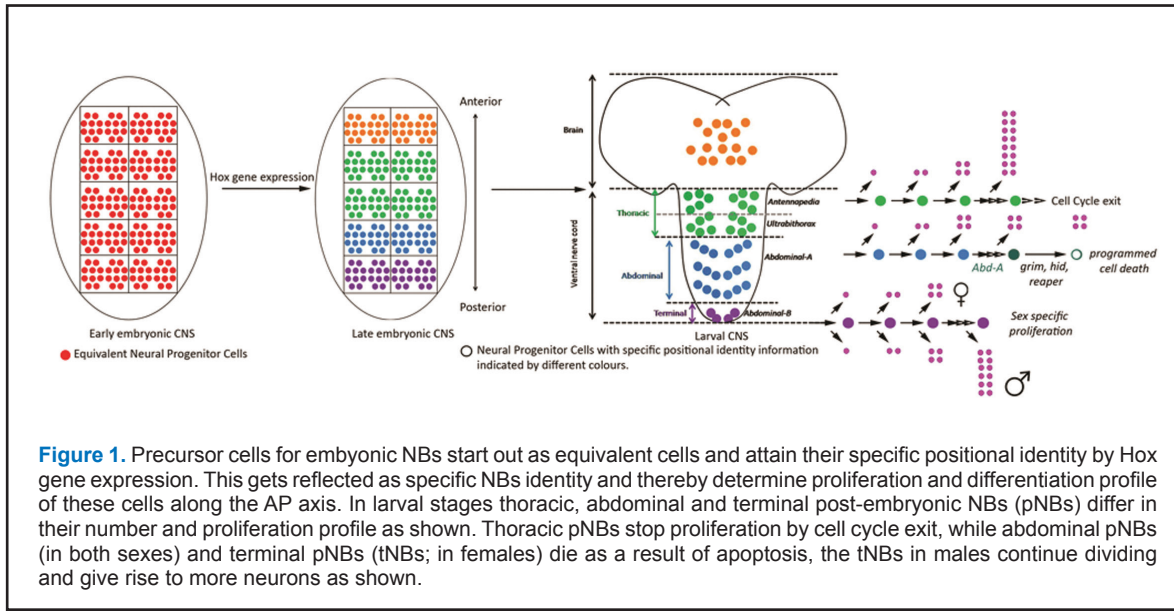
1. Understanding the molecular function of Hox gene *Abdominal-A (Abd-A)* in larval CNS patterning.
 2. Understanding the role of Hox gene *Deformed (Dfd)* in patterning of larval subesophageal ganglia.
 3. Investigating the role of Abdominal-B (*Abd-B*) and Double-sex (*Dsx*) in terminal CNS patterning.
- 1. Understanding the molecular function of Hox gene *Abdominal-A (Abd-A)* in larval CNS patterning.**

Drosophila CNS comprise of two optic lobes, brain and ventral nerve cord (VNC). The

molecular basis of role of Hox genes in patterning VNC of the CNS is not well investigated. Abdominal region of the *Drosophila* larval CNS has a less number of neurons compared to its thoracic counterpart. Hox gene *Abd-A* is known to cause programmed cell death (apoptosis) of neural progenitor cells (also called Neuroblasts-**NBs**) and therefore limit the number of neurons in abdominal region of CNS (Figure 1). The apoptosis is known to be mediated through activation of *reaper*, *hid* and *grim* (**RHG**) family of genes. The precise molecular details of how *Abd-A* cause NB apoptosis are unknown. Genetic evidence suggests a role for a helix-loop-helix transcription factor Grainyhead (*Grh*) along with *Abd-A* in control of this apoptosis. Characterization of the molecular basis of this link is primary goal of this project. Furthermore, since *Grh* is involved in NB apoptosis and is not expressed in neuronal progeny refractory to this apoptosis, it is of interest to define *grh* regulation in these cells which keeps *grh* "on" in the NBs and "off" in the neuronal progeny of NBs.

Summary of work done until the beginning of this reporting year (1 April 2016-31 March, 2017).

The relevant enhancer for the activation of **RHG** family of apoptotic genes in NBs lies within 23kb genomic region referred to as **NBRR-Neuroblast Regulatory Region**. We had narrowed down



the apoptotic enhancer region down to 717 bp. Enhancer was also genetically isolated by generating a smaller deletion (*NBRR-22*) of 14.5 kb which in transheterozygotic combination with already existing 54Kb deletion of *NBRR* called *MM3* (Figure 3) gives ectopic NBs in the abdominal region of CNS at late third instar stage (LL3 stage). We tested and found (by RNA interference and ectopic expression) that apoptotic *enhancer-lacZ* line is responsive to Abd-A, Grh and Notch. We also find that Notch signaling has a direct role transcriptional activation of RHG genes in abdominal NBs instead of activating pulse of AbdA as reported earlier. Subsequently, potential Hox, Exd and Grh binding sites were identified in 717bp enhancer and tested for binding by EMSA. The *in vivo* relevance of these binding motifs was also to be assessed by testing the capacity of reporter expression by enhancer mutagenized for these binding sites.

Considering the importance of Grh in NBs we are trying to identify *grh* regulators in larval NBs. We had narrowed down 4kb enhancer of *grh* responsible for its expression in CNS by sub-fragmentation to 600 bp. Currently this region is being further analysed to identify transcriptional factors that could be regulating *grh* differentially in NBs versus neurons. A set of 465 transcription factors were knocked down by RNA interference in abdominal and thoracic NBs to identify regulator of *grh* gene by scoring for downregulation of Grh protein expression, we could not identify any regulator of *grh* gene from this screen. This indicates a possibility that

grh has a complex transcriptional regulation with no one specific factor being critical to regulate its expression in CNS. Congruent to this some of the preliminary experiments suggest possibility of additional *grh* enhancers.

Details of progress made in current reporting year (1 April, 2017-31 March, 2018)

Since Extradenticle (Exd) and Homothorax (Hth) are the HD containing cofactors employed by Hox genes under various cellular contexts, we checked their role in larval abdominal NB apoptosis by use of genetic mutants and RNA interference mediated knockdown of the genes. Hth has been known to be critical for nuclear localization of Exd. Interestingly, we observed that while Exd plays an important role in larval NB apoptosis but Hth is not critical for the same. This suggests that there is a Hth independent mode of nuclear localization of Exd protein, this is currently being investigated.

Next we tested the importance of the Hox-Exd and Grh binding motifs *in vivo* by mutagenizing them in the enhancer, and testing the its capacity to activate *lacZ* reporter in larval NBs. In first construct, Grh binding sites in all 8 motifs (present in 717 bp) were mutagenized leaving Hox-Exd binding sites intact (*717-Grh^{mutant}-lacZ*). In the second construct Hox-Exd and Grh binding sites across all the 8 motifs were mutagenized (*717-Hox-Exd-Grh^{mutant}-lacZ*). In third construct, we identified and mutagenized all 7 recognizable Su(H) binding sites (RTGRGAR). We found that reporter *lacZ* expression in abdominal NBs was abrogated in all the three mutant versions of the

enhancer in late L3 stage, while it was normal in early L3 stage. This suggested that mutagenized motifs play a crucial role in sustenance of the expression of the apoptotic genes and are not critical for initiation of their expression in early stages.

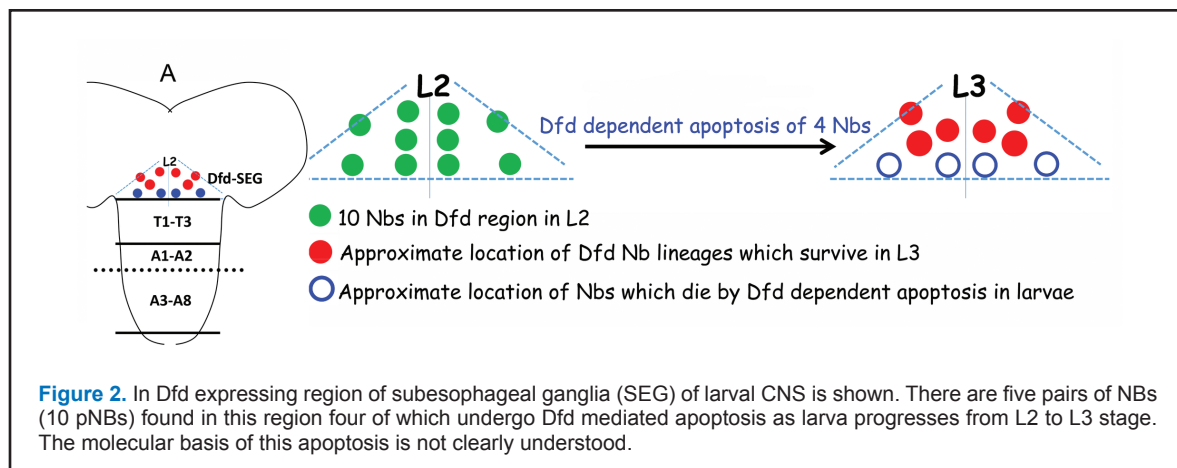
In our analysis with *717-Su(H)^{mutant}-lacZ*, we found that its expression in NBs was slightly delayed in early larval stages, but in late L3 stage, like other mutant *enhancer-lacZ* lines, its expression was completely missing from NBs. This implied that Notch signaling along with Hox, Exd and Grh has a direct role in NB apoptosis and Notch signaling specifically has a role in determining the timing of apoptosis initiation and most importantly in maintenance of the enhancer activity during this apoptosis.

Since our results suggest that above mentioned motifs are important for maintenance but not initiation of the enhancer activity, DNA motifs

necessary for enhancer initiation are being identified to understand their role along with Notch signaling in apoptotic enhancer firing.

2. Understanding the role of Hox gene *Deformed (Dfd)* in patterning of larval subesophageal ganglia.

Hox genes express in neural progenitor cells of CNS during embryonic stages of development (as represented in Figure 1) but how does their expression patterns the nervous system is not well understood. *Deformed (Dfd)* is known to express in the cells of maxillary (Mx) and mandibular (Mn) segments of subesophageal ganglion (SEG) of embryonic and larval CNS (Figure 2). Gnathal segment of embryonic CNS give rise to part of subesophageal ganglia (SEG) of larval CNS (which expresses Hox proteins *Dfd*, *Scr* and *Antennapedia*). This project focuses on understanding role of *Dfd* in NB apoptosis in larval SEG.



Summary of work done until the beginning of this reporting year (1 April 2016-31 March, 2017).

In larval SEG 36 NBs have been reported (18 segmental pairs) in second instar larval (L2) stage. Out of these 36 NBs, 10 NBs (5 pairs) are found in *Dfd* expressing region of SEG (also referred to as *Dfd*-SEG). Four out of these 10 NBs undergo *Dfd* mediated apoptosis as larva progresses from L2 to L3 stage (Figure 2). We tested and found *Grh* to be expressed in NBs found in *Dfd*-SEG in all the cells in early L2 stage and 6 remaining NBs in late L3 stage. Interestingly, we observed that Hox and *Grh* code for NBs (*Grh⁺/Hox⁻*) and associated progeny (*Grh⁻/Hox⁺*) in a lineage was same in *Dfd*-SEG as well as in abdominal region of CNS.

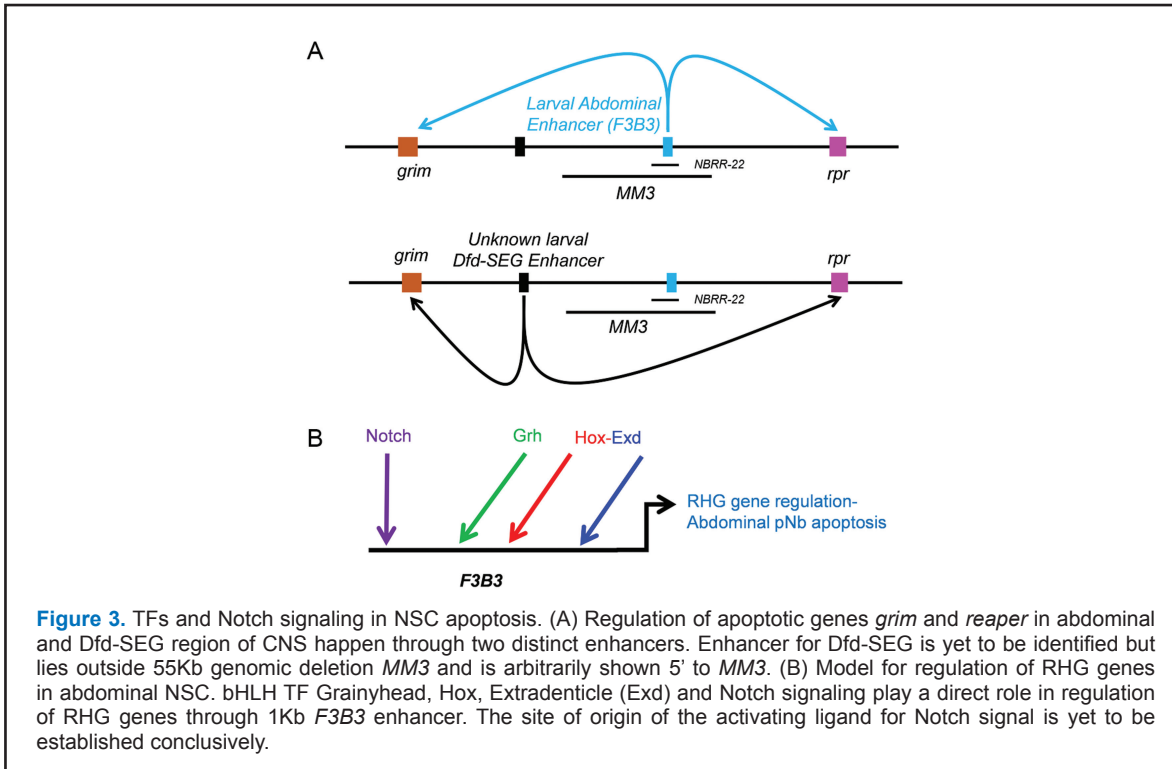
This suggest that like in abdominal NBs *Dfd*-SEG apoptosis may be dependent on *Grh*, and is triggered by change in Hox/*Grh⁺* state of NB to Hox⁺/*Grh⁺* state. This prompted us to test the functional role of *Grh* in apoptosis of 4 NBs in *Dfd*-SEG during development.

Details of progress made in current reporting year (1 April, 2017-31 March, 2018)

We tested the functional role of *Grh* by knocking down its expression in larval CNS using specific genetic mutant combination and RNA interference. Congruent to the idea we observed ectopic NB lineages in late L3 VNCs. The numbers of lineages were in agreement with the fact that there are 10 NBs reported in *Dfd*-SEG of wild type CNS in L2 stage, of which 4 undergo apoptosis by early L3 stage. These results show that similar to its role in abdominal

segments, Grh also plays an important role in NB apoptosis in Dfd-SEG region. In order to identify the genomic location of the enhancer responsible for apoptosis of 4 NBs in Dfd-SEG, we counted the number of ectopic NBs in this region in late L3 stage for various deletion combinations for *NBRR*. We could recover

only 6 NBs in Dfd-SEG region in case of trans-heterozygotic combination of genomic deletions *M22/MM3*, *MM3/MM3* (Figure 3), implying that enhancer responsible for activation of apoptosis of NBs in Dfd-SEG is different from abdominal apoptotic enhancer and lies outside 22Kb *NBRR* and 54Kb genomic region deleted in *MM3* allele.



3. Investigating the role of *Abdominal-B (Abd-B)* and *Double-sex (Dsx)* in terminal CNS patterning.

AbdB expresses in the terminal region of VNC. In larval stages 12 NBs are reported in this region, 8 of these have been suggested to stop dividing in both males and females at mid L3 stage of development. The remaining 4 NBs which we refer to as sex-specific terminal NBs (**tNBs**) express transcription factor Doublesex (*Dsx*). These *Dsx*⁺ tNBs die in females in early larval stages and continue dividing in males till late larval stages, giving rise to male specific neurons. *Dsx* is the most downstream member of sex specification hierarchy and has a male and female specific isoform. The hypothesis for this part of work is that *Abd-B* and *Dsx* play a role in sex specific proliferation and apoptosis of these tNBs. Although the role of the sex determining hierarchy and Hox gene *Abd-B*, in growth and differentiation of *Drosophila* genital discs is worked out, little is known about the mechanism

of the same and how sex determination hierarchy and *Abd-B* intersects with cell proliferation and survival behavior of tNBs in the larval VNC. We intend to test the interaction between *Abd-B* and *Dsx* in gender specific proliferation and apoptosis of these cells.

Summary of work done until the beginning of this reporting year (1 April 2016-31 March, 2017).

It has been reported that female specific isoform of *Dsx* (*Dsx*^F) is responsible for the apoptosis of sex-specific tNBs in females, while these cells continue dividing in males. The molecular mechanism behind the phenomenon of apoptosis in females and how *Dsx*^M play a role in tNB proliferation in males is not known so far. It also needs to be investigated how sex specific tNBs are different from other 8 NBs in the same region which stop dividing at mid L3 stage of development.

We have found so far that *Abd-B*, *Grh* and *Dsx* express in tNBs in CNS of both male and female

larvae. In our analysis with *grh* mutants we found ectopic NB in the Abd-B region of female (as well as male) larval CNS compared to controls where no NBs are reported at the same stage. Interestingly none of these cells were found to be positive for Dsx (a marker for sex-specific tNBs). This suggests that apoptosis of Dsx+ tNBs in females is independent of Grh. A similar analysis with single mutants of *grim* and *reaper* (members of *RHG* family of apoptotic genes) also did not result in any Dsx+ NBs, while *grim-rpr* double mutants showed many surviving NBs in females larval VNC, four of these NBs expressed Dsx. This indicates that Dsx+ tNB apoptosis in females required both *grim* and *reaper* genes. This also suggests that remaining 8 NBs which were earlier thought to undergo cell cycle exit actually undergo apoptosis. Subsequently we find that enhancer for the Dsx+ tNB apoptosis lies within 14.5kb region of the genome like in case of abdominal NBs. Interestingly, we also find that apoptotic *enhancer-lacZ* lines are sex-specific in their expression, and did not express in Dsx+ tNB of males. This indicates that the enhancer for apoptosis of Dsx+ tNB is female specific and lies within 717bp of genomic region of the *NBRR*.

Details of progress made in current reporting year (1 April, 2017-31 March, 2018)

We had found a direct role of Notch in AbdA mediated NB apoptosis in larval abdominal segments. In order to check role of Notch in tNB apoptosis, we carried out RNAi mediated knockdown of Notch gene in tNBs. We found that Notch knockdown resulted in ectopic NBs, but none of these tNBs were Dsx+. This suggested that while Notch signalling plays a role in death of Dsx- NBs in terminal region it has no role in

Dsx+ tNBs.

We had already found that 717 bp abdominal apoptotic enhancer is utilized by Dsx+ NBs for their apoptosis in females, therefore we decided to investigate this enhancer in detail. We could identify 10 Dsx binding sites with potential AbdB binding (AT rich) sequences on 717bp abdominal apoptotic enhancer. All of these motifs were tested for Dsx and AbdB binding alone as well as together. We found that out of 10 sites 6 sites bound Dsx and all 10 sites bound AbdB. Of these 6 Dsx binding sites which bound both Dsx and AbdB, 3 sites showed cooperative interaction of both AbdB and Dsx on EMSA, suggesting that Dsx and AbdB could interact with each other on the DNA. This observation is significant considering that AbdB is HD containing TF while Dsx is a Zn finger protein. The detailed characterization of this interaction is ongoing. Simultaneously the role of *Drosophila* cell cycle genes like *Cyclin, A, B, E* and *E2F* are being tested for their contribution in continued sex specific proliferation of Dsx+ tNBs proliferation in male larval CNS.

Publication

1. Khandelwal R, Sipani R, Govinda Rajan S, Kumar R and Joshi R (2017). Combinatorial action of Grainyhead, Extradenticle and Notch in regulating Hox mediated apoptosis in *Drosophila* larval CNS. *PLoS Genet* 13(10): e1007043.

Other publications

2. Bakshi A and Joshi R. Understanding the regulation of neural stem cell proliferation in *Drosophila* central nervous system. *J Neuroscience Research* (in press).

LABORATORY OF FUNGAL PATHOGENESIS

Understanding the pathobiology of an opportunistic human fungal pathogen
Candida glabrata

Faculty	Rupinder Kaur	Staff Scientist & Wellcome Trust -DBT India Alliance Senior Fellow
Ph.D Students	Vandana Sharma Mubashshir Rasheed Priyanka Bhakt Anamika Battu Kundan Kumar Fizza Askari Mahima Sagar Sahu	Senior Research Fellow (till June 2017) Senior Research Fellow Senior Research Fellow Senior Research Fellow Junior Research Fellow Junior Research Fellow Junior Research Fellow
Other Members	S Surya Vamshi Reshma Chowdary Alokam T.S. Jalajaveronica Rani Rachana Roshan Dev Naveen Kumar Rajaram Purushotham Deepak Kumar Choudhary Romila Moirangthem	Technical Officer National Postdoctoral Fellow Research Associate (till June 2017) Research Associate (since August 2017) Research Associate (since Dec 2017) Project SRF Project SRF Project JRF
Collaborators	CV Srikanth Arunaloke Chakrabarti Debasis Biswas Suman S Thakur	RCB, Faridabad PGIMER, Chandigarh AIIMS-Bhopal, Bhopal CCMB, Hyderabad

Candida species account for 70 to 80% of bloodstream fungal infections with *Candida glabrata* being the second most frequently isolated *Candida* species after *C. albicans*. Despite being a successful pathogen, *C. glabrata* lacks few key fungal virulence traits, and appears to rely primarily on alternative mechanisms to survive the nutrient-poor, hostile environment of the human host. Research in our laboratory is aimed at a better understanding of pathogenesis and antifungal drug resistance mechanisms of *C. glabrata*.

Project 1: Functional genomic analysis of *C. glabrata*-macrophage interaction

Objectives

1. Screening of a *C. glabrata* mutant library for altered survival profiles
2. Identification and analysis of genes required for survival *in vitro* and *in vivo*

Summary of the work done until the beginning of this reporting year

Using an *in vitro* system comprised of the human monocytic cell line THP-1, we demonstrated that wild-type *C. glabrata* cells are able to impede phagolysosome acidification, survive the reactive oxygen species generated and replicate in THP-1 macrophages. We further screened a Tn7 insertion mutant library, representing 50% of the *C. glabrata* genome, for altered survival in macrophages, and identified 53 novel genes required for intracellular survival and/or proliferation. One of identified gene, *CgVPS15*, codes for a regulatory subunit of the class III phosphoinositide 3-kinase (PI3K). Through generation and characterization of *Cgvps15* Δ and *Cgvps34* Δ deletion strains, which lack the PI3K regulatory and catalytic subunit, respectively, we showed that *CgVps15* and *CgVps34* are essential for intracellular survival, vacuolar protein sorting and virulence

in *C. glabrata*. We also showed that CgVps34 catalyzes the conversion of phosphatidylinositol to phosphatidylinositol-3-phosphate, and is required for maintenance of iron homeostasis. Notably, *Cgvps34Δ* cells were found to be deficient in the retrograde transport of the CgFtr1 iron permease from the plasma membrane to the vacuole in response to high environmental iron, which led to elevated susceptibility of the *Cgvps34Δ* mutant to surplus iron.

Details of the progress made in the current reporting year (April 1, 2017 - March 31, 2018)

As discussed above, wild-type (*wt*) *C. glabrata* cells undergo intracellular proliferation while the *Cgvps34Δ* mutant is killed in human THP-1 macrophages. Since one of the macrophage defense mechanisms is to restrict iron availability to engulfed pathogens to control their proliferation, we sought to examine if the inability of the *Cgvps34Δ* mutant to survive the intracellular milieu of macrophages is owing to its deregulated iron metabolism and/or probably high intrinsic iron requirement. For this, we first checked intracellular behaviour of *wt* and *Cgvps34Δ* cells pre-grown either in the iron-limited or iron-surplus medium. Proliferation of *wt* and survival of *Cgvps34Δ* cells in macrophages remained unaffected by prior iron chelation or supplementation, thereby, precluding any effect of internal iron reserves on intracellular replication. Further, although the macrophage environment is presumed to be a low-iron environment, macrophage-internalized *C. glabrata* wild-type cells are known to downregulate the expression of high-affinity iron transport genes after 10 h of co-incubation, raising the possibility that the macrophage internal milieu either is an iron-rich environment, or requirement of the high-affinity iron uptake system is less at the late stage of infection. To decipher the iron status of macrophages, we checked the localization of CgFtr1-GFP, which is located on the cell membrane in an iron-poor environment, in macrophage-internalized *C. glabrata* cells. We found CgFtr1-GFP to be present exclusively on the cell membrane in macrophage-ingested *wt* and *Cgvps34Δ* cells (**Figure 1A**) indicating that *C. glabrata* cells encounter iron limitation in macrophages.

Next, to examine the effect of varied extracellular iron concentration in the medium upon survival of the *Cgvps34Δ* mutant in macrophages, we infected *wt* and *Cgvps34Δ* cells to THP-1 macrophages cultured in iron-restricted [serum-

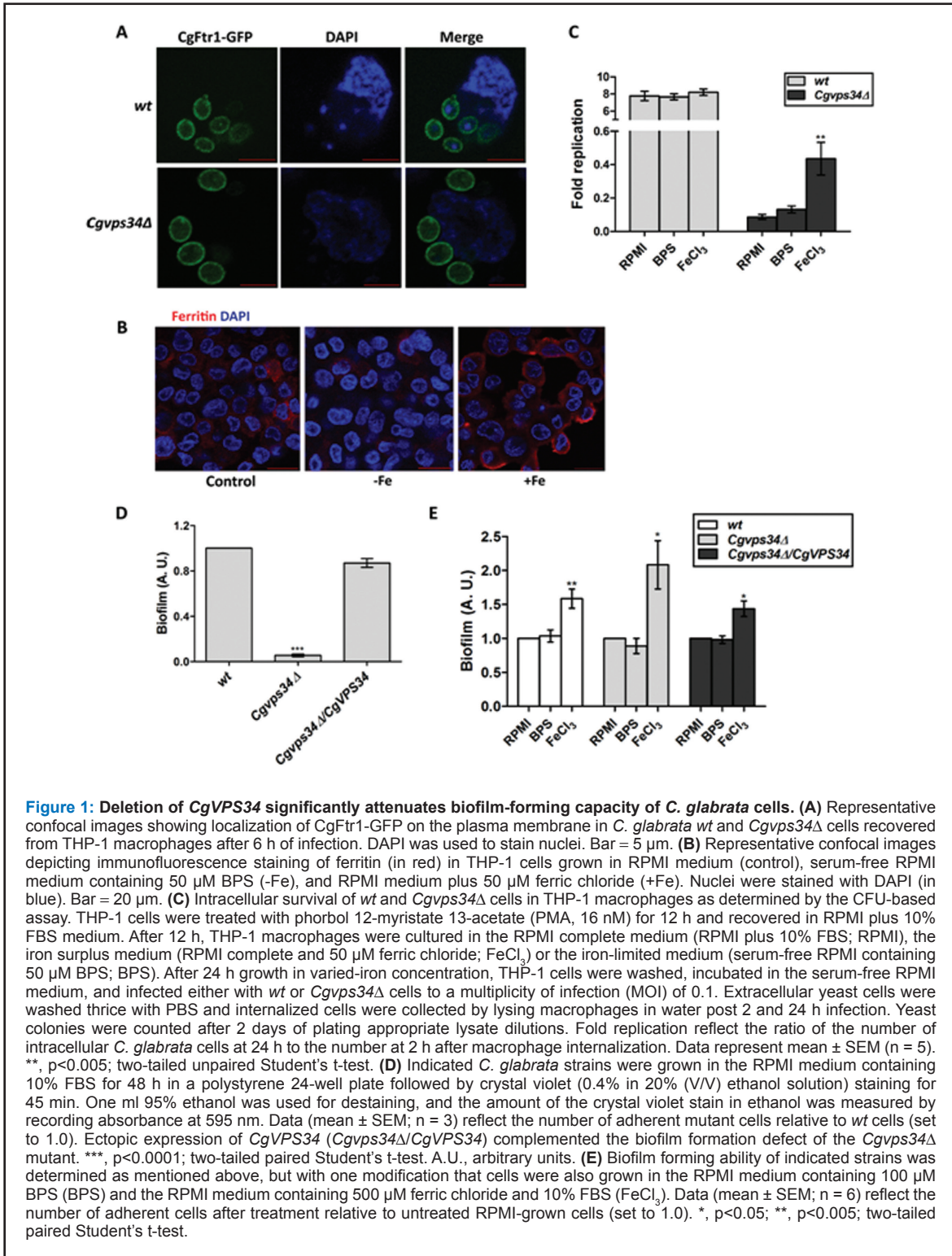
free RPMI medium containing extracellular iron chelator, BPS (bathophenanthroline disulfonate)] and iron-supplemented (serum and ferric chloride-containing RPMI) media. Iron in macrophages is stored in the form of an iron storage protein, ferritin. To check if macrophage culturing in the aforementioned-media alters intracellular levels of ferritin, we performed immunofluorescence as well as Western analysis using the anti-ferritin antibody. THP-1 cells grown in regular RPMI medium and RPMI medium containing BPS displayed very low levels of ferritin expression while surplus iron medium-cultured THP-1 cells exhibited intense ferritin staining (**Figure 1B**). Similarly, Western analysis showed a significant increase in intracellular levels of ferritin upon growth in the ferric chloride-supplemented medium while ferritin protein levels were undetectable in RPMI- and RPMI plus BPS medium-cultured THP-1 cells. Together, these results indicate that RPMI plus BPS and RPMI plus ferric chloride media mirror low and high environmental iron conditions, respectively. Importantly, no appreciable differences in macrophage viability were observed upon culturing in iron-deplete and iron-replete media, as checked by the MTT assay.

Further, intracellular proliferation of *wt* cells remained unaffected by external iron concentration indicating that iron availability is not the limiting factor for intracellular replication of *C. glabrata* cells (**Figure 1C**). Intriguingly, while survival rate of the *Cgvps34Δ* mutant remained unaffected in iron-restricted macrophages, a 5-fold higher survival was observed in iron-supplemented macrophages compared to RPMI medium-grown macrophages (**Figure 1C**). These data suggest that higher extracellular iron levels promote the survival of *Cgvps34Δ* cells in macrophages which could be attributed, in part, to better growth of the *Cgvps34Δ* mutant in iron-rich macrophages.

Iron availability is also known to modulate the ability of bacterial pathogens to form biofilms. Hence, to investigate the effect of extra iron on biofilm-forming ability of *C. glabrata*, we measured the capacity of *wt* and *Cgvps34Δ* cells to form biofilm under normal, iron-deplete and iron-replete conditions. We found the biofilm-forming capacity of *Cgvps34Δ* cells to be 10-fold lower compared to that of *wt* cells in the normal growth medium (**Figure 1D**). Environmental iron content has previously been

reported to regulate adherence of *C. glabrata* to Lec2 epithelial cells, with surplus-iron medium-grown cells exhibiting 3-fold higher adherence to Lec2 cells compared to regular-iron medium-cultured cells. Consistent with these results,

ability of *wt C. glabrata* cells to form biofilms was 60% higher in the high-iron environment (**Figure 1E**). Similarly, a 2-fold increase in biofilm formation was observed in the *CgVps34Δ* mutant under surplus iron conditions



(Figure 1E). Of note, BPS medium-grown *wt* and *Cgvps34Δ* cells formed biofilms on polystyrene plates to the same extent as corresponding RPMI medium-grown cells (Figure 1E). These data demonstrate that *CgVPS34* disruption led to abrogation of two important virulence traits, biofilm formation and intracellular survival, of *C. glabrata* and this abolishment may contribute to the reduced virulence of the *Cgvps34Δ* mutant. Studies are currently ongoing to examine if high iron-induced increase in biofilm formation is due to overexpression of Epa family of cell surface adhesins.

Project 2: Characterization of glycosylphosphatidylinositol-linked aspartyl proteases in *Candida glabrata*: role in pathogenicity

Objectives

1. Molecular and biochemical characterization of *C. glabrata* yapsins
2. Identification and characterization of physiological substrates of *C. glabrata* yapsins

Summary of the work done until the beginning of this reporting year

A family of eleven putative glycosylphosphatidylinositol (GPI)-linked, cell surface-associated aspartyl proteases is a major virulence determinant of *C. glabrata*. These proteases, also referred as yapsins, are encoded by *CgYPS1-11* genes. Previously, we have shown the pivotality of *C. glabrata* yapsins to several pathobiological processes including maintenance of cell wall composition and architecture, pH and vacuole homeostasis, intracellular survival and virulence.

Details of the progress made in the current reporting year (April 1, 2017 - March 31, 2018)

The *Cgyps1-11Δ* mutant, that lacks all eleven yapsins, is known to display altered cell wall composition and is killed in macrophages. However, the underlying molecular basis for mutant cell death is not known. As cell wall polysaccharides (β -glucan, mannan and chitin) are known to modulate the host immune response, we hypothesized that the altered cell wall constitution of the *Cgyps1-11Δ* mutant could lead to overactive macrophages resulting in its death. To test this, we first determined the transcriptional response, through genome-wide microarray analysis, of THP-1 macrophages to infection with *wt* and *Cgyps1-11Δ* cells. We found

THP-1 cells to respond to *C. glabrata* infection by transcriptional induction of negative regulators of cytokine secretion and repression of MAPK, PI3K and TNF signaling pathways. Analysis of the gene expression pattern of *wt*- and *Cgyps1-11Δ*-infected macrophages revealed that although they share 89 common upregulated genes, the *Cgyps1-11Δ* mutant invokes a subdued and differential immune response in macrophages primarily through upregulation of viral response genes and downregulation of ventricular septum morphogenesis genes. To corroborate these data, we measured the levels of 12 cytokines, IL-1 α , IL-1 β , IL-2, IL-4, IL-6, IL-8, IL-10, IL-12, IL-17A, IFN- γ , TNF- α and GM-CSF (Granulocyte-macrophage colony-stimulating factor), in the culture media of either uninfected THP-1 cells or THP-1 macrophages infected with *wt* and *Cgyps1-11Δ* cells. *C. glabrata* infection led to no significant production of any cytokine in THP-1 macrophages but for the pro-inflammatory cytokine IL-1 β . A 1.5-fold and 2.0-fold higher level of IL-1 β was observed in *wt*-infected macrophages compared to uninfected macrophages (Figure 2A).

IL-1 β is a key mediator of the inflammatory response and synthesized as a pro-protein by activated macrophages which, upon proteolytic cleavage by the caspase-1 enzyme, is converted to its active form. The caspase-1 itself gets activated upon assembly of the antifungal NLRP3 inflammasome complex which consists of NLRP3, ASC adaptor protein and procaspase-1. The spleen tyrosine kinase (Syk) is required for activation of the NLRP3 inflammasome. Hence, we next checked the status of Syk signaling, and found a 1.4- and 2.3-fold higher Syk phosphorylation in *wt*-infected and *Cgyps1-11Δ*-infected macrophages, respectively, compared to uninfected THP-1 cells (Figure 2B). This indicates a hyperactivated Syk in *Cgyps1-11Δ*-infected macrophages. Consistently, the Syk inhibitor R406 rescued mutant cell death in THP-1 cells, while having no effect on intracellular proliferation of *wt* cells (Figure 2C). Further, R406 treatment also abolished IL-1 β production in *C. glabrata*-infected macrophages (Figure 2A). Also, *wt* and *Cgyps1-11Δ* cells displayed 1.3-fold higher replication and 2-fold better survival, respectively, in THP-1 macrophages, which were treated with the NLRP3 inflammasome inhibitor MCC950 (Figure 2D). The MCC950 treatment also abolished IL-1 β production in THP-1 cells implicating Syk signaling and NLRP3 inflammasome in IL-1 β production.

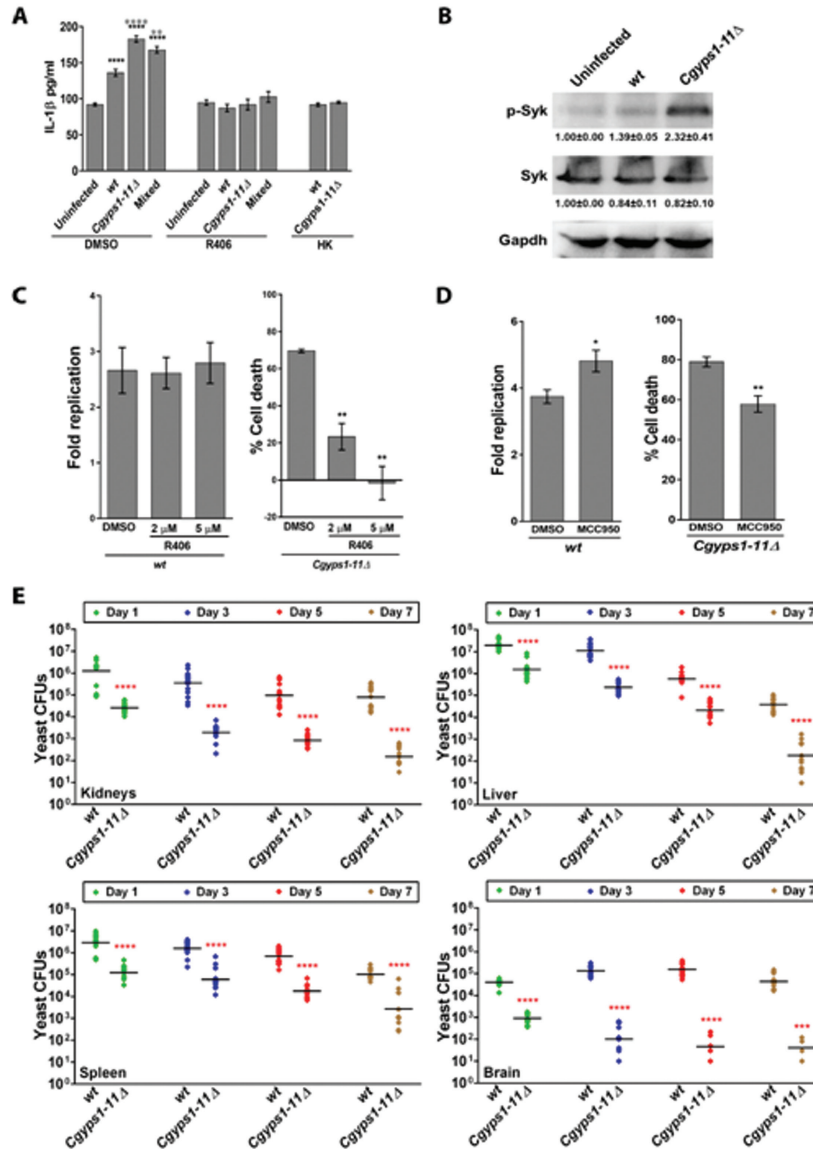


Figure 2: *Cgyps1-11 Δ* -induced IL-1 β production in THP-1 macrophages is dependent on Syk. (A) Measurement of secreted IL-1 β in DMSO- or R406-treated, *C. glabrata*-infected THP-1 cells. THP-1 macrophage infection was done with *C. glabrata* cells at a MOI of 1:1, and IL-1 β levels in the culture supernatant were measured after 24 h using the human IL-1 β ELISA Set II kit. 'HK' indicates heat-killed dead *C. glabrata* cells which were obtained after incubation at 95°C for 20 min. 'Mixed' infection refers to co-infection of THP-1 cells with *wt*- and *Cgyps1-11 Δ* cells. Notably, infection with mixed culture and heat-killed cells led to a 1.6-fold and no induction of IL-1 β production, respectively. Statistically significant differences in IL-1 β levels between uninfected and *C. glabrata*-infected, and *wt*- and *Cgyps1-11 Δ* -infected macrophages are indicated by black and grey asterisks, respectively. **, $p < 0.01$, ****, $p < 0.0001$; unpaired two-tailed Student's t-test. **(B)** A representative immuno blot illustrating phosphorylated Syk in uninfected, and *wt*- and *Cgyps1-11 Δ* -infected THP-1 macrophages. THP-1 cell extracts containing 120 μ g protein were resolved on 10% SDS-PAGE and probed with anti-phospho Syk, anti-Syk and anti-Gapdh antibodies. Gapdh was used as a loading control. For quantification, intensity of individual bands in three independent Western blots was measured using the ImageJ densitometry software. Total Syk and phosphorylated Syk signal in each lane was normalized to the corresponding Gapdh signal (considered as 1.0). Data (mean \pm SEM) are presented as fold change in signal intensity levels in infected samples compared to uninfected samples (taken as 1.0) underneath the blot. Intracellular survival of *wt* and *Cgyps1-11 Δ* mutant in R406-treated **(C)** and MCC950-treated **(D)** THP-1 macrophages. Either 2 and 5 μ M of R406 **(C)** or 15 μ M of MCC950 **(D)** was added to THP-1 macrophages 2 h prior to *C. glabrata* infection, and infection was continued in the presence of R406 and MCC950. Fold replication for *wt* cells indicates the ratio of the number of intracellular *C. glabrata* cells at 24 h to that at 2 h post infection. % cell death for the *Cgyps1-11 Δ* mutant indicates viability loss of mutant cells in DMSO- and R406/MCC950-treated THP-1 cells between 2 h and 24 h of infection, as determined by measurement of intracellular CFUs at these two time points. Data represent mean \pm SEM ($n = 3$). *, $p < 0.05$; **, $p < 0.01$; unpaired two-tailed Student's t-test. **(E)** Kinetics of infection of *C. glabrata* *wt* and *Cgyps1-11 Δ* cells in BALB/c mice. Mice were infected intravenously, sacrificed at indicated days, and fungal burden in kidneys, liver, spleen and brain was determined using cfu-based assay. Diamonds represent yeast cfus recovered from organs of the individual mouse, while the horizontal line indicates the cfu geometric mean ($n = 12-16$) for each strain. Statistically significant differences in cfus between *wt*- and *Cgyps1-11 Δ* -infected mice are marked (***, $p < 0.001$, ****, $p < 0.0001$; Mann-Whitney test). Of note, we could retrieve *Cgyps1-11 Δ* cfus from brain of four mice only out of 14 infected animals 7 dpi.

Collectively, our data suggest that Syk-dependent enhanced production and secretion of IL-1 β is deleterious for intracellular survival of *C. glabrata*, and, that, CgYapsins are pivotal to suppression of this inflammatory response. Studies to identify macrophage receptors that are involved in recognition of *C. glabrata* cells, are currently underway.

The *Cgygs1-11 Δ* mutant-induced enhanced production of IL-1 β in THP-1 macrophages prompted us to examine its effects *in vivo*. As the role of CgYapsins in colonization and dissemination of *C. glabrata* cells is yet to be characterized, we first studied the kinetics of *C. glabrata* infection in the mouse model of systemic candidiasis. Assessment of fungal burden in four target organs, kidneys, liver, spleen and brain, after 1, 3, 5 and 7 days post infection (dpi) revealed that, at day 1, mouse kidneys were colonized with 1.2×10^6 *wt* cells while only 2.6×10^4 *Cgygs1-11 Δ* cells represented renal fungal burden in the *Cgygs1-11 Δ* -infected mice (**Figure 2E**). Similarly, 13- to 45-fold lower cfus were recovered from other organs of the *Cgygs1-11 Δ* -infected mice compared to the *wt*-infected mice 1 dpi (**Figure 2E**) indicating that CgYapsins are required for initial colonization and dissemination of *C. glabrata* cells. As the time course progressed, there was a constant decrease in the number of yeast cells which were harvested from kidneys, liver and spleen of the *wt*-infected mice (**Figure 2E**). These results preclude any significant multiplication of *C. glabrata* cells in these mouse organs.

Strikingly, the fungal burden in brain at 3 dpi was 3-fold higher than that observed at 1 dpi

(**Figure 2E**) suggesting that *C. glabrata wt* cells either take longer to reach brain or they undergo multiplication in the brain. Brain cfus for the *wt* strain remained similar between 3 and 5 dpi while a 2-fold decrease was observed 7 dpi (**Figure 2E**). Notably, *Cgygs1-11 Δ* cells failed to colonize and/or migrate to the brain in substantial numbers, with a drastic decline in the mutant number at later time points (**Figure 2E**). In fact, of 14 *Cgygs1-11 Δ* -infected mice, mutant cells were recovered from the brain of only four mice 7 dpi (**Figure 2E**). The *Cgygs1-11 Δ* mutant did not fare well in other organs either with mouse organ fungal burden decreasing sharply (**Figure 2E**). Together, these data suggest that CgYapsins are required for colonization, dissemination and persistence on prolonged infection of *C. glabrata* in brain, kidneys, liver and spleen as well as for plausible replication in the brain during early stages of infection. Studies are currently ongoing to investigate if rapid clearance of the *Cgygs1-11 Δ* mutant from mouse organs is due to induction of a pro-inflammatory cytokine response.

Publications

Research papers published in the calendar year 2018

1. Rasheed, M., Battu A. and Kaur, R. Aspartyl proteases in *Candida glabrata* are required for suppression of the host innate immune response. *Journal of Biological Chemistry* (In press).

LABORATORY OF GENOMICS AND PROFILING APPLICATIONS

Faculty	Madhusudan Reddy Nandineni	Staff Scientist
PhD Students	Anujit Sarkar	Senior Research Fellow (till April 2017)
	Soumya Rao	Senior Research Fellow
	Mugdha Singh	Senior Research Fellow
	Saphy	Junior Research Fellow
Other Members	Anujit Sarkar	Research Associate (since Aug. 2017)

Objectives:

1. Human genetic diversity studies among various population groups in India
2. Dissection of plant-fungal interactions in the chilli-*Colletotrichum* pathosystem

Project 1: Human genetic diversity studies among various population groups in India.

Summary of work done until the beginning of this reporting year (upto March 31, 2017)

India is known for its rich cultural, linguistic, geographic as well as genetic diversity. To investigate the genetic richness in India, researchers in the past had extensively employed DNA-based markers like short tandem repeats (STRs) and single nucleotide polymorphisms (SNPs) located on autosomes, Y-chromosome and mitochondria. Furthermore, several SNPs were reported that are associated with externally-visible characteristics (EVCs) or phenotypes like skin, hair and eye colour, body height, etc.

In the previous report, we described the association between SNPs and human skin colour variation (measured as melanin index; MI), which is one of the most conspicuously visible variable traits in humans. Based on previous studies, 30 SNPs were tested in the Indian populations for their association to the phenotype. The SNPs were genotyped using the GoldenGate[®] assay on BeadXpress[®] (Illumina, Inc. USA) according to the manufacturer's instructions and the data was filtered based on call rate and minor allele frequency (MAF) of the SNPs for the whole dataset.

In another aspect of this study, 22 autosomal STRs were used to study genetic similarities among the populations and it was found that they are very useful for forensic human identification (HID) purposes as well. Similarly, to study Y-chromosomal STRs (Y-STRs)-based genetic diversity in Indian populations, 346 individuals

from 11 States were genotyped employing PowerPlex[®] Y23 (PPY23) (Promega, Madison, WI, USA) chemistry. The panel demonstrated high values of forensic parameters (discrimination capacity (DC) = 0.9855491329, match probability (MP) = 0.003044349 and haplotype diversity (HD) = 0.999845377), indicating their applicability for forensic investigations. Further, Whit Athey's haplogroup predictor tool returned R1a as the most abundant Y-chromosomal haplogroup in India.

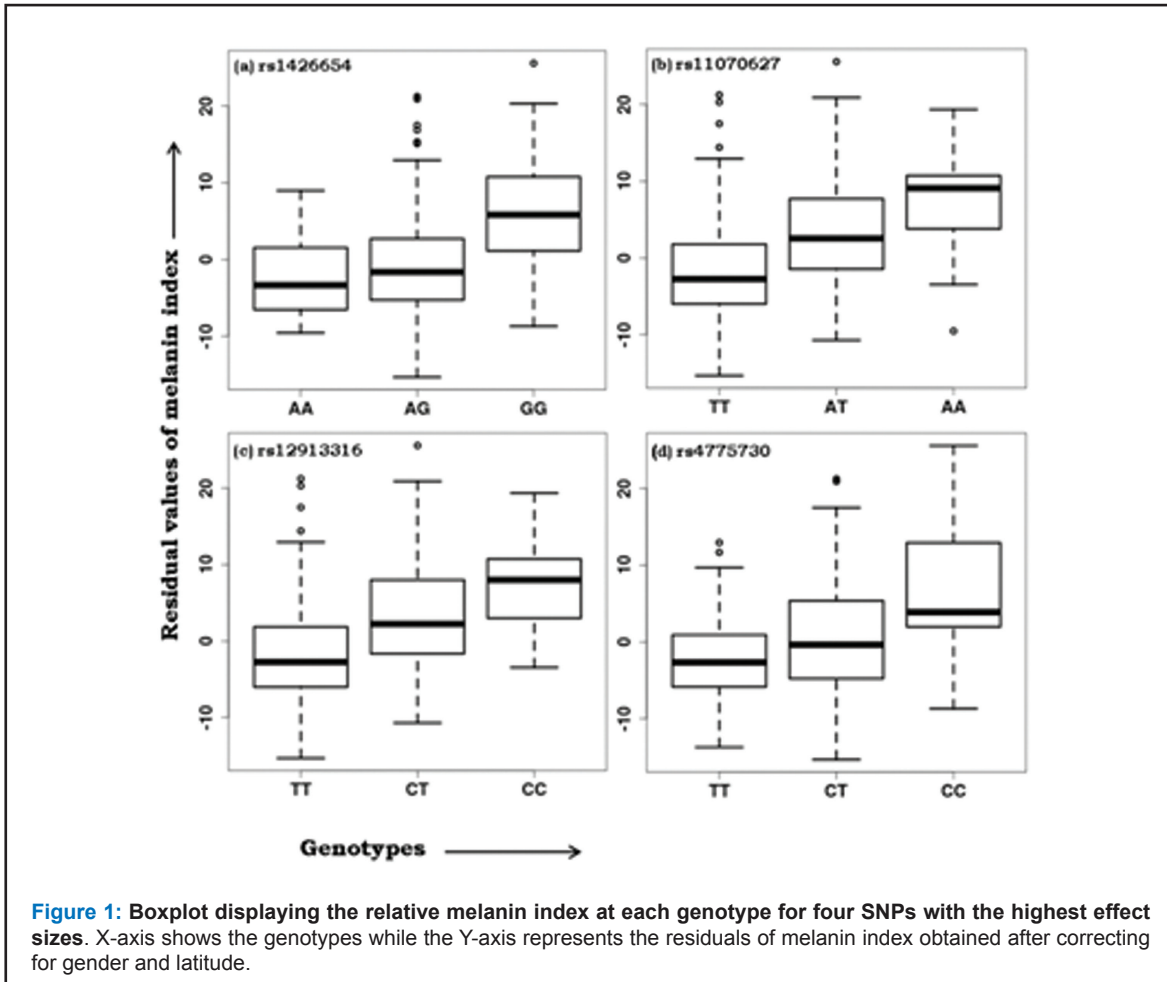
Details of progress made in the current reporting year (April 1, 2017- March 31, 2018):

a. Association of genetic variants with human skin colour in Indian populations:

The genotypes obtained were filtered based on call rate and MAF of the SNPs and further analysis was performed on 22 SNPs. MI was observed to be highly variable (23-71) in the samples across Indian populations, with the populations from North India showing lowest MI (mean ~39), while those from South India had the highest MI (mean ~48), whereas the populations from West and East India displayed intermediate MI (mean ~41). This was supported by regression analyses, which showed that latitude of a location (a measure of distance from equator) was significantly associated to skin colour ($p = 6.61 \times 10^{-16}$) and could explain 19.41% of the variance in skin colour. This was in agreement with previous observations that the abundance of ultraviolet (UV) radiation (represented by latitude) at a location was directly proportional to the MI of the resident human populations. Gender was not significantly associated with the MI in this study ($p = 0.148$). Association studies for the 22 SNPs towards residual melanin index (MI corrected after nullifying the effects of latitude and gender) suggested that nine among them were significantly associated with the phenotype in the Indian populations. For each of the four SNPs with the highest effect size viz., rs1426654

(4.89), rs11070627 (4.91), rs12913316 (4.85) and rs4775730 (3.95), Figure 1 shows the effect of allele dosage pertaining to darker pigmentation on the MI of the samples. rs1426654 genetic variant is well-known for affecting the human skin colour worldwide and rs11070627, which is in high

linkage disequilibrium (LD) with rs1426654 ($D' = 90$), was previously reported to be associated with the phenotype in populations of South Asia. Overall, these nine SNPs could explain ~31% of the variance in MI among Indian populations.



Haplotype analysis of these nine SNPs (all located on chromosome 15), identified two associated blocks. The first block consists of rs2924566 (C/T) and rs4775730 (C/T), whereas the second one consists of rs1426654 (A/G), rs11070627 (A/T) and rs12913316 (C/T). The alleles G, A and C from the second block were significantly associated ($p = 3.29 \times 10^{-14}$) and contributed to the highest effect size (5.32), greater than any single SNP. This study would contribute to the better understanding of the molecular determinants of skin pigmentation and might have potential applications in forensic genetics/forensic 'phenotyping'.

b. Human genetic variation studies in Indian populations based on expanded Y-chromosomal STRs:

In addition to the individuals genotyped previously, 61 male samples (making a total of 407 individuals from 12 States in India), representing major geographic regions of India, were studied employing 23 Y-STRs incorporated in PowerPlex® Y23 (PPY23) (Promega, Madison, WI, USA) and the corresponding accession numbers were obtained from Y Chromosome Haplotype Reference Database (YHRD) (<http://www.yhrd.org>) for each of the population

group. The principle co-ordinate analysis (PCoA) among these populations showed that they were in close vicinity to each other and >60 percent of the variation was explained by the first two axes, with first and second axis explaining 39.42 and 26.32 percentage of the total variance, respectively. Discriminant analysis of principal components (DAPC) showed that individuals belonging to the same geographic region were closely spaced in their individual cluster and all the 12 clusters were found overlapping at the center of the plot.

Further, haplogroup assignment indicated that R1a (51.5%), H (16.2%) and L (15.8%) were the major haplogroups present in the country and accounted for more than three-fourths of the populations. The occurrence of R1a was observed to decrease from North to South, while an increase in occurrence the haplogroup L was observed from North to South. On the other hand, haplogroup H was observed to be distributed uniformly across the country.

On comparing these populations with 129 worldwide populations, it was observed that expectedly they were in close proximity to the other Indian populations studied in the past (Gujarati Indians in Texas, Indians in Singapore, South-Indian (Tamils). Interestingly, it was also observed that few other populations were closer to Indian populations viz., Italy (Calabria), Barnaya-Hungary (Romani), London-UK (British-Asians), Lebanon, Iraq, Bolivia (Mestizo), Panama, Hungary (Budapest), Bolivia (Native-Americans), Estonia, Latvia and Lithuania (Vilnius). The peninsula of Italy is believed to have witnessed many waves of migrations and there are arguments supporting the diffusion of Indo-European languages in Southern Italy in historical times via the Mediterranean route. The proximity of Italian and Indian samples might be the resultant of historical events of migration that would have led to sharing of language and patrilineal genes. The genetic similarity observed between Romanis and Indians is supported by various anthropological and linguistic studies. Previous studies employing genome-wide scans support the observation that the Romanis are genetically close relatives of Indians, whereas the genetic closeness of Indians and British-Asians from London-UK can be attributed to the common ethnicity of these two populations. Populations from Bolivia and Panama have been shown to possess Amerindian components, which bring them genetically closer to the extant Indian populations. It has been postulated that

the Amerindian component in Native American populations might be the result of different and chronological waves of migration from Asia. The genetic affinity of populations belonging to Lebanon and Iraq from the Levant region to Indian populations may be attributed to their geographical proximity, which is also supported by the findings from a previous study. The geographic position of the Levant countries is at the cross-roads of Africa, Eurasia and South Asia and thus might have witnessed the ancient migration of humans out of Africa. The populations from India in this study showed interesting proximity to the Baltic (Estonia, Latvia and Lithuania) populations as well. This could be explained based on the assumption that the Indo-European influence was one of the factors responsible for the genetic make-up of contemporary populations from Baltic regions. The patrilineal proximities of Indian populations to Italians, Romanis, British-Asians, Levantines, Native Americans and Baltics suggest a series of migratory events that led to the current sharing of languages and genes.

Project 2: Dissection of plant-fungal interaction studies in the Chilli - *Colletotrichum* pathosystem.

Summary of work done until the beginning of this reporting year (up to March 31, 2017):

Colletotrichum truncatum (formerly called as *C. capsici*) is the most predominant species in India causing chilli anthracnose leading to both pre- and post-harvest losses. With the availability of whole genome sequence for chilli and several *Colletotrichum* species, the chilli – *Colletotrichum* pathosystem offers an excellent model for studies on the infection process and molecular interactions between the host and fungal pathogen. The present study aims to identify and characterize pathogenicity genes in *C. truncatum* to get an insight into different aspects of its biology, life-style and host specificity through whole genome sequencing of the *C. truncatum* and insertional mutagenesis.

We had previously reported the *de novo* whole genome sequencing of *C. truncatum* employing Illumina HiSeq platform that consisted of 80 scaffolds with a total length of 55.3 Mb. Phylogenetic analyses placed *C. truncatum* close to *C. gloeosporioides* and *C. orbiculare*, which enabled comparative genomics studies. The draft genome assembly of *C. truncatum* was assessed to be 100% complete by Core Eukaryotic Genes Mapping Approach (CEGMA)

and tBLASTn based on coverage of orthologues of all 458 core eukaryotic genes (CEGs). The genome was annotated into consensus gene models by combining the transcript evidence obtained through RNA-sequencing of fungus with homology-based and *ab initio* approaches. Secretome including all the secreted proteins of an organism is the most important category of genes in the pathogenic fungi. A stringent pipeline of tools was used to identify 1,257 proteins that were highly likely to be secreted, 310 of which were predicted to be effectors that are released at host-pathogen interphase and manipulate the host immune responses during fungal invasion.

Details of progress made in the current reporting year (April 1, 2017- March 31, 2018):

a) Identification and comparative analysis of important pathogenicity genes

1. Carbohydrate Active enZymes (CAZymes)

The carbohydrate metabolizing enzymes play an important role in the degradation of fungal and plant cell wall components (chitin, cellulose, hemicellulose, pectin, etc.) and utilization of plant polysaccharides during the host colonization by pathogenic fungi. The *C. truncatum* genome had 1,036 genes encoding 147 different CAZyme families as identified by hidden Markov model (HMM) search against dbCAN database. 449 of *C. truncatum* genes associated with 88 CAZyme families were predicted to be secreted. The important classes of CAZymes like glycoside hydrolases (GH), carbohydrate esterases (CE), polysaccharide lyases (PL), auxillary activities (AA) and carbohydrate-binding modules (CBMs) were identified in its genome. Almost all members of chitin- and cellulose-binding CBM1 family that might play a role in penetration and early infection, were predicted to be secreted; while pectin-degrading families GH28, GH78, PL1 and PL3 and cutinases from CE5 that may help in colonization of fruits with thick cuticle and complex cell walls of fruits rich in pectins, were specifically expanded in *C. truncatum* as compared to other *Colletotrichum* species and related fungi.

2. Proteases

Proteases complement the plant cell wall degrading enzymes and protect the fungi from host pathogenicity-related (PR) proteins released during fungal invasion. Batch BLAST search against MEROPS protease database (<http://merops.sanger.ac.uk>) identified 258 genes in *C.*

truncatum belonging to 76 families of proteases and 10 genes belonging to 4 protease inhibitor families. The metalloproteases (101) comprised the largest category of proteases, indicating a possible link between certain metalloproteases and necrotrophic lifestyle of this fungal pathogen, followed by serine (85) and cysteine proteases (40). The largest protease family was prolyl aminopeptidase (S33) followed by subtilisins (S08). 71 secretory proteases were detected that included 35 serine and 29 metalloproteases. 13 out of 20 subtilisins, the alkaline proteases, were predicted to be secreted that may enable the fungus to withstand local alkalization of the host tissue due secretion of ammonia by the fungus. The two chitin-degrading, secreted fungalysins (M36), CTRU_012332 and CTRU_004392, shared only 50% identity with each other, while the former showed 91% identity to a *C. fructicola* fungalysin, indicating their independent acquirement and evolution in *C. truncatum* genome.

3. Secondary metabolism-related genes

The secondary metabolites (SMs) produced by the fungal phytopathogens are known to be associated with their pathogenicity, virulence and host range. The genes encoding the enzymes for the production of the SMs are located in the form of a cluster in the genome and their expression is transcriptionally co-regulated. *C. truncatum* had 73 SM gene clusters, the highest among all the fungi analysed. The SM backbone genes were mainly represented by 50 polyketide synthases (47 PKS and 3 PKS-like genes), 27 non-ribosomal protein kinases (17 NRPS and 10 NRPS-like genes), 9 dimethylallyl transferases (DMAT) and 4 PKS-NRPS hybrid genes. The orthologue analysis identified 18 core SM backbone genes that were present in all the *Colletotrichum* species, including 6 PKS, 8 NRPS and 4 DMAT genes.

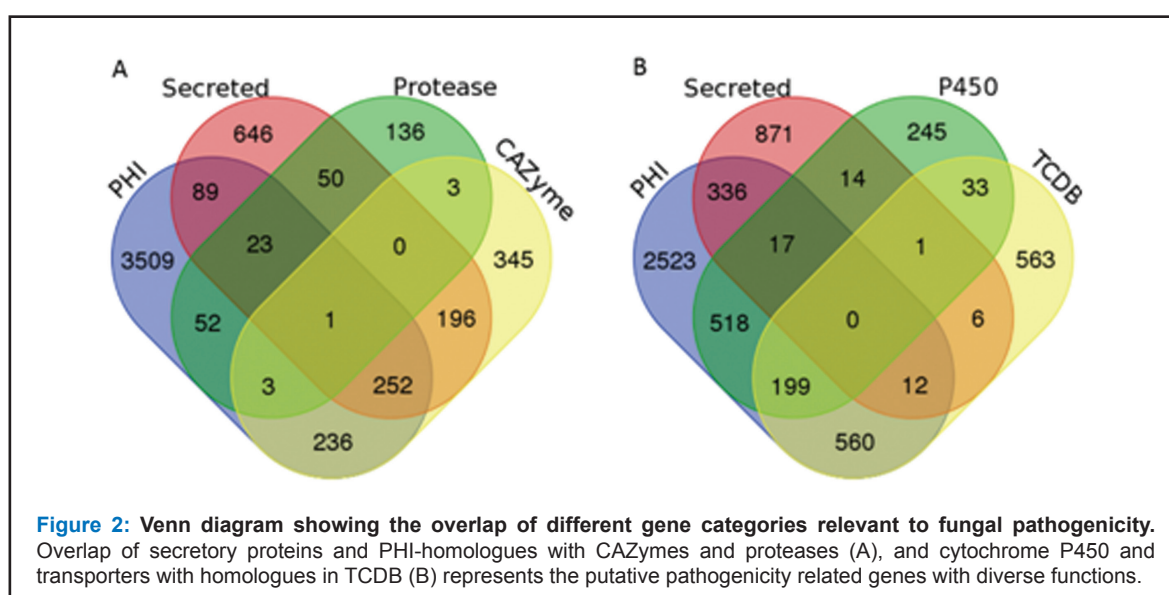
The two categories of genes, apart from transcriptional regulators, often found associated with SM clusters are cytochrome P450 monooxygenases (P450s) that play a key role in fungal pathogenicity and metabolism, and transporters that export toxic metabolites from fungal cells during host invasion. Both these categories were found to be highly expanded in *C. truncatum* genome. 1,345 genes of *C. truncatum* had homologues in fungal cytochrome P450 database while 1,374 genes had homologues in Transporter Classification Database (TCDB) with 340 genes belonging to the Major Facilitator

Superfamily (MFS, 2.A.1), the largest category of secondary carriers involved in nutrient uptake and transport of toxins and drugs. The second most expanded transporter family in *C. truncatum* was nuclear pore complex (NPC, 1.I.1) with 82 genes, involved in transport of mRNA and proteins across the nuclear envelope, followed by ABC transporter family (3.A.1) with 56 genes, which contains both uptake and efflux transport systems driven by ATP hydrolysis.

b) Identification of homologues in Pathogen-Host Interactions database (PHI-base)

PHI-base comprises of the genes associated with pathogenicity that are experimentally

validated in different pathogens. A total of 4,165 genes with homologues in the PHI-base were identified in *C. truncatum*, 15.7% (2,156) of which were important for the fungal pathogenicity and virulence. Out of 4,165 genes, 1,470 genes were associated with reduced virulence, 263 with loss of pathogenicity, 287 with mixed phenotype, 59 with hypervirulence and 38 with effector functions. The majority of these genes belonged to different functional categories relevant for pathogenicity including secreted proteases, CAZymes, P450s and transporters (Figure 2).



Publications:

Research papers published in the calendar year 2017

1. Anujit Sarkar and Madhusudan R. Nandineni, (2017). Development of a SNP-based panel for human identification for Indian populations. **Forensic Science International: Genetics**, 27: 58-66.
2. Mugdha Singh and Madhusudan R. Nandineni, (2017). Population genetic analyses and evaluation of 22 autosomal STRs in Indian populations. **International Journal of Legal Medicine**, 131: 971-973.

Research papers published in 2017-2018 (until 31st March 2018)

1. Soumya Rao and Madhusudan R. Nandineni, (2017). Genome sequencing and comparative genomics reveal a repertoire of putative pathogenicity genes in chilli anthracnose fungus *Colletotrichum truncatum*. **PLoS ONE**, 12(8): e0183567.
2. Anujit Sarkar, Mark Stoneking, and Madhusudan R. Nandineni (2017). Unraveling the human salivary microbiome diversity in Indian populations. **PLoS ONE**, 12(9): e0184515.
3. Anujit Sarkar and Madhusudan R. Nandineni (2018). Association of common genetic variants with human skin color variation in Indian populations. **American Journal of Human Biology**, 30(1): e23068.

LABORATORY OF IMMUNOLOGY

Understanding the tumorigenesis and its regulation

Principal Investigator	Sunil K Manna	Staff Scientist
Ph D Students	Raveendra Babu M Pankaj Gupta Shashank Saurav Aher Abhishek Taterao	Senior Research Fellow Senior Research Fellow Senior Research Fellow Junior Research Fellow
Other Members	T Navaneetha	Technical Assistant
Collaborators	Biswadev Bishayi Tushar Basu Baul Sudit Mukhopadhyay	Calcutta University, Kolkata NEHU, Shilong NIT, Durgapur

Objectives

1. Understanding and regulation of inflammatory and tumorigenic responses.
2. Understanding the role of Profilin in regulation of tumorigenesis.
3. Understanding and regulation of advanced glycation endproducts (AGE)-mediated lipogenesis and autophagy.

Summary of work done until the beginning of this reporting year (April 1, 2016 - March 31, 2017)

MITF inhibition is the main cause of resveratrol mediated cell death but not NF- κ B:

Resveratrol (3,5,4'-trihydroxystilbene) is a polyphenolic compound, which is a natural component of grapes, peanuts, berries and especially red wine. It is known for its anti-oxidant and cardio-protective functions. Recent researches were focused on its anti-cancerous properties. Here we investigated the mechanism of its anti-melanoma activity. Resveratrol significantly activated cell death in A375 melanoma cells, compared to other natural and synthetic compounds. Resveratrol induces more cell death in melanoma than the melanoma specific drug, vemurafenib, compared with PC3, HT29 and MDA MB-231. This suggests resveratrol is a potent melanoma inhibitor compared to other types of cancers. MITF is the most important transcription factor for melanoma survival, proliferation and differentiation. Resveratrol suppressed levels of MITF and its DNA binding activity. Overexpressed MITF inhibited resveratrol mediated cell death, which further strengthened this view. Resveratrol inhibited both NF- κ B and MITF transcription factors, whereas BAY 11-7082 (NF- κ B inhibitor) inhibited only partially.

Our data suggest that inhibition of NF- κ B is the reason for general cancer cell death, but MITF inhibition must be the main reason or an additive contributor for melanoma specific cell death. Overall, resveratrol induced potent melanoma cell death by inducing apoptosis. These data warrant further study of mechanism, upstream of MITF, in order to improve resveratrol based chemotherapy for melanoma.

Role of ERK and p53 in resveratrol mediated melanoma cell death:

We have already established resveratrol-mediated inhibition of MITF and activation of melanoma cell death. As the MAPK pathway with gain of function mutations in B-Raf (especially V600E B-Raf) is the most activated signaling mechanism in melanoma, we hypothesized that resveratrol might be inhibiting the MAPK pathway similar to vemurafenib. To our surprise, it activated phosphorylation of many kinases such as ERK1/2, Akt and AMPK α . It also activated p53, showing its role in apoptosis induced by resveratrol. Both B-Raf or MEK1/2 inhibitors were unable to inhibit p-ERK1/2 or MITF downregulation. Specific ERK inhibitor, SCH772984 was used to explore the mechanism of cell death downstream of ERK. SCH772984 is a dual inhibitor, where it inhibits activity of p-ERK as well as its phosphorylation by upstream MEK1/2. This compound partially inhibited ERK phosphorylation and p53 activation, but not PARP cleavage. Even the co-treatment of SCH772984 with resveratrol did not decrease the cell death caused by resveratrol, suggesting there is more in the mechanism than what meets the eye. We further wanted to explore the role of p53. We made stable cell lines expressing shRNA for p53. Knockdown of p53 rescued approximately 25% of resveratrol mediated cell death, indicating

p53's need for resveratrol. Overexpression of MITF in p53 knock down background rescued it even further. These data conclude that both inhibition of MITF and activation of p53 can have role in mechanism of cell death. Whereas, knock down of MITF in p53 knock down background, brought the cell death equal to just MITF knock down levels. This allocates more importance to MITF inhibition as p53 knock down cannot rescue cell death. Our findings conclude that resveratrol activates many signaling intermediates. ERK1/2 is one of them, which could be involved in p53 mediated apoptosis. We need further evidence to establish role of ERK1/2 in resveratrol mediated p53 activation, as this is necessary for melanoma cell death.

Details of progress in the current reporting year (April 1, 2017 - March 31, 2018)

1) Suppression of IKK, but not activation of p53 is responsible for cell death mediated by naturally occurring oxidized tetranortriterpenoid

Tetranortriterpenoids (limonoids) obtained from the neem tree (*Azadirachta indica*) have gained significant attention due to their anti-proliferative properties. Here we are investigating the role of a highly oxidized tetranortriterpenoid, azadirachtin on induction of the cell death.

Azadirachtin induces cell death in various cell types. The HepG2, THP1, A549, MCF7 and Sf9 were treated with increasing doses of azadirachtin for 72 h and cell death was found to increase in a concentration-dependent manner in these cells (Figure 1A). Moreover, lactate dehydrogenase (LDH) assay performed from the culture supernatant of treated cells did not show any cytolysis to these cells even at high doses of azadirachtin (data not shown).

Azadirachtin potentiates cell death in Hepatocarcinoma cells. HepG2 cells were treated with increasing concentrations of azadirachtin for 72 h and staining of the cells and their nuclei were visualized microscopically. The nuclear staining by propidium iodide (PI) showed increasing number of the fragmented nuclei at higher concentrations of azadirachtin. Further, the cell death was confirmed by fluorescence microscopy using Live and Dead cytotoxicity assay (Figure 1C1). Cells were treated with increasing concentration of azadirachtin and the cleavages of PARP and caspase 8 were determined by Western blot. The intensities of cleaved bands of PARP (85 kDa) and caspase 8 increased upon azadirachtin treatment in

a concentration-dependent manner (Figure 1B). Finally, apoptosis was confirmed by Flow cytometry by Annexin V/PI staining. Percentages of both early (Q3) and late (Q2) phases of apoptosis was increased by azadirachtin (Figure 1C2).

Azadirachtin activates p53. The tumor suppressor p53 is the major determinant for therapeutics-mediated cell death. Therefore, the transcriptional activation of p53 upon azadirachtin treatment was studied by gel shift assay in wild-type p53 expressing HepG2 cells. Azadirachtin showed enhance p53 DNA-binding activity with increasing doses, whereas the binding completely disappear in the presence of excess unlabeled p53 oligonucleotides (Figure 1D). As the functions of p53 are regulated by its post-translational modifications, the phosphorylation of p53 at Ser15 and Ser46 were determined by Western blot. No phosphorylation of the p53 was observed in a dose-dependent treatment with azadirachtin; however, there was an increase in the levels of p53 as well as its dependent gene, p21 (Figure 1E). On the contrary, doxorubicin treated cells showed enhanced phosphorylation as well as increased amounts of p53 and p21. This increased amount of p53 prompted us to check the stability of p53 upon azadirachtin treatment. Furthermore, the amount of p53 protein increased with increasing concentration of azadirachtin as determined by Western blot (Figure 1E). These data suggest that azadirachtin might be activating p53 to promote cell death.

Azadirachtin interacts with Mdm2 in silico. Since azadirachtin enhanced the stability of p53, we speculated that azadirachtin might be interacting with p53 directly or through Mdm2. To test our hypothesis, the binding between azadirachtin and p53 or Mdm2 was analyzed *in silico* using Autodock. Docking studies revealed the existence of Mdm2-Azadirachtin interaction, as shown by strong binding energy values. Interestingly, the hydrophobic residues of Mdm2 were common for azadirachtin and p53 binding (Figure 1F). Particularly, the His96 residue of Mdm2, which forms a hydrogen bond with azadirachtin was involved in the Mdm2-p53 interaction.

Azadirachtin potentiates cell death regardless of p53 status. To address whether p53 has any differential role in azadirachtin-mediated cell death, isogenic HCT116 wild-type and HCT116 p53 negative cells were used. Upon treatment with different concentrations of azadirachtin, the amount of Mdm2 decreased upon azadirachtin

treatment in wild-type cells, but high basal levels of Mdm2 were observed in p53 negative cells, which did not decrease upon azadirachtin treatment. We also found that there was an increase in the level of p53 due to treatment with azadirachtin in wild cells (Figure 1G). These data suggest that the induction of cell death mediated by azadirachtin was not related to activation of p53.

NF- κ B rescues azadirachtin-mediated cell death. To confirm whether suppression of NF- κ B is involved in azadirachtin-mediated cell death, we transfected cells with p65 and cell viability was assayed with MTT cytotoxicity assay. Azadirachtin treatment in vector transfected cells showed 47% ($p < 0.005$) and 66% ($p < 0.01$) cell death at 36 and 48 h, respectively whereas, in p65-transfected cells, azadirachtin was unable

to induce a significant amount of cells death at any time of treatment (Figure 1H). These data suggest that azadirachtin-mediated cell death predominantly depends upon deregulation of NF- κ B.

In this study, we are exploring the molecular interactions of azadirachtin with key signaling molecules that lead to tumor cell death using *in vitro* and *in silico* approaches. For the first time, we show that azadirachtin binds to Mdm2 and inhibits Mdm2-mediated degradation of p53. However, the cell death is not dependent upon p53 and instead, azadirachtin mediates cell death predominantly by the direct inhibition of IKK activation. This study provides a mechanistic insight to the action of azadirachtin and proposes it as a potent chemotherapeutic agent in targeting tumors with non-functional p53.

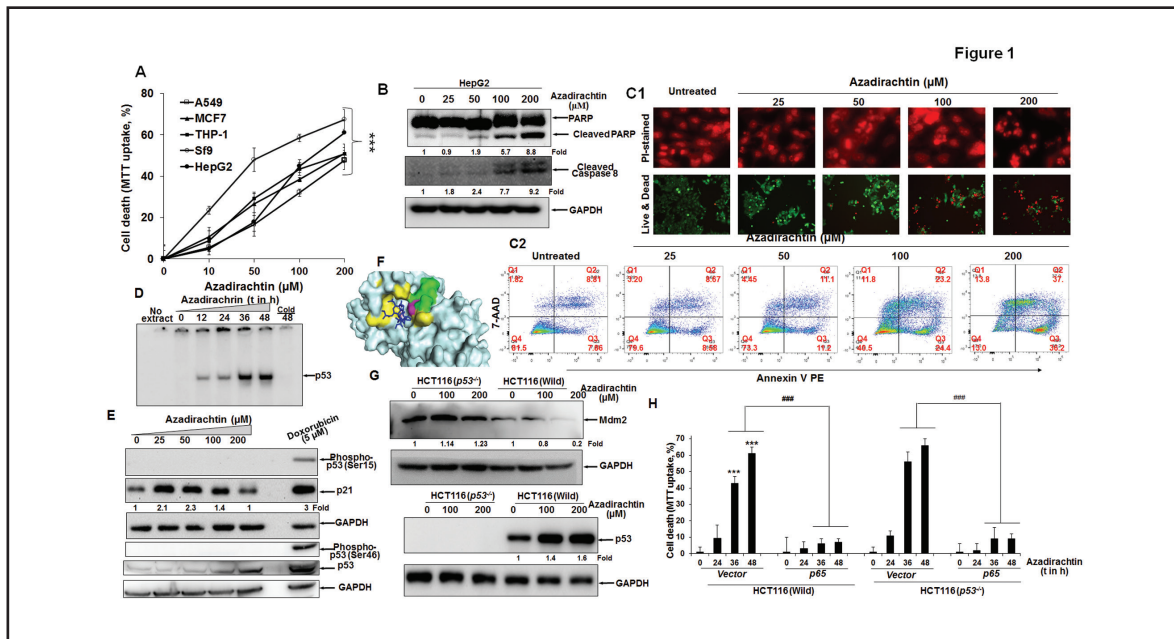


Figure 1: Azadirachtin induces cell death by decreasing Mdm2, but not NF- κ B and upregulating p53. Several cell types (5000/well of 96-well plate in triplicate) were cultured overnight and incubated with different concentrations of azadirachtin for 72 h. MTT assay was done and indicated in percentage of cell death, considering the untreated cells' value as 0% cell death. The experiment was repeated at least thrice and the data were plotted as mean \pm S.E.M. *** $P < 0.001$ (one-way ANOVA and Tukey's multiple comparisons test) (A). HepG2 cells were treated with different concentrations of azadirachtin ranging from 0 to 200 μ M for 72 h and cleavages of PARP and Caspase 8 were carried out by Western blot using whole cell extracts (B). HepG2 cells, treated with increasing concentrations of azadirachtin for 72 h were incubated with 'Live & Dead' assay solution for 30 min. Cells were viewed under fluorescence microscope. The number of apoptotic cells (red color) and live cells (green color) (C1). HepG2 cells were treated with different concentrations of azadirachtin for 48 h stained with annexin V-PE and 7-AAD and flow cytometry was done (C2). HepG2 cells were treated with 100 μ M azadirachtin for different time. The p53 DNA binding were assayed by EMSA from nuclear extract of azadirachtin-treated cells for different times (D). The phospho-p53 (Ser-46 and -15), p53 and GAPDH were measured by Western blot from whole cell extracts in azadirachtin (different concentrations) and doxorubicin (5 μ M) treated cells (E). The p53 overlap with azadirachtin in the presence of Mdm2. Mdm2 indicated in light cyan color, p53 in green, Azadirachtin in blue. The common residues obtained in docking are marked in yellow color (F). Isogenic HCT116 cells were treated with different concentrations of azadirachtin for 72 h. Mdm2 and p53 was measured from WCE by Western blot (G). GAPDH was used as loading control. Isogenic HCT116 cells, transfected with vector, p65, and NF- κ B-luciferase constructs for 3 h washed and cultured for 12 h, were treated with 100 μ M azadirachtin for a different times. Cell death was measured by MTT assay and indicated in percentage (H).

2) Developing Organometallic anticancer compounds based on Organotin.

Chemoprevention is considered as a promising strategy in the field of cancer therapy and suppressing or reversing the process of tumor formation has gained much attention. Synthesis and spectroscopic properties of seven new dibutyltin(IV) compounds of 2-*{(E)-4-hydroxy-3-[(E)-4-(aryl)iminomethyl]phenyldiazenyl}*benzoic acids (L^nHH' ; $n = 2-8$) with general formula $\{[Bu_2Sn(L^nH)]_2O\}_2$ (**1-7**) have been reported. The key question is whether ferrocenyl-organotin combination of organometallics is better substitute for existing chemotherapeutic drugs for cancer. The structures of dibutyltin(IV) compounds **1-3**, **6** and **7** were accomplished from single crystal X-ray crystallography which reveal the common ladder-type structure with two *endo*- and two *exo*-Sn atoms (Figure 2A). To elucidate the effect of the dibutyltin(IV) compounds to regulate tumorigenic response, A375 human melanoma cells were incubated with various concentrations of dibutyltin(IV) compounds and cell viability was estimated. The cell deaths as shown by the red-stained cells were increased in a concentration dependent manner by these compounds (Figure 2B). The inhibition of cell viability as determined by MTT assay was increased by these compounds in a concentration dependent manner and among these compound **6** showed much potency. The IC_{50} values, as determined from the MTT assay data, were indicated in nM. Cell death was further supported by the caspase 8 cleavage

(Figure 2C), and PARP cleavage by compound **6** (Figure 2D). The role of p53, a tumor suppressor was determined on tin-mediated cell death. Dibutyltin(IV) compounds increased cell death in HCT116 p53 wild type as well as in the HCT116 p53 knocked out [HCT116 (p53^{-/-})] cells (Figure 2E). These data collectively suggest that p53 has no role in tin-mediated cell death. Dibutyltin(IV) compound **6** was shown to decrease the binding of diphenylhexatriene (DPH), a membrane binding fluorescence probe, in a concentration-dependent manner (Figure 2F). Doxorubicin and oleandrin were used as positive control for membrane microviscosity altering agents. These data suggest that the dibutyltin compound **6** is very potent to alter microviscosity of the cell membrane.

Dibutyltin(IV) compound **6** showed emission maximum at 565 nm when it was scanned in the range 500 to 700 nm, keeping excitation wavelength at 512 nm in the fluorimeter (Figure 2G1). Similarly, the excitation maximum was determined as 512 nm when it scanned between 400 to 550 nm, keeping the emission maximum at 565 nm (Figure 2G2). Cells were incubated with compound **6** and then visualized under a confocal microscope, keeping at emission and excitation maxima for different times. The auto-fluorescence of compound **6** was observed for 8 h and it disappeared later time of incubation in the cells (Figure 2G3). These data suggest that compound **6** shows the auto-fluorescence and inactivated/degraded inside the cells upon incubation period.

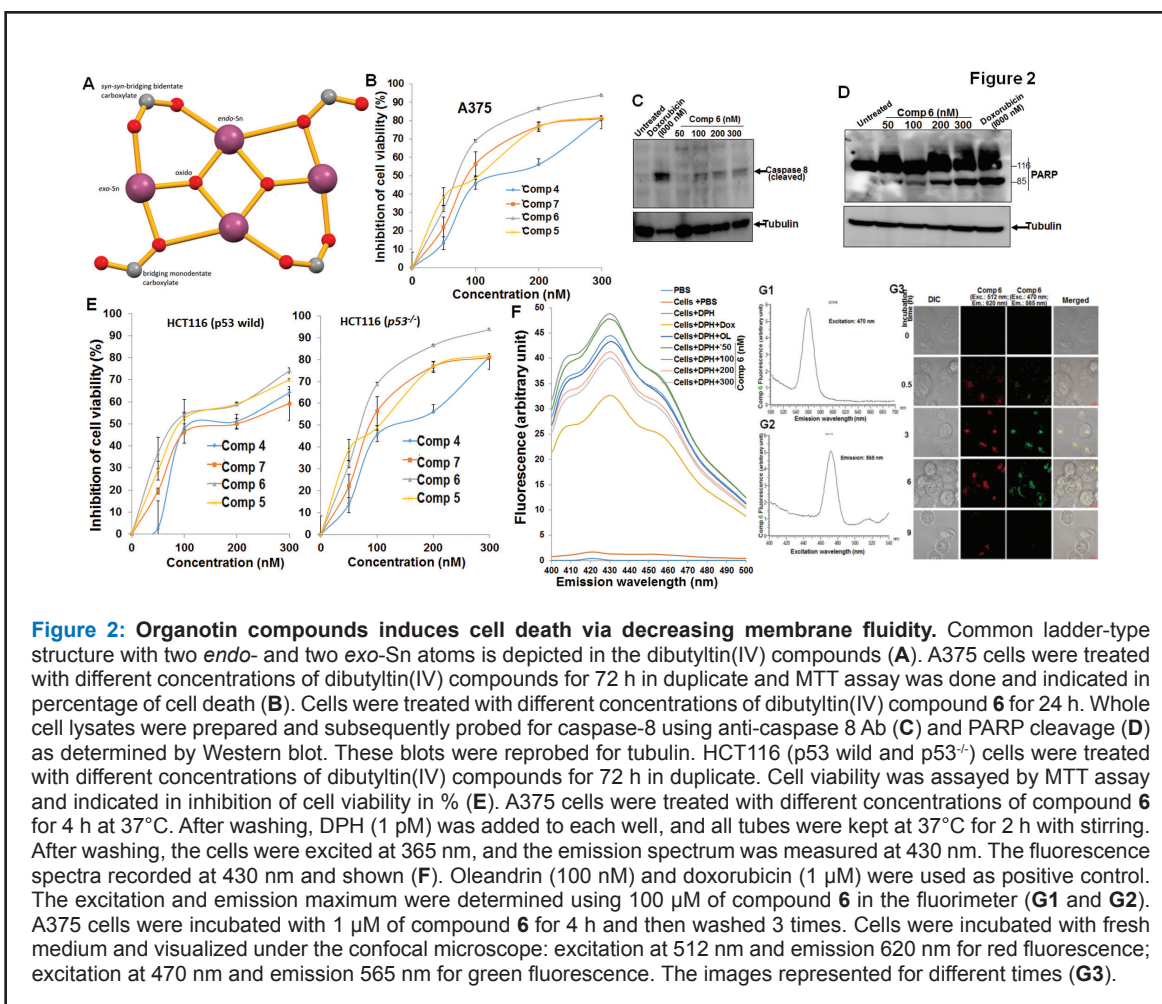


Figure 2: Organotin compounds induces cell death via decreasing membrane fluidity. Common ladder-type structure with two *endo*- and two *exo*-Sn atoms is depicted in the dibutyltin(IV) compounds (A). A375 cells were treated with different concentrations of dibutyltin(IV) compounds for 72 h in duplicate and indicated in percentage of cell death (B). Cells were treated with different concentrations of dibutyltin(IV) compound 6 for 24 h. Whole cell lysates were prepared and subsequently probed for caspase-8 using anti-caspase 8 Ab (C) and PARP cleavage (D) as determined by Western blot. These blots were re-probed for tubulin. HCT116 (p53 wild and p53^{-/-}) cells were treated with different concentrations of dibutyltin(IV) compounds for 72 h in duplicate. Cell viability was assayed by MTT assay and indicated in inhibition of cell viability in % (E). A375 cells were treated with different concentrations of compound 6 for 4 h at 37°C. After washing, DPH (1 pM) was added to each well, and all tubes were kept at 37°C for 2 h with stirring. After washing, the cells were excited at 365 nm, and the emission spectrum was measured at 430 nm. The fluorescence spectra recorded at 430 nm and shown (F). Oleandrin (100 nM) and doxorubicin (1 μM) were used as positive control. The excitation and emission maximum were determined using 100 μM of compound 6 in the fluorimeter (G1 and G2). A375 cells were incubated with 1 μM of compound 6 for 4 h and then washed 3 times. Cells were incubated with fresh medium and visualized under the confocal microscope: excitation at 512 nm and emission 620 nm for red fluorescence; excitation at 470 nm and emission 565 nm for green fluorescence. The images represented for different times (G3).

Publications

Peer-reviewed journals

(i) Research papers published in 2017:

- Basu Baul TS, Kehie P, Duthie A, Guchhait N, Raviprakash N, Mokhamatam RB, Manna SK, Armata N, Scopelliti M, Wang R, Englert U (2017) Synthesis, photophysical properties and structures of organotin- Schiff bases utilizing aromatic amino acid from the chiral pool and evaluation of the biological perspective of a triphenyltin compound. **Journal of Inorganic Biochemistry** 168: 76-89.
- Basu Baul TS, Dutta D, Duthie A, Guchhait N, Rocha BGM, Guedes da Silva MFC, Mokhamatam RB, Raviprakash N, Manna SK (2017) New dibutyltin(IV) ladders: Syntheses, structures and, optimization and evaluation of cytotoxic potential employing A375 (melanoma) and HCT116 (colon carcinoma) cell lines in vitro. **Journal of**

Inorganic Biochemistry 166: 34-48.

- Verma N, Manna SK. (2017) AGE potentiates cell death in p53 negative cells via upregulation of NF-kappaB and impairment of autophagy. **Journal of Cellular Physiology** 232(12): 3598-3610.

(ii) Research papers published in 2018 (until 31st March 2018):

None

(iii) Research papers in press (as on 31st March, 2018):

- Gupta P, Zaidi AH, Manna SK.* (2018) Suppression of IKK, but not activation of p53 is responsible for cell death mediated by naturally occurring oxidized tetranortriterpenoid. **Journal of Cellular Biochemistry** 2018 May 8. doi: 10.1002/jcb.26879. [Epub ahead of print] PMID: 29738082

LABORATORY OF MAMMALIAN GENETICS

Epigenetic mechanisms underlying developmental pathways

Principal Investigator	Sanjeev Khosla	Staff Scientist
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Project 1: Specialized chromatin structures as epigenetic imprints to distinguish parental alleles

Summary of work done until the beginning of this reporting year (up to March 31, 2017):

We had previously performed functional analysis of a knock-out strain of mice which had the second intron of the imprinted *Neuronatin* gene replaced by a *Neo^R*-cassette at its endogenous locus. In the previous years we had examined the effect of this replacement on the expression of the imprinted *Neuronatin* gene and its phenotypic consequences.

Details of progress made in the current reporting year (April 1, 2017- March 31, 2018)

Generation and characterization of *NNΔI²* mice

The Neomycin resistant cassette (*Neo^R*) was present in place of the second intron of *Neuronatin* in the *NNΔI² (Neo^R+)* mice. To negate the possibility that the allelic misregulation of *Neuronatin* was due to the presence of *Neo^R*, we deleted this cassette from the *NNΔI²/Neo^R+* mice by crossing these mice with a Cre-recombinase expressing mice strain 129_*alpl*^{tm1 (cre)} Nagy.

Homozygous or heterozygous *NNΔI²* mice were crossed with C57BL/6J to generate WT,

PKO, MKO and HZ *NNΔI²* mice to examine the effect of *Neuronatin* second intron deletion on *Neuronatin* expression. Quantitative RT-PCR performed on RNA isolated from brain and embryos (13.5 d.p.c) of WT, PKO, MKO and HZ *NNΔI²* mice showed no expression of *Neuronatin* in PKO and HZ *NNΔI²* mice while the expression of *Neuronatin* remained unaltered in MKO *NNΔI²* mice (Figure 1A) indicating that the deletion of the second intron from the normally expressed paternal allele leads to loss of *Neuronatin* expression. However, *Neuronatin* second intron deletion from the maternal allele has no effect on *Neuronatin* expression.

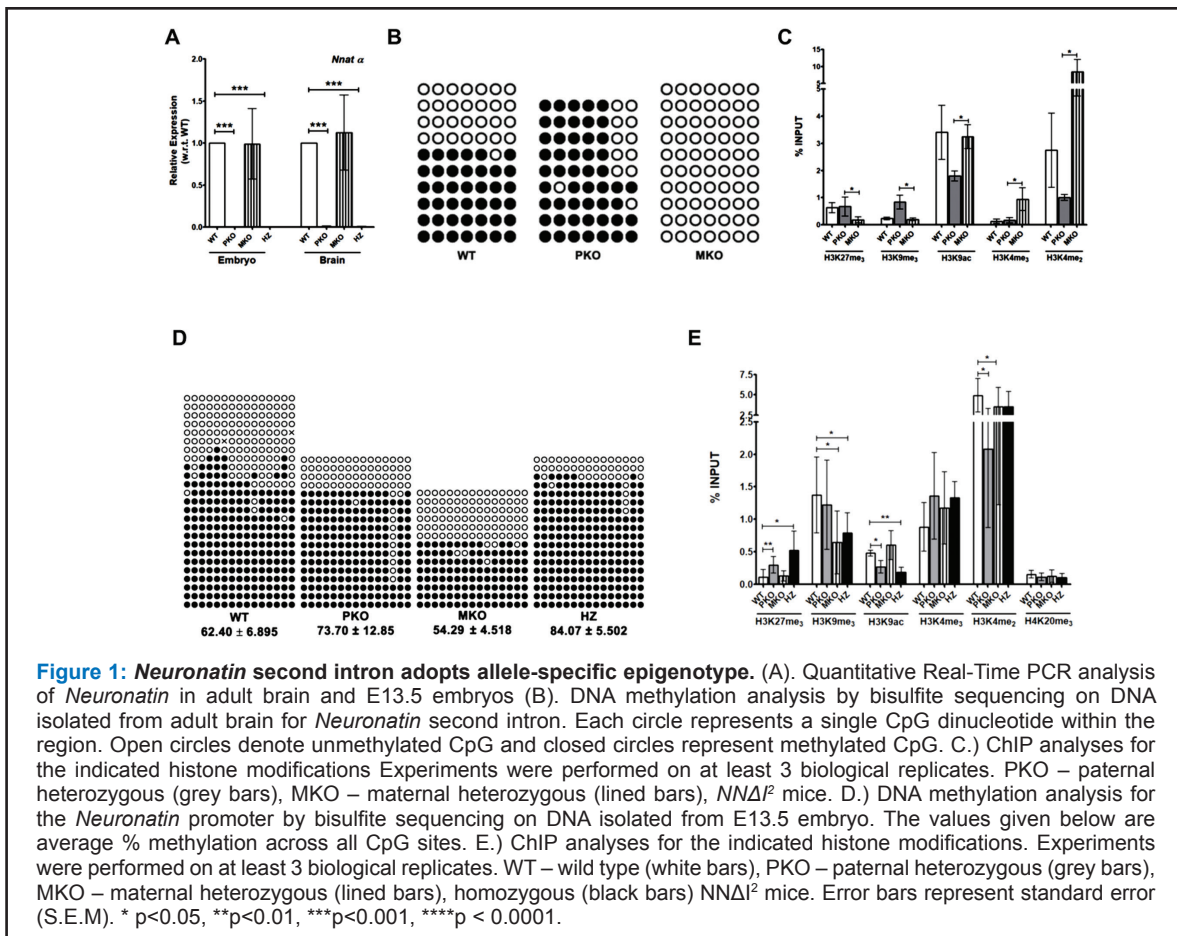
Imprint Control Regions (ICR) exhibit allele-specific DNA methylation and histone modification profile. To examine whether the second intron of *Neuronatin* possesses these requisite characteristics of an ICR, the paternal (PKO *NNΔI²* mice, with second intron only on the maternal allele) and maternal (MKO *NNΔI²* mice, with second intron only on the paternal allele) heterozygous *NNΔI²* mice were examined for DNA methylation and histone modification status at the second intron of *Neuronatin*. DNA methylation status of *Neuronatin* second intron was examined in embryo (13.5d.p.c), brain and liver by bisulfite sequencing. In all the three

tissues, the second intron of *Neuronatin* was, fully methylated in PKO *NNΔI²* mice (second intron present only on the maternal allele), and unmethylated in MKO *NNΔI²* mice (second intron present only on the paternal allele) as compared to the wild type mice (have both the alleles of the second intron) which showed 50% methylation (Figure 1B for adult brain). Thus, the second intron of *Neuronatin* is unmethylated on the paternal allele and methylated on the maternal allele in all the tissues examined.

To examine the histone modification profile of the second intron of *Neuronatin*, ChIP was performed for H3K27me3, H3K9me3 (inactive chromatin marks), H3K4me2, H3K9ac, and H3K4me3 (active chromatin marks) modifications, on chromatin isolated from brain tissues of WT, PKO and MKO heterozygous *NNΔI²* mice. The *Neuronatin* second intron was associated with active chromatin marks such as H3K4me3, H3K4me2 and H3K9ac on the expressed paternal allele and the inactive chromatin marks such as H3K9me3 and H3K27me3 were associated with the silent maternal allele of *Neuronatin* (Figure 1C).

The epigenetic status of an imprinted loci is dependent on its ICR. Deletion of ICRs has been shown to change the DNA methylation and histone modification profile of imprinted genes at the respective imprinted locus. Examination of DNA methylation by bisulfite sequencing on genomic DNA isolated from embryos (E13.5 d.p.c) of wild type and PKO, MKO and HZ *NNΔI²* mice showed gain of *Neuronatin* promoter methylation in PKO and HZ *NNΔI²* mice in comparison to WT (Figure 1D). Our results indicate that the deletion of the second intron can alter *Neuronatin* promoter DNA methylation in an allele-specific manner.

Concomitant with loss of *Neuronatin* expression from the paternal allele, we also observed a significant loss of the active chromatin marks such as H3K9ac, H3K4me2 and gain of repressive chromatin mark H3K27me3 in PKO and HZ *NNΔI²* mice (Figure 1E). Our data suggests that changes in histone modifications particularly that of H3K9ac, H3K4me2 and H3K27me3 at *Neuronatin* promoter is involved in the regulation of its expression.



Project 2: Host epigenetic response to infection

Summary of work done until the beginning of this reporting year (up to March 31, 2017)

We have previously identified mycobacterial encoded DNA methyltransferase (Rv2966c) and a histone methyltransferase (Rv1988) which have the ability to methylate cytosines and histone H3 respectively in the host genome in a non-canonical manner. We had also identified host SUV39H1 protein to be upregulated and relocalised during mycobacterial infection.

Details of progress made in the current reporting year (April 1, 2017- March 31, 2018)

In a preliminary experiment, where we examined the expression profile of several histone methyltransferases and demethylases in THP1

macrophages upon *M. bovis* BCG infection, we had found increase in the expression of SUV39H1 (KMT1A), the histone H3K9 methyltransferase. In addition to being overexpressed in infected cells, SUV39H1 was also found to be predominantly localised in the cytoplasm and the cell surface.

Further examination of SUV39H1 localisation in the cytoplasm indicated that it was localized to the phagosomal fraction where it was found to be interacting with the mycobacterial bacilli (Figure 2A). SUV39H1 is a histone methyltransferase that specifically methylates histone H3 lysine 9 in the host nucleus. We were intrigued with its colocalisation and binding with *M. bovis* BCG in the phagosomes. In the literature it is known that mycobacterium binds to host cells through alpha-laminin. In this interaction, mycobacterial protein HupB binds to alpha-laminin (HupB or Rv2986c

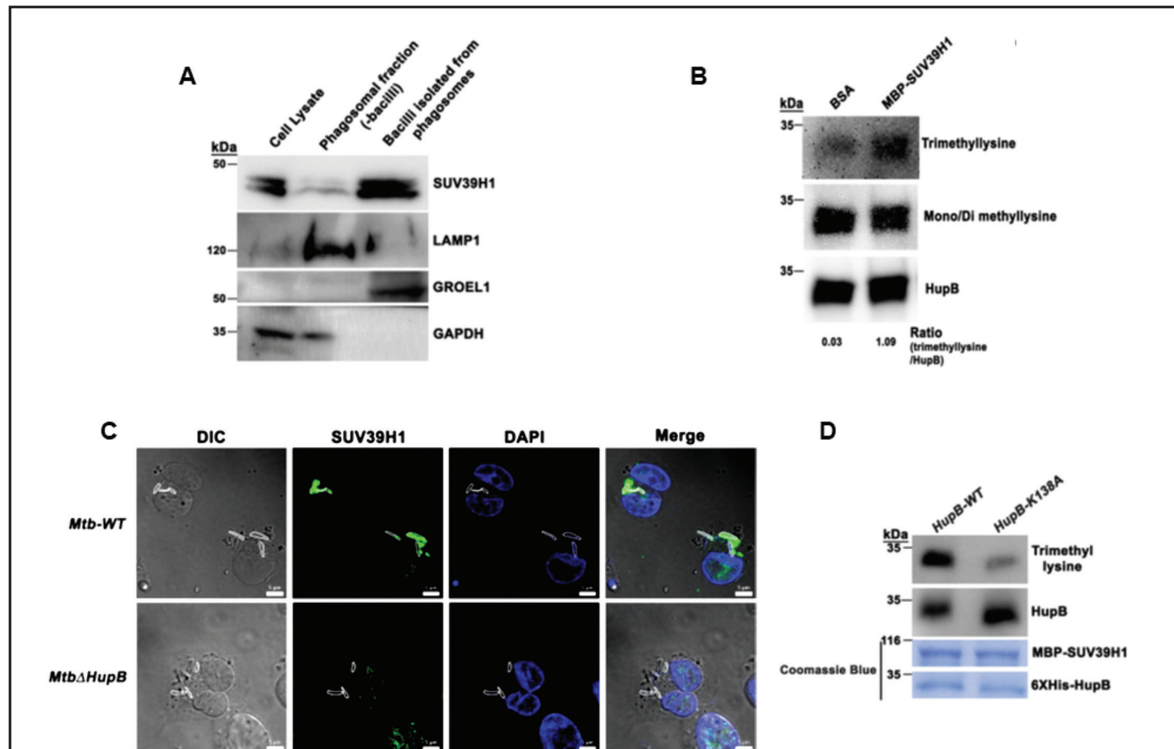


Figure 2: SUV39H1 trimethylates HupB at lysine 138. A.) *M. bovis* BCG bacilli were isolated from the phagolysosomal fraction of infected THP1 macrophages and examined for SUV39H1 by western blotting. To rule out contamination by cytoplasmic and phagosomal host proteins, the western blot was probed for LAMP1 (phagosomal marker) and GAPDH (cytoplasmic marker). The blot was also probed for GroEL1, a mycobacterial protein B.) SUV39H1 trimethylates HupB. Recombinant MBP-SUV39H1 or BSA (control) and *M. bovis* BCG lysate were incubated in presence of SAM followed by western blotting with mono/di methyl lysine (middle panel) or trimethyl lysine (upper panel) specific antibodies. The blot was also re probed with HupB antibody. The ratio of the normalised signal for trimethyllysine antibody and HupB antibody is given below the panels. C. THP1 macrophages infected with wild type *M. tuberculosis* H37Rv or *MtbΔhupB* strains were examined for the localisation of SUV39H1 protein. The outline of some of the internalised mycobacterial bacilli as observed in DIC is marked in white. The cells were counterstained with DAPI. Scale bar - 5 μ m. D.) Recombinant 6XHis-HupB (Hup-WT) or 6XHis-hupBK138A (HupB-K138A) proteins expressed and purified from *M. smegmatis* (lowermost panel) were incubated with recombinant MBP-SUV39H1 (second panel from bottom) in presence of SAM, western blotted and probed with Trimethyl lysine and HupB (control) antibodies.

is also known as Laminin Binding Protein, LBP). Interestingly, HupB is histone like protein from mycobacteria and has been shown to be present both in the mycobacterial cytosol and on the cell wall. Methyltransferase assay with SUV39H1 and *M. bovis* BCG lysate in presence of SAM as a methyl group donor was analysed by western blotting using either mono/di methyl lysine or trimethyl lysine specific antibodies. Only one band, which corresponded to HupB, was detected in the whole blot when the trimethyl antibody was used as a probe (Figure 2B). Since SUV39H1 was found associated with mycobacterial bacilli in the phagosomes we also probed whether this association was through interaction of SUV39H1 with HupB, THP1 macrophages were infected with wildtype and HupB mutant (in which hupB gene had been deleted, *MtbΔhupB*) *M. tuberculosis* H37Rv strains. SUV39H1 antibody stained mycobacterial bacilli (marked by white outline) in the wild type *M. tuberculosis* H37Rv strain but not in *MtbΔhupB* strain (Figure 2C)

SUV39H1 methylates lysine at position 9 in the histone H3. Using the amino acid sequence motif (TK--ARK---KAP) that encompasses H3K9, we found that this motif was present only in the *M. tuberculosis* HupB protein (amino acid 132 to 144, Figure 5E and EV3A). To test whether SUV39H1 methylates HupB protein at the lysine present within the central ARK motif (K138), lysine at

138 position in recombinant 6XHis-HupB was mutated to alanine (6XHis-HupB^{K138A}) by site-directed mutagenesis. A significant decrease in the level of HupB methylation was observed for 6XHis-HupB^{K138A} mutant as compared to 6XHis-HupB (Figure 2D).

Publications:

In 2017

1. Dev RR, Ganji R, Singh SP, Mahalingam S, Banerjee S, Khosla S (2017) Cytosine methylation by DNMT2 facilitates stability and survival of HIV-1 RNA in the host cell during infection. **Biochemical Journal** 474: 2009-2026.

In 2018

1. Yaseen I, Choudhury M, Sriharan M, Khosla S. (2018) Histone methyltransferase SUV39H1 participates in host defense by methylating mycobacterial histone-like protein HupB. **EMBO Journal** 37:183-200.
2. Anwar T, Sen B, Aggarwal S, Nath R, Pathak N, Katoch A, Aiyaz M, Trehanpati N, **Khosla S**, Ramakrishna G. (2018) Differentially regulated gene expression in quiescence versus senescence and identification of ARID5A as a quiescence associated marker. **Journal of Cell Physiology** 233:3695-3712.

LABORATORY OF MOLECULAR CELL BIOLOGY

Signal transduction pathways in macrophages and host-pathogen interaction in tuberculosis

Faculty	Sangita Mukhopadhyay	Staff Scientist
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	Vishwanath Jha	Senior Research Fellow
	Komal Dolasia	Senior Research Fellow
	Shruti Srivastava	Senior Research Fellow
	KM Rohini	Senior Research Fellow
	Ravi Pal	Junior Research Fellow
	Manoj Kumar	Junior Research Fellow
	Priyanka Dahiya	Junior Research Fellow (since July 2017)
Other Members	R. Nagender Rao	DST-SERB Project Investigator
	Niteen Pathak	Senior Technical Officer
	Philip Abraham	Research Associate
	Madhu Babu Battu	DST- National Postdoctoral Fellow
	Rahila Qureshi	ICMR Senior Research Fellow
	Faiza Nazar	Project Assistant
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	Dr G. Narahari Sastry	IICT, Hyderabad
	Dr. Sudip Ghosh	NIN, Hyderabad
	Dr. JRC Reddy	IICT, Hyderabad
	Prof. Anand Kondapi	University of Hyderabad, Hyderabad
	Dr. Gaddam Sumanlatha	Osmania University, Hyderabad
	Dr. Vijaya Laxmi Valluri	Bhagwan Mahavir Medical Research Centre, Hyderabad
	Dr. Vinay Nandikoori, NII	New Delhi

Objectives

Signal transduction pathways in macrophages regulating its innate-effector functions and how various candidate proteins of *Mycobacterium tuberculosis* (Mtb) interfere with macrophage signaling cascades to modulate host's protective responses against the bacilli.

Project I: Mycobacterial PknG inhibits phagosome-lysosome (P-L) fusion by targeting the Rab711

Details of progress made in the current reporting year (April 1, 2017 - March 31, 2018)

The mycobacterial protein kinase G (PknG) is a serine/threonine kinase and is known to be secreted and released into the cytoplasm of bacteria-infected macrophages. PknG acts as an important virulent factor inhibiting P-L fusion.

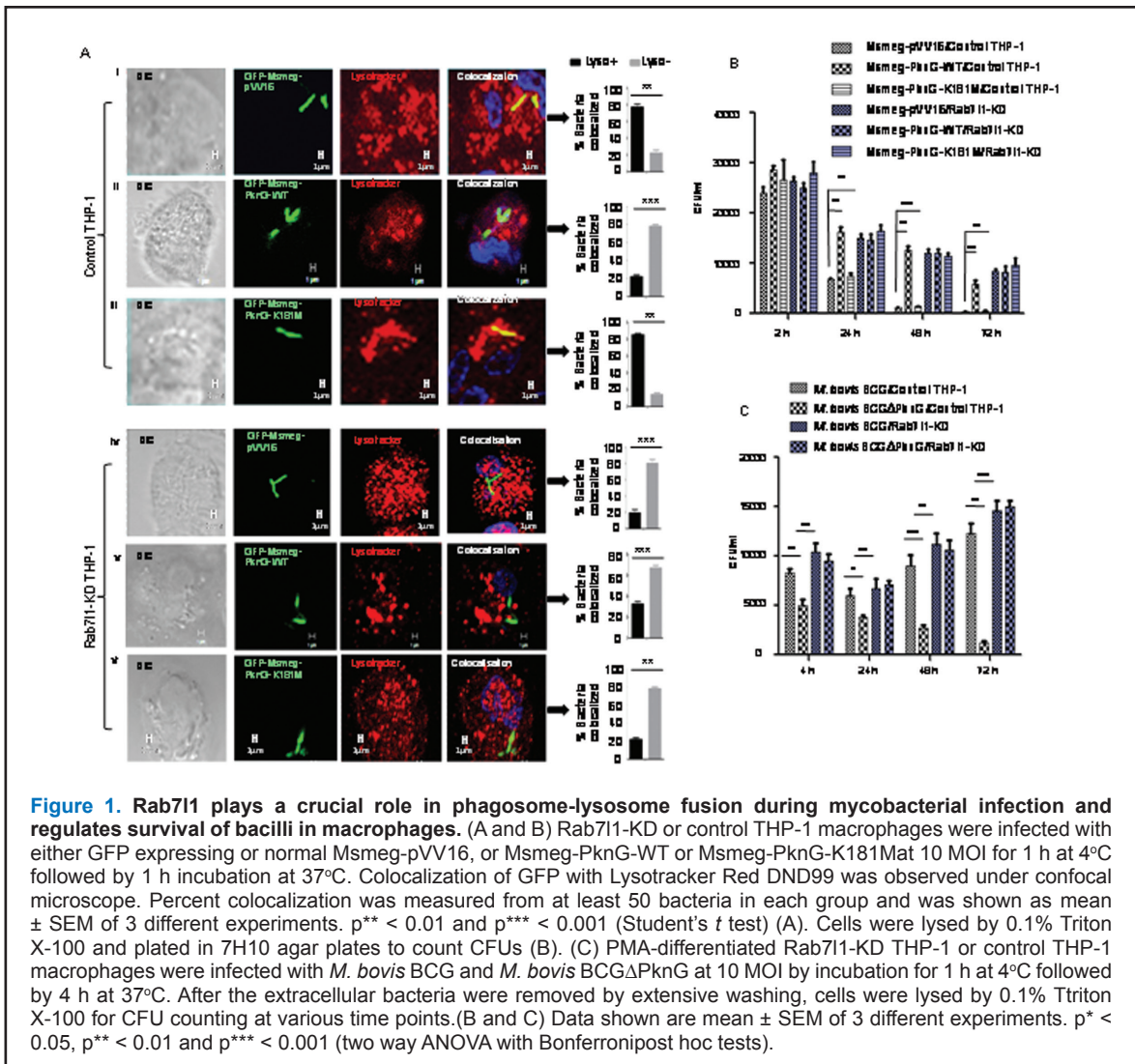
Interestingly, the kinase activity of PknG was found to be critical for inhibition of P-L fusion. However, the exact mechanism by which PknG inhibits P-L fusion during mycobacterial infection remains poorly understood. It is possible that PknG directly interacts with key host proteins involved in phagosomal maturation process and inhibits P-L fusion. To validate our hypothesis, in the present study, we carried out protein-protein interaction studies using a yeast two hybrid (Y2H) system where a leukocyte library was screened using Mtb PknG as bait and were able to confirm that a host RabGTPase protein Rab711 (Rab29 in mouse), is an interacting partner of PknG.

Rab711 plays a crucial role in P-L fusion and survival of mycobacteria in macrophages:

Since we observed that PknG could physically interact with Rab711, we speculated that Rab711

probably plays a role in P-L fusion and PknG targets the Rab711 to inhibit P-L fusion. Therefore, we generated a Rab711 knock-down stable THP-1 cell line (Rab711-KD) using Rab711-specific shRNA. Rab711-KD or control THP-1 cells were differentiated into macrophages using PMA and infected with GFP expressing *M. smegmatis* (which lacks PknG expression) carrying either PknG-WT (Msmeg-PknG-WT) or PknG-K181M (Msmeg-PknG-K181M, a PknG mutant carrying a point mutation in which the lysine residue at position 181 is substituted with methionine), was also used. The K181M mutation in the kinase domain of PknG makes it functionally defective for its kinase activity or harboring the backbone vector alone (Msmeg-pVV16). Colocalization of bacteria associated GFP fluorescence was monitored to a marker of acidic compartment (LysoTracker Red DND-99) after 1 h of infection.

Expectedly, green fluorescence associated with Msmeg-pVV16 and Msmeg-PknG-K181M was found to be predominantly colocalized with LysoTracker Red in control THP-1 macrophages (Figure 1Ai, 1Aiii). However, a significant reduction in colocalization with LysoTracker Red was observed in case of Msmeg-PknG-WT in these cells (Figure 1Aii) indicating a role of PknG in avoiding P-L fusion by the infecting bacteria as reported by others. Interestingly, none of these strains were found to be significantly colocalized with LysoTracker Red in Rab711-KD THP-1 macrophages (Figure 1Aiv-1Avi). The inability of the nonpathogenic *M. smegmatis* strains to be localized in lysosomes, whether carrying PknG or not, in Rab711 knock-down cells indicate that Rab711 may play a role in P-L fusion and trafficking of mycobacteria into the lysosomes.



Since inhibition in P-L fusion was found to be directly correlated with intracellular survival of mycobacteria, next we examined whether *M. smegmatis* or PknG-deficient *M. bovis* BCG (*M. bovis* BCG Δ PknG) that are known to be eliminated efficiently by the activated macrophages could survive better in Rab711-KD macrophages. As expected, Msmeg-PknG-WT was found to survive better than Msmeg-pVV16 in control THP-1 macrophages. However, in case of Msmeg-pVV16 and Msmeg-PknG-K181M, an increased CFU count was observed in Rab711-KD macrophages as compared to control THP-1 macrophages (Figure 1B). Also, expectedly, *M. bovis* BCG Δ PknG had significantly lower CFU counts as compared to *M. bovis* BCG strain in control THP-1 cells highlighting a role of PknG in regulating intracellular survival of bacteria (Figure 1C). However, in Rab711-KD macrophages both these strains had no significant differences in survival and also the CFU counts of *M. bovis* BCG Δ PknG were significantly higher when compared with control THP-1 macrophages (Figure 1C). Taken together, these observations suggest that mycobacterial PknG confers a survival advantage to the bacteria inside the macrophages possibly by regulating the Rab711 function.

PknG inhibits Rab711 GTPase activity by blocking transition of Rab711-GDP to Rab711-GTP:

Since we observed the role of Rab711 in P-L fusion, the probable mechanism by which PknG manipulates Rab711 signaling pathway to block P-L fusion was investigated next. PknG did not affect endogenous expression of Rab711 to inhibit P-L fusion. Also PknG failed to directly phosphorylate Rab711 *in vitro*. Thus, PknG-mediated inhibition of P-L fusion is not due to either reduction in the cellular levels of Rab711 or phosphorylation of Rab711. As Rab711 is a known RabGTPase, we examined whether PknG prevented Rab711 function by inhibiting its GTPase activity. Accordingly, THP-1 macrophages were infected with either Msmeg-pVV16 or Msmeg-PknG-WT or Msmeg-PknG-K181M and GTPase assay was carried out. Interestingly, Msmeg-PknG-WT-infected cells had significantly reduced levels of GTPase activity as compared to those infected with Msmeg-pVV16 or Msmeg-K181M. When Rab711 was pulled down using GTP-agarose beads from these infected cells, level of GTP-bound Rab711 was found to be reduced in cells infected with Msmeg-PknG-WT as compared

to cells either expressing PknG-K181M or the backbone vector alone. Similarly, when THP-1 macrophages were infected with *M. bovis* BCG or *M. bovis* BCG Δ PknG for 2 h and 4 h, GTPase activity was significantly decreased in cells infected with *M. bovis* BCG as compared to cells infected with *M. bovis* BCG Δ PknG at both the time points. Decreased Rab711 GTPase activity was associated with reduced levels of GTP-bound Rab711 in *M. bovis* infected cells. These results indicated that GTPase activity of Rab711 was inhibited by PknG and the kinase function of PknG was essential to inhibit Rab711 GTPase activity. These data together hint a role of PknG-WT in blocking conversion of inactive Rab711-GDP to active Rab711-GTP and thus explain observed lower GTPase activity of Rab711 in the presence of kinase active PknG. Therefore, we speculated that PknG probably interacts with the Rab711-GDP to interfere its conversion to GTP-bound form of Rab711. To further confirm this hypothesis, constitutively active (Q67L, remain locked in the GTP-bound conformation, deficient in GTPase activity) and inactive (T21N, unable to bind GTP, remain locked in the GDP-bound state) mutants of Rab711 were generated and interaction of PknG-WT and PknG-K181M with these mutants was examined by Y2H one to one interaction assay. Interestingly, specific interaction of PknG-WT and PknG-K181M was observed with GDP-bound Rab711T21N but not with GTP-bound Rab711Q67L.

Earlier studies have demonstrated that Rab711-GDP is present in the trans-Golgi network (TGN) whereas Rab711-GTP is primarily localized in the cytosol and our study showed that PknG is translocated to trans-Golgi network where it interacts with Rab711-GDP. The GTP-bound Rab711 was found to be predominantly recruited to phagosomes, which was crucial for subsequent recruitment of phagosomal-lysosomal markers like EEA1, Rab7 and LAMP2 leading to P-L fusion. Thus it appears that Rab711 plays a critical role in phagosomal maturation process. PknG interacts with Rab711-GDP and inhibits Rab711-GDP/GTP transition, resulting in reduced recruitment of Rab711-GTP and subsequently recruitment of key phago-lysosomal markers (EEA1, Rab7, LAMP2), thus resulting in inhibition of phagosome-lysosome fusion.

Future studies

Our future studies are aimed at i) deciphering the mechanism by which the kinase activity of PknG is responsible for manipulation of Rab711 function and ii) how Rab711-GTP gets recruited

to phagosomes and plays a role in regulating phagosome-lysosome fusion process.

Project II: PPE18 protein of *M. tuberculosis* (Mtb) as a therapeutic to treat septicemia

Summary of work done until the beginning of this reporting year

We have earlier demonstrated that an Mtb protein belonging to the PPE family, PPE18 binds to TLR2 and causes IL-10 induction in macrophages *via* activation of p38 MAPK. Also, its interaction with TLR2 leads to phosphorylation of SOCS3 which then physically interacts with the I κ B α -NF- κ B/rel complex, thus preventing phosphorylation and degradation of I κ B α and nuclear translocation of p50 and p65 NF- κ B and c-rel transcription factors. As a consequence of this, there is downregulation of transcription of NF- κ B regulated genes like IL-12 and TNF- α . PPE18 selectively downregulates pro-inflammatory immune responses. These properties of PPE18 can be exploited to dampen the effects of extreme inflammation observed in situations such as sepsis. With this rationale, we decided to test rPPE18 (recombinantly purified PPE18) as a therapeutic agent in a mouse models of sepsis. Our studies revealed that treatment of mice with rPPE18 reduces TNF- α level, generates M2 macrophage, improves clinical symptoms and survival of mice suffering from septicemia induced by intraperitoneal injection of a high dose of *E. coli*. We next checked if rPPE18 reduced TNF- α , ALT, creatinin and organ damage; and improved survival in a mouse model of CLP-induced polymicrobial sepsis.

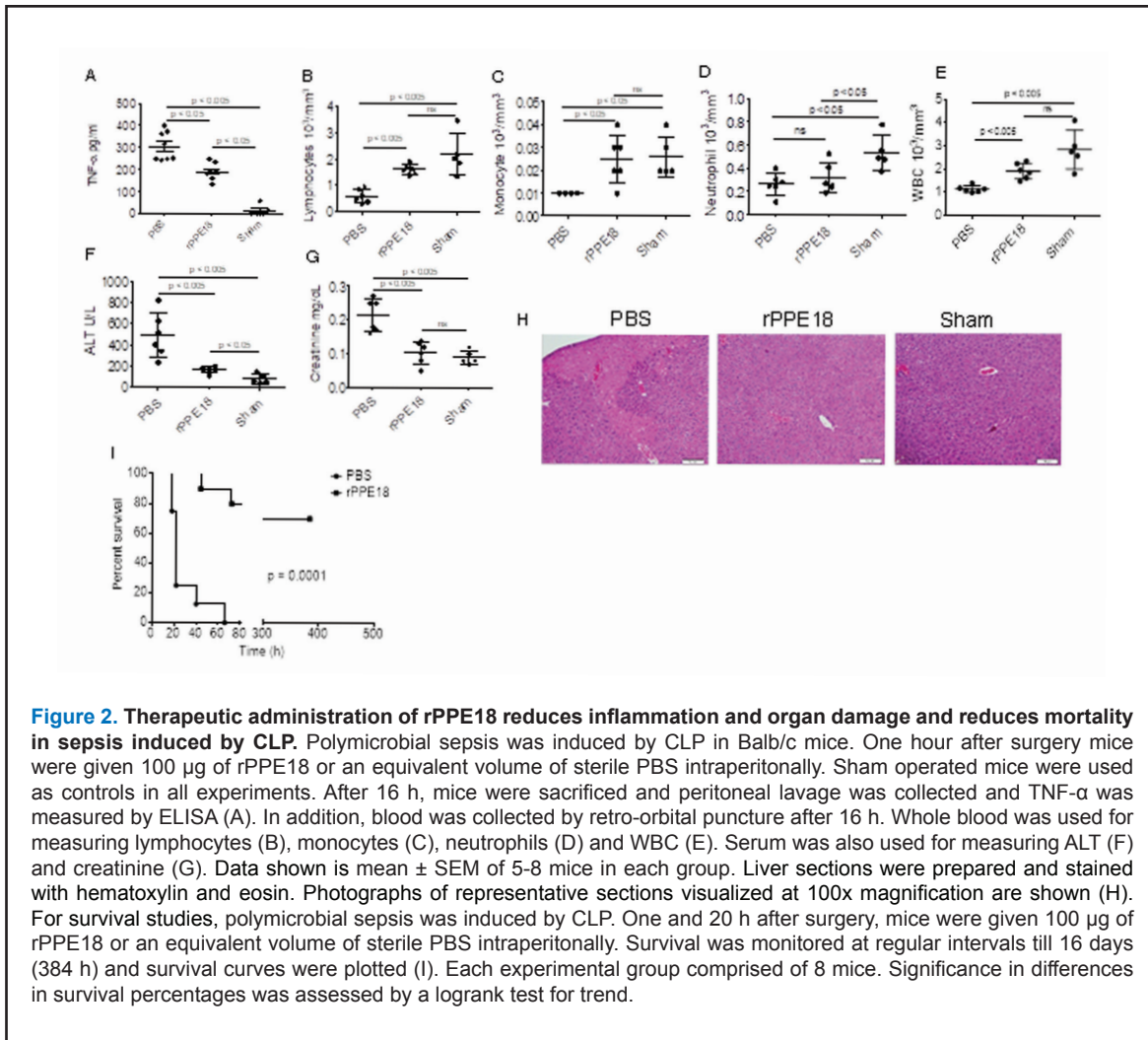
Details of progress made in the current reporting year (April 1, 2017 - March 31, 2018)

Administration of PPE18 improves survival in a mouse model of polymicrobial sepsis induced by cecal ligation and puncture (CLP): In all our studies so far, we had used a system where intra-peritoneal injection of a high doses of *E. coli* BL21 was used to induce sepsis. Administration of high doses of *E. coli* BL21 lead to a rapid rise in TNF- α and resulted in 100 % mortality by 40 h. This model allowed us to study the effects of rPPE18 and dissect its mechanism of action in sepsis. However, we next wished to study the effect of rPPE18 in polymicrobial sepsis induced by CLP which is a more physiological model of sepsis. In this model, the effects of recombinantly purified PPE18 were studied on TNF- α ; WBC; numbers of lymphocytes, monocytes and neutrophils; liver and kidney function and ultimately on survival.

Polymicrobial sepsis induced by CLP increased levels of TNF- α in the peritoneal lavage which were significantly reduced in mice which received rPPE18 therapy (Figure 2A). We however, could not measure TNF- α in blood serum. This is consistent with earlier reports. CLP also resulted in dramatic reduction in numbers of lymphocytes (Figure 2B), monocytes (Figure 2C), and neutrophils (Figure 2D). This reduction in numbers was reflected in the decrease in WBC post CLP induced sepsis (Figure 2E). Administration of rPPE18 restored lymphocyte (Figure 2B), monocyte (Figure 2C) and WBC (Figure 2E) numbers comparable to sham operated mice. CLP also resulted in reduction in blood neutrophil levels. However, neutrophil numbers were not significantly but marginally higher in rPPE18 treated mice (Figure 2D).

As CLP is known to cause liver and kidney damage, we next assessed liver and kidney function by measuring serum ALT and creatinine levels. We observed increase in serum ALT (Figure 2F) and creatinine (Figure 2G) levels 16 h post CLP. Therapeutic administration of rPPE18 significantly reduced elevated ALT and creatinine levels. This effect on ALT levels was also reflected in differences in liver pathology between the two groups of animals. Mice subjected to CLP but not treated with rPPE18 showed marked necrosis and liver damage compared to sham operated mice while rPPE18 treated CLP mice had more hypertrophy (Figure 2H), which can be a non-adverse effect and hypervacuolation which may be a host protective responses. These data suggest that rPPE18 can provide protection from polymicrobial sepsis-induced organ damage.

Finally, we studied the effect of rPPE18 on survival of mice subjected to CLP-induced polymicrobial sepsis. Mice which received PBS post CLP had a median survival time of 22 h while those that received rPPE18 had significantly improved survival. In the rPPE18 treatment group, 75% mice were still surviving when the last death in the PBS group was registered at approximately 60 h post CLP (Fig. 6I). Mice were monitored till 16 days and the survival percentage in rPPE18 treated group at this time was 70% (Fig. 6I). These data indicate that rPPE18 by virtue of its ability to reduce TNF- α levels and prevent organ damage can provide protection in polymicrobial sepsis. Importantly, results from these experiments validate previous observations made using the model of *E. coli* BL21 induced septic shock.



Future Studies: We next aim to use peptide fragments of PPE18 which are as effective as the full length protein in the endotoxic septic shock treatment. Also we plan to use a pharmaceutically acceptable carrier (nanoparticle) for this treatment as nanoparticles are delivery systems that enhance stability.

Publications

i) Research papers published in the calendar year 2017

1. Bhat KH, Srivastava S, Kotturu SK, Ghosh S and Mukhopadhyay S. (2017). The PPE2 protein of *Mycobacterium tuberculosis* translocates to host nucleus and inhibits nitric oxide production. **Scientific Reports** 7: 39706.
2. Abraham PR, Pathak N, Pradhan G, Sumanlatha G and Mukhopadhyay S. (2017). The N-terminal domain of *Mycobacterium*

tuberculosis PPE17 (Rv1168c) protein plays a dominant role in inducing antibody responses in active TB patients. **PLoS ONE** 26: e0179965.

3. Mukhopadhyay S* and Ghosh S*. (2017). *Mycobacterium tuberculosis*: what is the role of PPE2 during infection? **Future Microbiology** 12: 457-460 (Invited Editorial Article).
4. Rameshwaram NR*, Shrivastava R*, Pradhan G*, Singh P and Mukhopadhyay S. Phagosome-lysosome fusion hijack - An art of intracellular bacteria. (2017). **Proceedings of the Indian National Academy of Sciences** 83: 533-548.

ii) Research papers published in 2017-18 (until 31st March 2018 only)

1. Dolasia K, Bisht MK, Pradhan G, Udgata A and Mukhopadhyay S. (2018). TLRs:

Shaping the landscape of host immunity.
International Reviews of Immunology 37:
3-19.

**(iii) Research papers in press as on 31st
March 2018**

1. Singh P*, Rameshwaram NR*, Ghosh S and Mukhopadhyay S. (2018). Cell envelope lipids

in the pathophysiology of *Mycobacterium tuberculosis*. *Future Microbiology*.

2. Ahmed A*, Dolasia K* and Mukhopadhyay S. (2018). *Mycobacterium tuberculosis* PPE18 protein reduces inflammation and increases survival in animal model of sepsis. *Journal of Immunology*.

*Equal contribution

LABORATORY OF MOLECULAR ONCOLOGY

Genomics and molecular genetics of cancer

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	Pratyusha Bala	Senior Research Fellow
	Ashmala Naz	Senior Research Fellow
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	Diya Sarkar	Junior Research Fellow (since Feb. 2018)
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	Raju Adduri Sita Rama	Research Associate (till Dec. 2017)
	Viswakalyan Kotapalli	Project Technical Assistant
	Padmavathi Kavadiyala	Project JRF
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Objectives

Identification and characterization of important deregulated genes/pathways in cancers prevalent in India.

Summary of work done until the beginning of this reporting year (upto March 31, 2017)

Tongue cancer: Up-regulation of *TP53* transcript in tumor samples was possibly due to the action of ZMAT3. *SMARCD1* was confirmed to be a novel transcriptional target of non-hotspot mutant p53.

Colorectal cancer (CRC): Ca²⁺/NFAT signalling was discovered to be enriched in Wnt- rectal cancer samples and to promote tumorigenic

features in CRC cell lines. *XPNPEP3* was identified as a putative transcriptional target of canonical Wnt/ β -catenin signalling.

Details of progress made in the current reporting year (April 1, 2017 - March 31, 2018)

Tongue cancer: We confirmed elevated expression of *SMARCD1* in tumor compared to normal tongue cancer samples using quantitative reverse transcription PCR in HNSCC (Figure 1A-C) and in other cancer types (Figure 1D). More importantly, increased *SMARCD1* expression predicted poor survival in HNSCC tumors harboring missense p53 mutation (Figure 1E). Thus, our results reveal *SMARCD1* to be a novel oncogenic target of mutant p53.

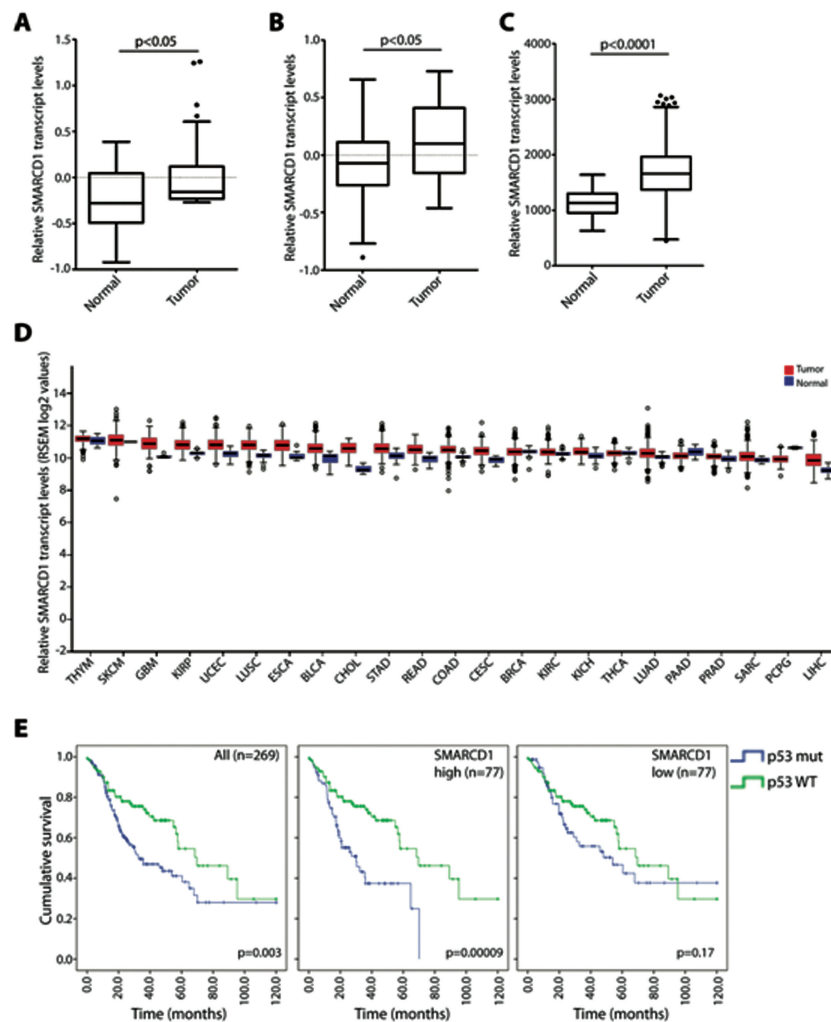
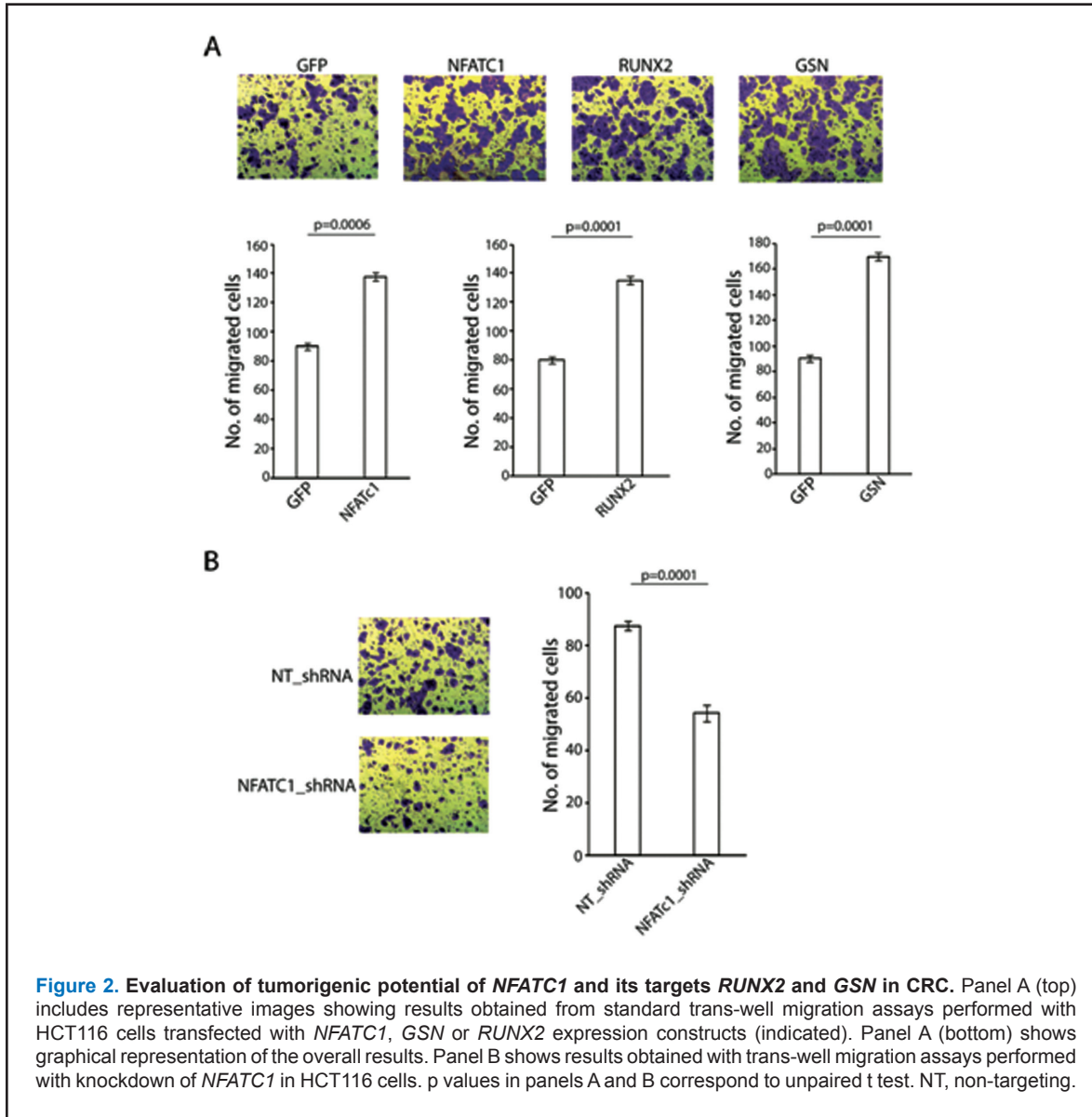


Figure 1. Clinical relevance of possible oncogenic role of *SMARCD1*. Panels A-D show increased *SMARCD1* expression in tumor compared to normal tissue samples. Panel A shows results of Q-PCR analysis of *SMARCD1* transcript levels performed on SCCOT and normal tongue tissue samples. Panel B shows comparison of *SMARCD1* mRNA levels in SCCOT tumor and matched normal samples ascertained from microarray-based genome-wide expression data generated in another study (Krishnan et al., 2015). Panels C and D show results of computational analysis of *SMARCD1* transcript levels performed on tumor vs normal samples in HNSCC (panel C) and several other cancers (panel D) on the Firebrowse database. Cancer types in panel D: THYM, thymus; SKCM, Skin Cutaneous Melanoma; GBM, Glioblastoma; KIRP, Kidney Renal Papillary Cell Carcinoma; UCEC, Uterine Corpus Endometrial Carcinoma; LUSC, Lung Squamous Cell Carcinoma; ESCA, Esophageal Carcinoma; BLCA, Bladder Urothelial Carcinoma; CHOL, Cholangiocarcinoma; STAD, Stomach Adenocarcinoma; READ, Rectal Adenocarcinoma; COAD, Colon Adenocarcinoma; CESC, Cervical Squamous Cell Carcinoma and Endocervical Adenocarcinoma; BRCA, Breast Carcinoma; KIRC, Kidney Renal Clear Cell Carcinoma; KICH, Kidney Chromophobe; THCA, Thyroid Carcinoma; LUAD, Lung Adenocarcinoma; PAAD, Pancreatic Adenocarcinoma; PRAD, Prostate Adenocarcinoma; SARC, Sarcoma; PCPG, Pheochromocytoma and Paraganglioma; LIHC, Liver Hepatocellular Carcinoma. p values correspond to unpaired (panels A and C) and paired (panel B) t test. Panel E shows survival analysis performed using the TCGA data on HNSCC samples. Left panel includes all samples stratified for p53 status (samples harboring missense mutant (n=155) or wild type (n=114) p53); middle panel includes all p53 WT samples and p53 mutant samples with *SMARCD1* levels above median z-score (n=77; *SMARCD1* high) whereas the right panel includes all p53 WT samples and p53 mutant samples with *SMARCD1* levels below median z-score (n=77; *SMARCD1* low). p values correspond to the log rank test.

CRC:

We evaluated ability of *NFATC1* and its transcriptional targets identified in our earlier study to modulate tumorigenic properties in CRC cells using both shRNA based knock down and

ectopic expression of *NFATC1*. Results revealed that *NFATC1* and its targets *GSN* and *RUNX2* significantly increased the migratory potential of CRC cells (Figure 2) thus confirming role of Ca^{2+} /NFAT signalling as a possible regulator of tumorigenesis in CRC.



In a separate study, ectopic expression of *XPNPEP3* promoted tumorigenic properties in CRC cells (Figure 3A). Immunohistochemistry on a CRC tissue microarray revealed increased expression in tumor compared to matched normal samples (Figure 3B). More importantly, *XPNPEP3* expression exhibited significant correlation with β -catenin nuclear localization status (Figure 3B-D). In addition, *XPNPEP3*

expression was upregulated in tumor compared to normal samples in published gene expression datasets for several cancers including CRC (Table 1). Finally, *XPNPEP3* expression correlated with poor survival in many cancers (Figure 3E). Our results therefore suggest *XPNPEP3* to be a novel transcriptional target of canonical Wnt/ β -catenin signalling with particular significance for CRC.

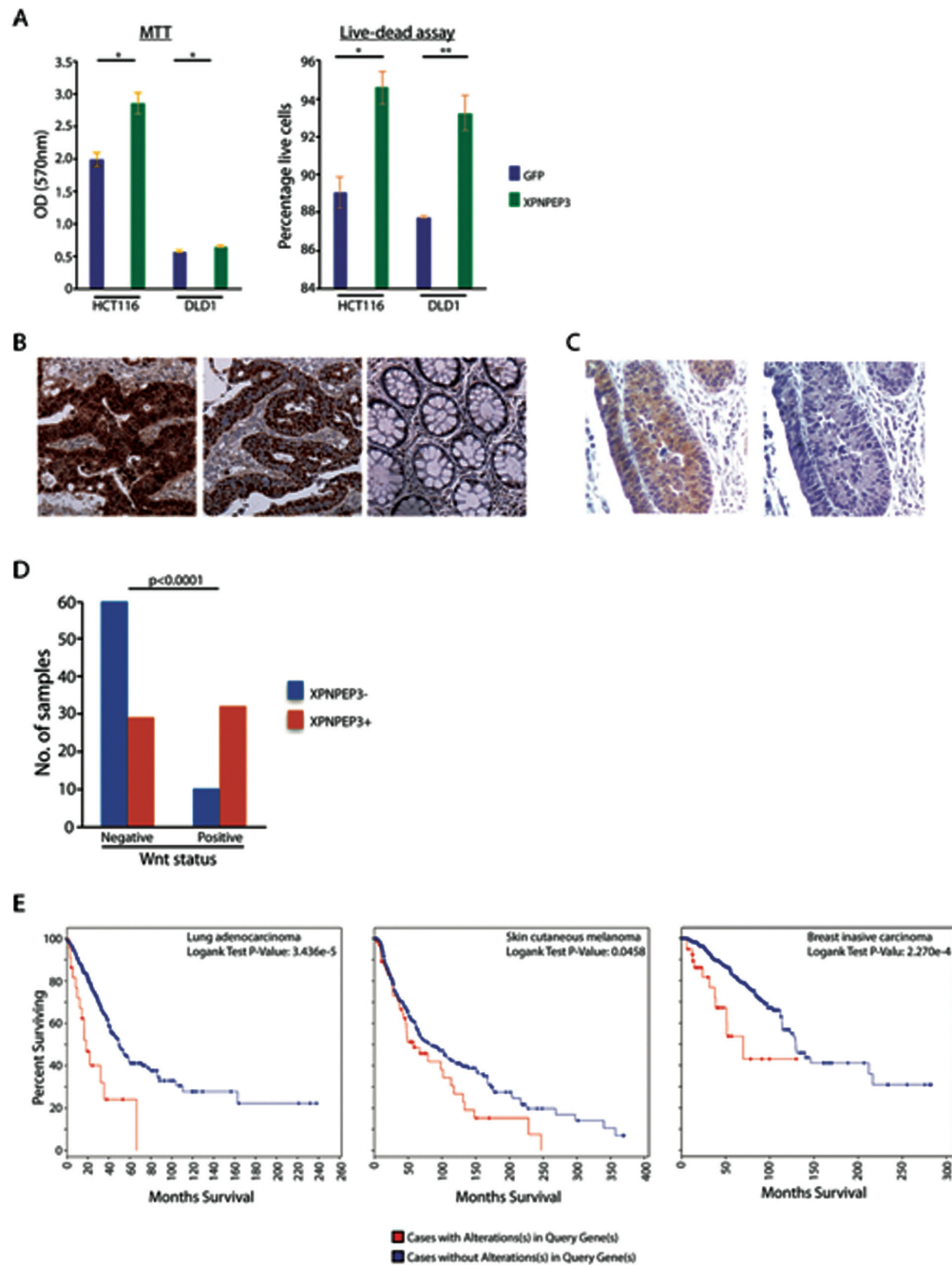


Figure 3. Analysis of XPNPEP3 expression in tumor samples. Panel A shows result of MTT (left) and live-dead (right) assays following ectopic expression of XPNPEP3 in two different CRC cell lines (indicated). GFP ectopic expression was used as control. p values correspond to unpaired 't' test (two tailed); *, <0.05 and **, <0.01. Panels B-D show results of CRC-TMA based analysis of XPNPEP3 and β -catenin expression. Panel B shows IHC results for a representative Wnt+ rectal cancer sample: tumor with nuclear positive β -catenin (left) and elevated expression for XPNPEP3 (middle). Result for the matched normal sample exhibiting negligible XPNPEP3 stain is shown on the right. Panel C shows IHC results for a representative Wnt- rectal cancer sample: tumor with nuclear negative β -catenin (left) and weak stain for XPNPEP3 (right). Panel D shows graphical representation of comparative analysis of XPNPEP3 and β -catenin expression in the TMA; fisher's exact test p-value is shown. Panel E depicts effect of XPNPEP3 on overall survival Kaplan-Meier estimate performed at the cBioPortal (www.cbioportal.org) for three different cancers (indicated).

Table 1. Up-regulation of *XPNPEP3* expression in tumor vs normal samples analyzed from the Oncomine database.

Datasets ^a	Analysis	Fold up-regulation (T vs N) ^b	p value
Sun Brain data set	Anaplastic Astrocytoma v/s Normal	2.87	7.55 E-5
Radvanyi Breast data set	Invasive mixed Breast carcinoma v/s Normal	5.87	0.011
	Invasive ductal Breast carcinoma v/s Normal	3.47	0.025
TCGA colorectal data set	Rectosigmoid adenocarcinoma v/s Normal	2.03	2.95 E-7
Sabates – Bellver colon data set	Rectal adenoma v/s Normal	2.99	7.74 E-6
	Colon adenoma v/s Normal	2.82	7.74 E-4
Zhan myeloma data sets	Soldering myeloma v/s Normal	2.83	5.51 E-6

^aExpression data-sets from the Oncomine database

^bRepresents fold increase of *XPNPEP3* transcript in tumor vs normal samples for the particular cancer type

Future plans and directions

1. Characterization of novel transcriptional targets of non-hotspot mutant p53.
2. Characterization of Ca²⁺/NFAT signalling pathway in Wnt- rectal cancer.

Publications

Research papers published in 2017

1. Chaudhary AK, Mohapatra R, Nagarajaram HA, Ranganath P, Dalal A, Dutta A, Danda S, Girisha KM and Bashyam MD (2017). The novel EDAR p.L397H missense mutation causes autosomal dominant hypohidrotic ectodermal dysplasia. *Journal of the European Academy of Dermatology and Venereology* 31:e17-e20.
2. Kumar R and Bashyam MD (2017). Multiple oncogenic roles of nuclear beta catenin. *Journal of Biosciences*, 42: 695-707.

Research papers published in 2018 (upto March 31, 2018)

Kumar R, Raman R, Kotappali V, Gowrishankar S, Pyne S, Pollack JR, Bashyam M (2018). Ca²⁺/

Nuclear Factor of Activated T cells signaling is enriched in early-onset rectal tumors devoid of canonical Wnt activation. *Journal of Molecular Medicine* 96: 135-146.

Research papers published in 2018 (in press upto March 31, 2018)

Kumar R, Kotapalli V, Naz A, Gowrishankar S, Rao S, Pollack JR, and Bashyam MD (2018). *XPNPEP3* is a novel transcriptional target of canonical Wnt/ β -catenin signalling. *Genes Chromosomes & Cancer*.

References:

N. Krishnan, S. Gupta, V. Palve, L. Varghese, S. Pattnaik, P. Jain, C. Khyriem, A. Hariharan, K. Dhas, J. Nair, M. Pareek, V. Prasad, G. Siddappa, A. Suresh, V. Kekatpure, M. Kuriakose, B. Panda, Integrated analysis of oral tongue squamous cell carcinoma identifies key variants and pathways linked to risk habits, HPV, clinical parameters and tumor recurrence, *F1000Res*, 4 (2015) 1215.

LABORATORY OF NEUROSPORA GENETICS.

Principal Investigator	Durgadas P. Kasbekar	Haldane Chair (till July 2017) INSA Senior Scientist (since July 2017)
PhD Student	Dev Ashish Giri	SRF
Other Members	A. Sheeba S. Rekha	Technical Officer II (till July 2017) Technical Assistant (till Aug. 2017)

Project: Efficient meiotic silencing of unpaired DNA (MSUD), though characteristic of the *Neurospora crassa* standard Oak Ridge (OR) strains, is atypical in *Neurospora*.

Objective: To understand why meiotic silencing of unpaired DNA (MSUD) is more efficient in *tester*^{OR} x OR crosses than in *tester*^{OR} x wild-strain crosses.

MSUD is an RNAi-mediated process that in the standard *Neurospora crassa* OR genetic background efficiently silences any gene that is not properly paired with its homologue during the meiosis of a sexual cross. The unpaired sequences are transcribed into 'aberrant RNA' that is made double-stranded and then processed into single-stranded MSUD-associated small interfering RNA (masiRNA) for use by a silencing complex to degrade complementary mRNA. MSUD does not occur in homozygous *tester A* x *tester a* crosses. Our laboratory has previously found that MSUD is often decidedly less efficient in crosses of the OR-derived MSUD tester strains with most (i.e. 72/80) wild-isolated strains (W). Either sequence heterozygosity in *tester* x W crosses suppresses MSUD (model 1), or the OR background represents the MSUD-conducive extreme in the range of genetic variation in MSUD efficiency (model 2). We tested MSUD in near-isogenic crosses made in a novel *N. crassa* wild-derived genetic background, called B/S, and also in the related species *N. tetrasperma*. If model 1 was correct, we would expect these crosses to display efficient MSUD, as in OR. But if model 2 was correct then isogenicity would have no effect, and MSUD would remain as inefficient in the B/S background as it was in the wild strains from which it was derived.

Summary of work done until the beginning of the reporting year (upto March 31, 2017). Of 80 wild-isolated strains tested in crosses with the OR-derived MSUD testers (*tester*^{OR}), only eight, designated as the "OR" type wild strains, showed a silencing phenotype comparable to that in *tester*^{OR} x OR crosses. Crosses with four wild

strains, designated the "Sad" type, failed to show MSUD and the remaining 68 strains showed an intermediate phenotype. One hypothesis (model 1) to explain these results is that genome sequence heterozygosity in the *tester*^{OR} x wild crosses causes a natural asynapsis and self-silencing of one or more "MSUD gene". Another hypothesis (model 2) is that natural populations harbor wide genetic variation in MSUD strength and the OR strains represent the MSUD-conducive extreme.

To test between the two models we constructed novel near-isogenic strains, B/S A and B/S a, from the Sad-type wild-isolated strains Bichpuri-1 a (B) and Spurger A (S), made new testers in them (*tester*^{B/S}), and examined MSUD in *tester*^{B/S} x B/S crosses. Care was taken to make the *tester*^{OR} x OR and *tester*^{B/S} x B/S crosses as completely analogous to each other as possible by inserting the same 2532 bp fragment (*r*^{ef}) from of the OR-derived *r*^t gene on chromosome 1 into precisely allelic chromosome 7 locations in the *tester*^{OR} and *tester*^{B/S} strains. Sequencing revealed that the B/S version of *r*^{ef} differs from the OR version by only 16 SNPs. MSUD-induced silencing of the *r*^t gene results in the production of round ascospores instead of wild-type spindle-shaped ones. The *tester*^{OR} x OR crosses produced > 90% round ascospores, whereas the *tester*^{B/S} x B/S crosses produced <50% round ascospores. Reassuringly, the *tester*^{OR} x *tester*^{B/S} crosses produced < 5% round ascospores, confirming that the ectopically located *r* gene sequence in the two testers were detected by the MSUD machinery as paired alleles, and hence did not trigger MSUD. These results supported model 2 and allowed rejection of model 1.

We introgressed the *tester*^{OR} transgene into strain 85 of the related species *N. tetrasperma*, and found MSUD was inefficient in *tester*^{Nt} x *N. tetrasperma* crosses. Together, our results suggested that efficient MSUD is not the norm in *Neurospora* and that the OR background represents the MSUD-conducive extreme in the

range of genetic variation in MSUD efficiency. We had also previously introgressed the *N. crassa* insertional translocations *T(EB4)* and *T(IBj5)* into *N. tetrasperma* and produced the new strains *T(EB4)^{Nt}* and *T(IBj5)^{Nt}*, in which nominally only the translocation breakpoints are from *N. crassa* but the rest of the genome is from *N. tetrasperma*. From the *T^{Nt} x N* crosses (*N* = normal sequence *N. tetrasperma*) we obtained *Dp(EB4)^{Nt}* and *Dp(IBj5)^{Nt}* progeny. As far as we are aware these were the first duplication strains made in *N. tetrasperma*.

Progress made in the current reporting year (April 1, 2017 - March 31, 2018)

1. Loci underlying the OR vs. B/S phenotypic difference.

We found that the “heterozygous” *tester^{OR}* x B/S and *tester^{B/S}* x OR crosses also showed inefficient MSUD (25-60% round ascospores), which suggested that the inefficient MSUD phenotype of B/S was dominant to the efficient MSUD phenotype of OR. Next, we crossed 102 f1 progeny from an OR *a* x B/S1 *A* cross with *tester^{OR}* of the opposite mating type and scored for MSUD. Crosses of 11 f1 progeny produced > 90% round ascospores, whereas the remaining 91 gave a spectrum in the 25-90% range. These results suggested that the efficient MSUD phenotype of the OR parent might require about three unlinked genes from the OR parent (11/102 is approx 1/8). In collaboration with Dr. K. T. Nishant (IISER-Tvm) we determined the Illumina genome sequence of the 11 OR-type f1s and found that they shared conserved chromosomes 1, 2, and 5 segments from OR. These segments might contain the loci that determine the OR-type phenotype. The loci might encode factors that provide additional regulatory cues to calibrate the MSUD response, and these factors are missing from OR. In future research we will increase the resolution of the mapping and try to identify the actual genes determining the OR vs. B/S difference.

2. Non-barren phenotype of *Dp*-heterozygous crosses in non-OR backgrounds.

In OR, crosses heterozygous for chromosome segment duplications (*Dp* x *N*) have for long been known to exhibit a severe MSUD-dependent barren phenotype. Since most *Neurospora* genetics studies over the past eight decades were done using the OR genetic background, the barrenness of *Dp*-heterozygous crosses was tacitly assumed to be generally true of all *Neurospora* backgrounds. Our laboratory

has previously shown that *Dps* can dominantly suppress the genome-defense process called repeat-induced point mutation (RIP), but the significance of such suppression was unclear given the generally assumed barrenness of *Dp*-heterozygous crosses. In the past year we for the first time examined the productivity of *Dp*-heterozygous crosses in *N. tetrasperma* which we showed has weak MSUD. *Dp(EB4)^{Nt}* *A* x 85 *a* and *Dp(IBj5)^{Nt}* *a* x 85 *A* represent *Dp*-heterozygous crosses made in the *N. tetrasperma* 85 background. Significantly, neither showed an obvious barren phenotype, and they were only quantitatively less productive than the corresponding control crosses *T(EB4)^{Nt}* *a* x 85 *A* and *T(IBj5)^{Nt}* *a* x 85 *A*. Specifically, *Dp(EB4)^{Nt}* *a* x 85 *a* and *T(EB4)^{Nt}* *a* x 85 *A* produced, respectively, 8.2×10^5 and 12.4×10^5 ascospores, and *Dp(IBj5)^{Nt}* *a* x 85 *A* and *T(IBj5)^{Nt}* *a* x 85 *A* produced 1.3×10^5 and 7.1×10^5 ascospores. These results suggest that the long accepted barrenness associated with *Neurospora* *Dp*-heterozygous crosses might in fact be limited to the OR-type backgrounds and that in non-OR populations *Dp*-mediated RIP-suppression might be more significant than has generally been appreciated. For example, it can explain the persistence of the *Tad* retrotransposon in the *N. crassa* Adiopodoumé strain while all other *Neurospora* strains examined contain only relics of *Tad* inactivated by the RIP mutational process. Possibly, crossover between non-allelic *Tad* copies in an ancestor of the Adiopodoumé strain created an insertional translocation that following a cross produced a *Dp* that suppressed RIP and sheltered *Tad* until its copy number increased sufficiently to render superfluous further *Dp*-mediated titration of the RIP machinery.

A manuscript describing these results was submitted to G3 Genes Genomes Genetics in May 2018, and we have received the reviews together with the invitation to submit a revised manuscript.

LNG Publications in 2017-18

(i) Primary research papers.

Nil

(ii) Other Publications.

1. Kasbekar, D. P. (2017). Ascus dysgenesis in hybrid crosses in *Neurospora* and *Sordaria* (Sordariaceae). *J. Genet.* 96: 457-463.
2. Kasbekar, D. P. (2017). Pushpa Mittra Bhargava (1928-2017). *Curr. Sci.* 113: 807-808.

3. Kasbekar, D. P. (2017). The science underlying use of DNA evidence to solve crime. Oct 26, LiveLaw.in (<http://www.livelaw.in/science-underlying-use-dna-evidence-solve-crime/>).
4. Kasbekar, D. P. (2018). Series. A cross-eyed geneticist's view 1. Making sense of the lamin B receptor, a chimeric protein. *J. Biosci.* 43: 235-237.

LABORATORY OF PLANT MICROBE INTERACTION

Understanding virulence mechanisms of *Xanthomonas* plant pathogens and interaction with host plants

Faculty	Subhadeep Chatterjee	Staff Scientist
PhD Students	Akanksha Kakkar	Senior Research Fellow
	Raj Kumar Verma	Senior Research Fellow
	Biswajit Samal	Senior Research Fellow
	Prashantee Singh	Senior Research Fellow
	Yasobanta Padhi	Senior Research Fellow
	Aditya Nigam	Junior Research Fellow (since Aug. 2017)
Postdoctoral fellow	Anindya Biswas	DBT-RA (since June 2017)
	Durga Bhavani	SERB-NPDF (since April 2018)
Other Members	Binod Bihari Pradhan	Technical officer
	Krishnamurty	Tradesman

Objectives

1. Identification and characterization of virulence factors of *Xanthomonas*
2. Role of cell-cell communication in *Xanthomonas* colonization and virulence
3. Function of protein secretion system in *Xanthomonas* and its role in virulence
4. Role of PAMP in pathogen recognition and plant defense response

Summary of work done until the beginning of this reporting year (April 1, 2016 – March 31, 2017)

Bacteria integrate extracellular cell-cell signalling or quorum sensing with intracellular signalling mediated by c-di-GMP to co-coordinately regulate diverse cellular processes. Although quorum sensing and c-di-GMP regulate diverse functions including motility, biofilm formation and production of virulence associated functions, their interplay and functional diversification of c-di-GMP turnover effectors in regulation of diverse functions remains undefined. In phytopathogen *Xanthomonas oryzae*, quorum sensing is mediated by diffusible signal factor (DSF), a fatty acid like signalling molecule which is involved in the regulation of several virulence associated functions including modulation of c-di-GMP effectors. However, it is still unclear how the c-di-GMP network regulates these traits. In an attempt to delineate the entire range of c-di-GMP functionality in *Xanthomonas oryzae* we constructed a deletion mutant library of 15 in-frame deletion mutants, targeting genes

predicted to be involved in c-di-GMP metabolism (biosynthesis or degradation) to understand the interplay between QS and complex c-di-GMP signalling network.

We have shown that a bacterial fatty acid cell-cell signaling molecule, DSF (diffusible signal factor) elicits innate immunity in plants. The DSF families of signaling molecules are highly conserved among many phytopathogenic bacteria belonging to genus *Xanthomonas* as well as in opportunistic animal pathogens. This is the first report of the role of bacterial cell-cell communication molecule in inducing host defense response and elucidates co evolution of host and bacterial communication signaling for adaptation in changing environmental condition.

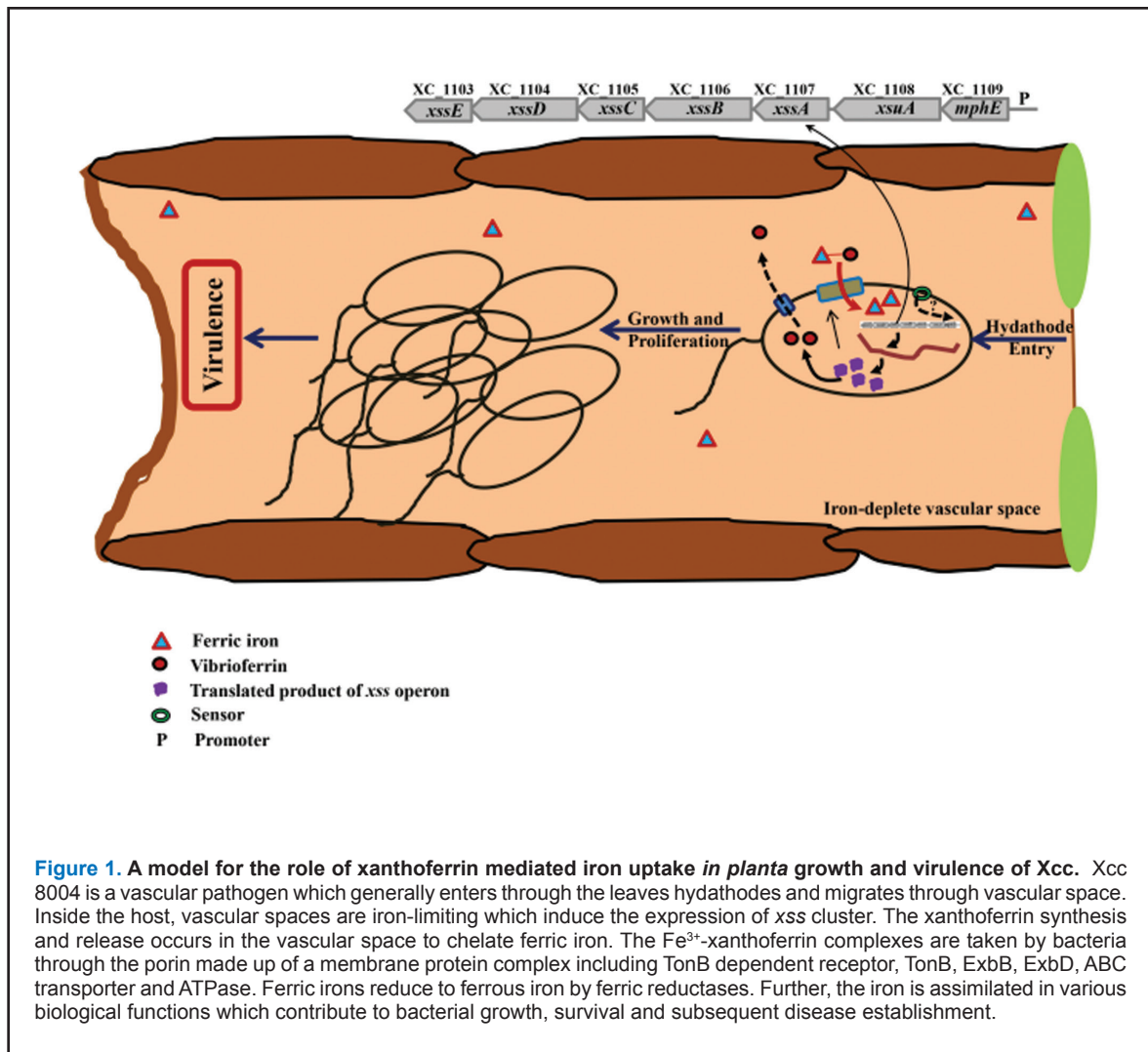
Details of the progress made in the current reporting year (April 1, 2016 – March 31, 2017)

Project 1: Role of iron in the virulence of *Xanthomonas* group of phytopathogen.

Xanthomonas campestris pv. *campestris* causes black rot, a serious disease of crucifers. *Xanthomonads* encodes a siderophore biosynthesis and uptake gene cluster *xss* (*Xanthomonas* siderophore synthesis) involved in production of a vibrioferrin type of siderophore. However, little is known about the role of siderophore in iron uptake and virulence of *X. campestris* pv. *campestris*. In this study, we show that *X. campestris* pv. *campestris* produces an α -hydroxy carboxylate type of siderophore (named xanthoferrin), which is required for growth under low-iron condition and

optimum virulence. A mutation in the siderophore synthesis *xssA* gene causes deficiency in siderophore production and growth under low-iron conditions. In contrast, the siderophore utilization $\Delta xsuA$ mutant was able to produce siderophore but exhibited a defect to utilize siderophore-iron complex. Our radiolabelled iron uptake studies confirmed that the $\Delta xssA$ and $\Delta xsuA$ mutants exhibited defects in ferric iron uptake. The $\Delta xssA$ mutant was able to utilize and transport exogenous xanthoferrin- Fe^{3+} complex, in contrast, the siderophore utilization or uptake mutant $\Delta xsuA$ exhibited defects in siderophore uptake. Expression analysis of *xss* operon using a chromosomal *gusA* fusion indicates that the *xss* operon is expressed during *in planta* growth and under low-iron conditions. Furthermore, exogenous iron supplementation in the cabbage leaves rescued the *in planta* growth deficiency of $\Delta xssA$ and $\Delta xsuA$ mutants. Our study reveals

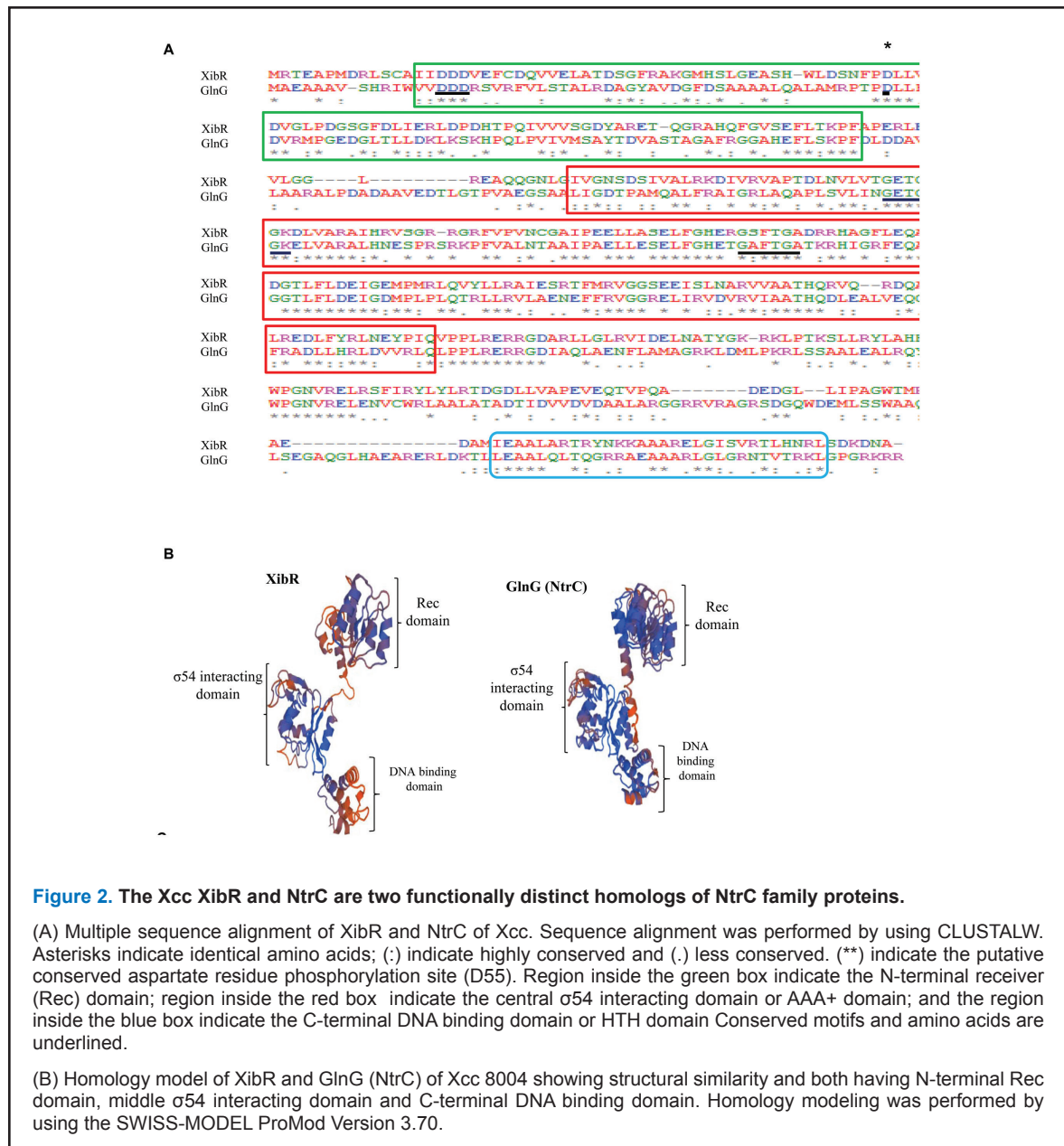
that the siderophore xanthoferrin is an important virulence factor of *X. campestris* pv. *campestris* which promote *in planta* growth by sequestering ferric iron. On the basis of our study, we have proposed a model which elucidate the role of xanthoferrin mediated iron uptake in establishing pathogenesis of Xcc under low-iron environment inside host (Figure 1). Xcc encounters iron depleted environment inside the host, which triggers the expression of xanthoferrin synthesis and uptake genes. Xanthoferrin is then released outside the bacterial cell where it starts scavenging ferric iron and eventually gets transported inside as xanthoferrin- Fe^{3+} complex through TonB dependent transporters and its auxiliary proteins ExbB and ExbD. Subsequent ferric reduction occurs inside the bacterial cell to convert Fe^{3+} to easily utilizable Fe^{2+} form, which is used by bacteria for various metabolic activities during growth and infection.



Project 2: Co-regulation of iron metabolism and virulence associated functions by iron and XibR, a novel iron binding transcription factor, in the plant pathogen *Xanthomonas*

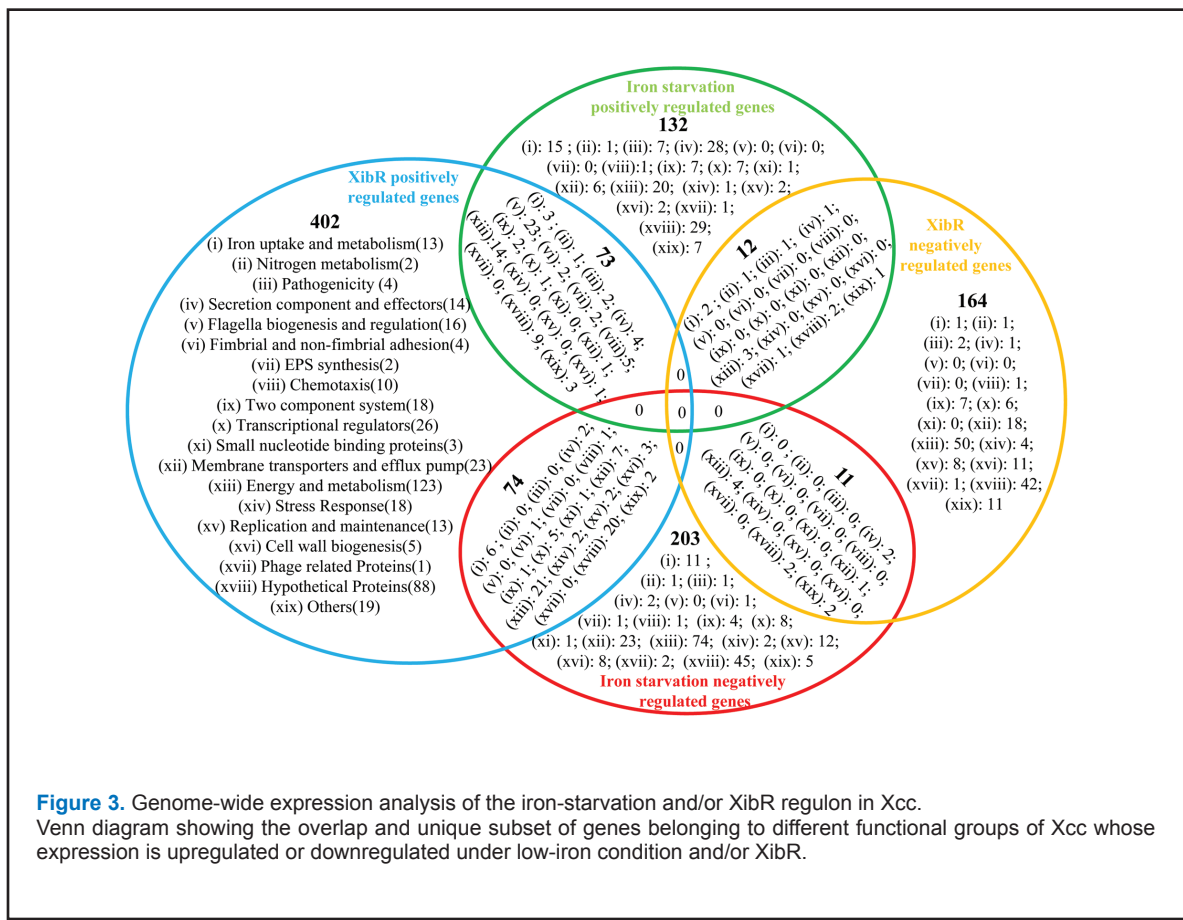
Abilities of bacterial pathogens to adapt to the iron limitation present in hosts is critical to their virulence. Bacterial pathogens have evolved diverse strategies to coordinately regulate iron metabolism and virulence associated functions to maintain iron homeostasis in response to changing iron availability in the environment. In many bacteria the ferric uptake regulator (Fur) functions as transcription factor that utilize

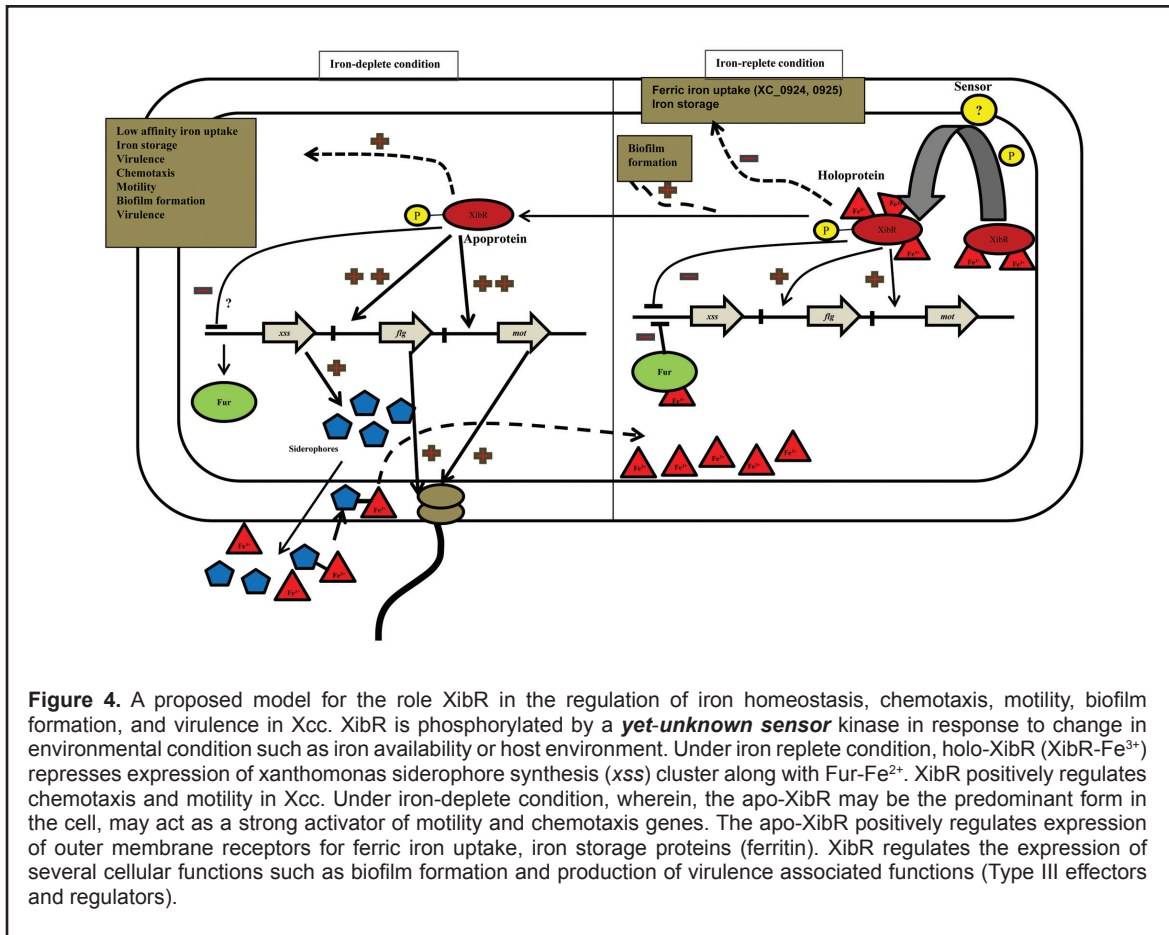
ferrous form of iron as cofactor to regulate transcription of iron metabolism and many cellular functions. However, mechanisms of fine-tuning and coordinated regulation of virulence associated function beyond iron and Fur-Fe²⁺ remain undefined. In this study, we show that a novel transcriptional regulator XibR (named *x*anthomonas *i*ron *b*inding *r*egulator) of the NtrC family, is required for fine-tuning and co-coordinately regulating the expression of several iron regulated genes and virulence associated functions in phytopathogen *Xanthomonas campestris* pv. *campestris* (Xcc) (Figure 2). Genome wide expression analysis



of iron-starvation stimulon and XibR regulon, GUS assays, genetic and functional studies of *xibR* mutant revealed that XibR positively regulates functions involved in iron storage and uptake, chemotaxis, motility and negatively regulates siderophore production, in response to iron (Figure 3). Furthermore, chromatin immunoprecipitation followed by quantitative real-time PCR indicated that iron promoted binding of the XibR to the upstream regulatory sequence of operon's involved in chemotaxis and motility. Circular dicorism spectroscopy showed that purified XibR bound ferric form

of iron. Electrophoretic mobility shift assay revealed that iron positively affected the binding of XibR to the upstream regulatory sequences of the target virulence genes, an effect that was reversed by ferric iron chelator deferoximine. Taken together, these data revealed that how XibR coordinately regulates virulence associated and iron metabolism functions in Xanthomonads in response to iron availability. Our results provide insight of the complex regulatory mechanism of fine-tuning of virulence associated functions with iron availability in this important group of phytopathogen (Figure 4).





Publications:

(i) Research papers published in the calendar year 2017:

1. Pandey SS, Patnana PK, Rai S, and Chatterjee S. (2017). Xanthoferrin, the α -hydroxy carboxylate type siderophore of *Xanthomonas campestris* pv. *campestris* is required for optimum virulence and growth inside cabbage. **Molecular Plant Pathology**. 18:949-962.
2. Pandey SS., Singh, P., Samal B, Verma RK, and Chatterjee S. (2017). Xanthoferrin Siderophore Estimation from the Cell-free Culture Supernatant of Different *Xanthomonas* Strains by HPLC. **Bio-protocol**. 17: DOI:10.21769/BioProtoc.2410.

(ii) Research papers in press as on 31st March 2018

1. Javvadi, S, Pandey SS, Mishra, A, Pradhan BB, and Chatterjee S. Bacterial cyclic β -(1,2)-glucans sequester iron to protect against iron-induced toxicity. **EMBO Reports**.
2. Pandey S.S, Patnana P.K, Padhi Y, and Chatterjee S. (2018). Low-iron conditions induces the hypersensitive reaction and pathogenicity *hrp* genes expression in *Xanthomonas* and is involved in modulation of hypersensitive response and virulence. **Environmental Microbiology and Environmental microbiology Reports**.

LABORATORY OF STRUCTURAL BIOLOGY

Structural aspects of protein synthesis in pathogenic organisms

Faculty	Prem S. Kaushal	Staff Scientist (since May 2017)
Ph D Students	Punam Bala	Junior Research Fellow (since Feb. 2018)
Other Members	Sheeba A. N. Sudheer	Technical Officer Skilled Work Assistant
Collaborators	Dr. Saman Habib	CDRI, Lucknow

Objectives

Our laboratory's research goal is to unravel the structural basis of the functioning of large macromolecular complexes, and thereby, to identify potential drug targets. We are applying a multidisciplinary approach of molecular biology, biochemistry, cryo- electron microscopy (cryo-EM), X-ray crystallography and bioinformatics. Following are the ongoing or proposed research projects

1. Protein synthesis in pathogenic organisms

(a) Protein synthesis in *Plasmodium falciparum*

(b) Protein synthesis and dormancy in *Mycobacterium tuberculosis*

2. Ribosome biogenesis

3. Methods for cryo- EM

Summary of work done until the beginning of this reporting year (upto March 31, 2017)

Lab activities started after 31st March 2017.

1. Protein synthesis in pathogenic organisms

Protein synthesis, the translation of the genetic code into the amino acid, have been an attractive drug target, nearly 40% of antibiotics targets different steps of protein synthesis in bacteria. However, protein synthesis in pathogenic organisms remains poorly understood. We are focusing on the structural aspects of protein synthesis mainly in; (a) the *Plasmodium falciparum* an intracellular obligate human parasite that causes the most lethal form of malaria, (b) the *Mycobacterium tuberculosis* (MTb) that causes deadly infectious disease, the tuberculosis. The aims of our studies are to find out new drug targets, improve the efficacy of presently used drugs and identify new inhibitors.

(a) Protein synthesis in *Plasmodium falciparum*

Every year, malaria kills nearly half a million peoples worldwide, with the majority of victims being children under 5 years of age. Owing to the emergence of parasite resistance to front-line drugs, there is an urgent need to find new antimalarial drug targets. The *P. falciparum* has three sites of protein synthesis the cytoplasmic ribosome and two organellar ribosomes, reside inside the mitochondria and another inside, a non-photosynthetic relict plastid, the apicoplast. The mitochondria and the apicoplast are believed to have be evolved through an endosymbiotic event. Therefore, mitochondrial ribosome (mitoribosome) and apicoplast ribosome (apicoribosome) are of prokaryotic origin which makes them an attractive target for antimalarial drugs. Several antibiotics, such as Tetracycline, Doxycycline, and Clindamycin etc, are currently used to treat malaria along with mainstream antimalarial drugs show delayed-death drug response, where the blood-stage parasite dies only in the cycle next to the one in which it has been exposed to the drug. The use of antibiotics as an antimalarial drug is known to trigger resistance in pathogenic bacteria. The organellar ribosome possesses several unique features. The mitoribosome would have ~15 r-proteins in SSU, ~26 r-proteins in LSU as compared to the bacterial ribosome (21 in SSU and 34 in LSU) and fragmented rRNA. One of the most distinct features of mitoribosome is that its rRNAs are highly fragmented with 23 to 190 nucleotides long and this is the highest fragmented ribosome known so far. What is the exact composition of mitoribosome and how the fragmented rRNA provides a structural scaffold in mitoribosome remains unknown? Similarly, the apicoplast translational machinery also possesses several unique features the apicoribosome contains a

lesser number of ribosomal proteins (16 in the small ribosomal subunit [SSU], and 21 in the large ribosomal subunit [LSU]), as compared to the bacterial ribosome (21 in SSU and 34 in LSU) [14].

Details of progress made in the current reporting year (April 1, 2017 - March 31, 2018)

We have initiated the sequence analysis and homology to glean insights in to the structural aspects of protein synthesis in organellar ribosomes. A homology modelling, performed using programs Phyre2 and I-TASSER, showed the apicoplast and mitochondrial translation factors possesses signal sequences, insertions and terminus extensions ranges from 8 to 120 amino acid residues long (Figure 1). Homology

modelling of apicoplast ribosomal proteins were also performed. The highest resolution (2.1 Å) structure of *E. coli* 70S ribosome, PDB ID; 4YBB was used a templet. The homology modeling showed some of the conserved protein in apicoribosome also possesses insertions and extensions (Figure 2). The homology modelling for mitoribosome is in progress. We have also initiated structural work. We have started the purification of RRF_{api} and EF-G_{api}. Both the proteins are not showing overexpress in BL21 DH3 and Rosetta cells. Further, we are checking overexpression in codon optimized, BL21 RIL cells. We are also planning to express in Pichia expression system. The clones for these proteins were kindly provided by Dr. Saman Habib, Scientist, CDRI, Lucknow.

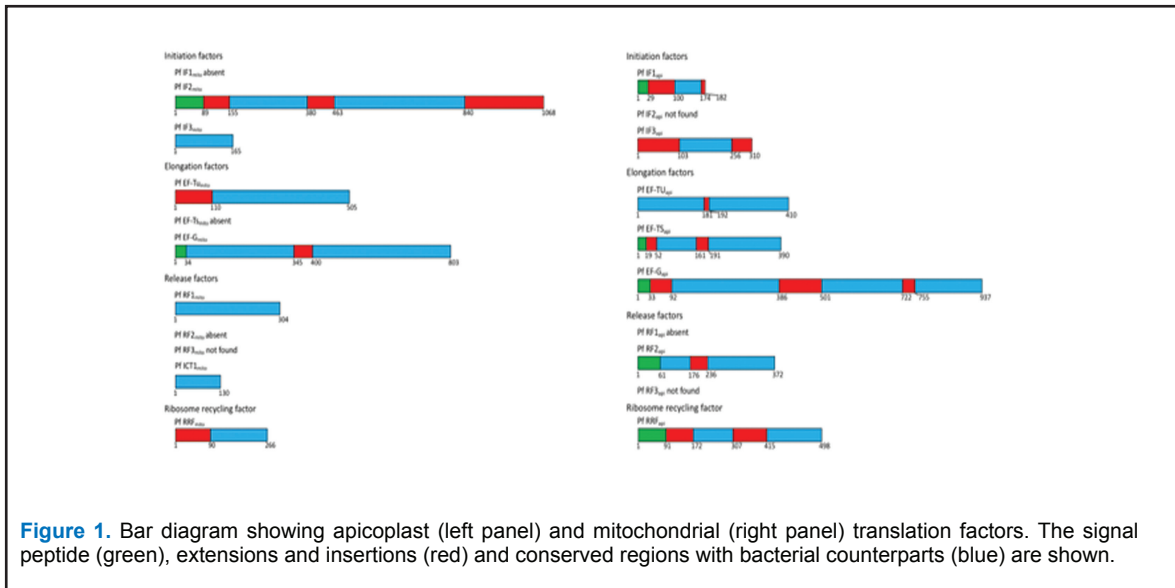


Figure 1. Bar diagram showing apicoplast (left panel) and mitochondrial (right panel) translation factors. The signal peptide (green), extensions (red) and conserved regions with bacterial counterparts (blue) are shown.

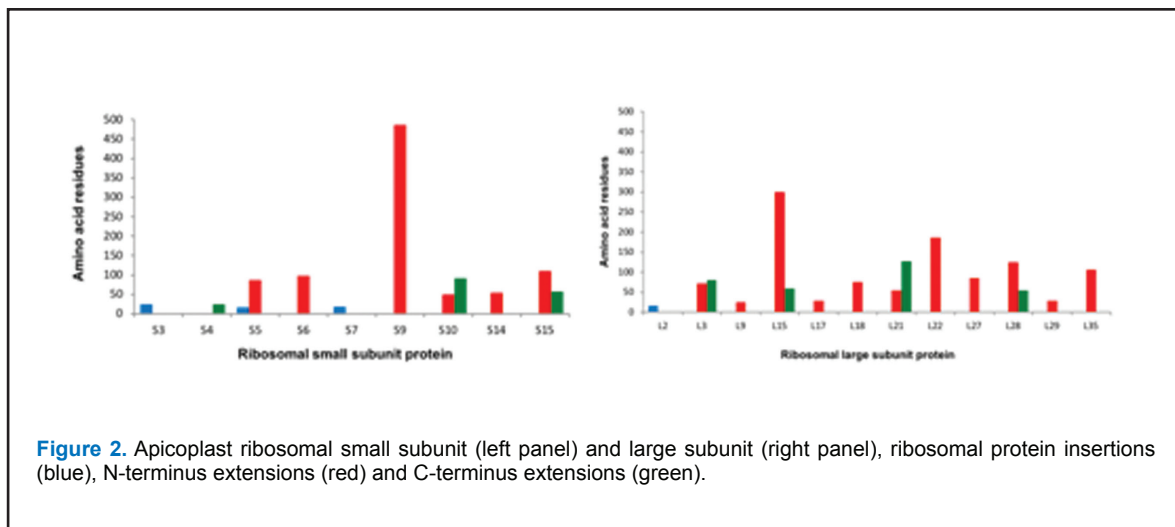
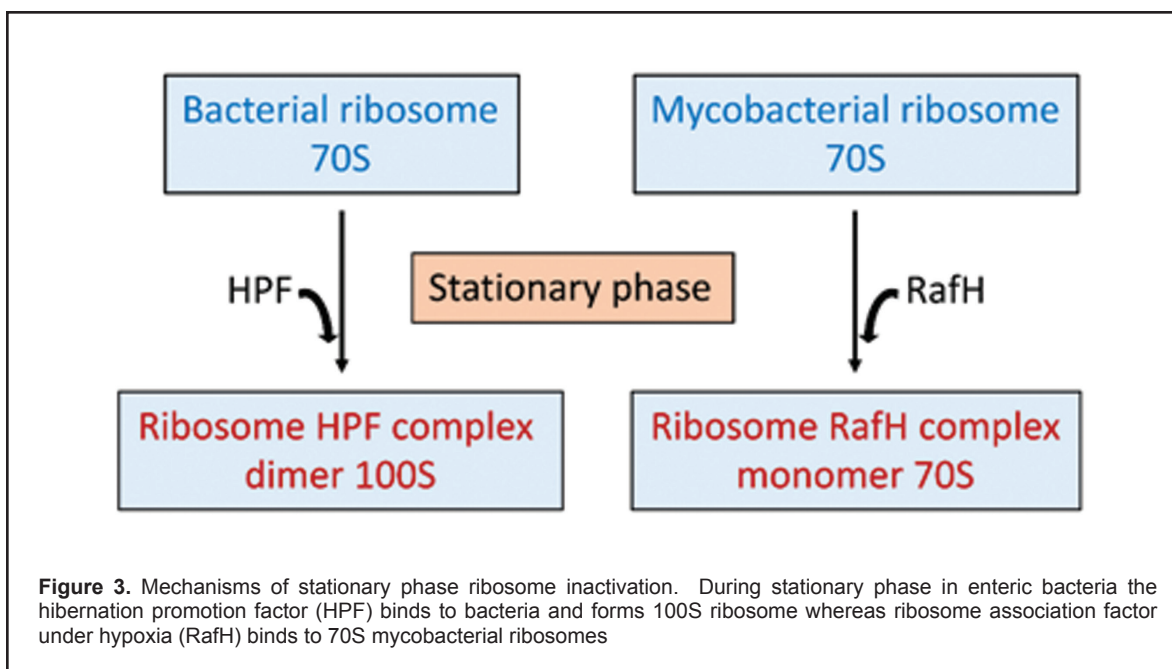


Figure 2. Apicoplast ribosomal small subunit (left panel) and large subunit (right panel), ribosomal protein insertions (blue), N-terminus extensions (red) and C-terminus extensions (green).

(b) Protein synthesis and dormancy in *Mycobacterium tuberculosis*

Tuberculosis (Tb) remains a major health threat to the human race. MTb has emerged with multi-drug resistant (MDR-Tb) and extensive-drug resistant (XDR-Tb) strains towards currently used drugs. Situation in India is more alarming by recent emergence of a total drug-resistant Tb strain. Hence, a better understanding of pathogen's life cycle is vital to facilitate the finding of new targets for rational drug design. MTb possesses a unique mechanism to establish a latent tuberculosis infection (LTBI), the dormancy state, capable of its long persistence in the host, even in the presence of functional host immune response. An estimated one third of the world's population has LTBI and eradication of tubercle bacilli in latent lesion by current drugs has proved to be inefficient. The molecular mechanisms of Mtb entry into dormancy and its maintenance of dormancy state remain vaguely known. The

rate of protein synthesis is slower down during dormancy. The dormancy survival regulon (DosR regulon) encodes 48 genes that appear to play crucial roles in dormancy. The ribosome-associated-factor under-hypoxia (RafH) which is encoded by *MSMEG_3935* gene is regulated by DosR regulon. Translation inhibition by RafH is a unique feature associated with Mtb ribosomes, it binds to 70S ribosome which appears to be dramatically different from that occur in enteric bacteria, where ribosomes form 100S dimers in static conditions (Figure 3). Our goal is to characterize all 48 genes expressed in LTBI and solve the cryo-EM structure of MTb ribosomes isolated from mycobacteria grown in different stress conditions such as hypoxia, low nutrient content, oxidative stress. Further, explore the unique features of ribosome-factor (express in stress) interactions, to find new drug targets and design an inhibitor against the dormant pathogen.



2. Ribosome Biogenesis

Ribosomes are ribonucleoprotein complexes of mega Dalton (mDa) sizes and are responsible for protein synthesis in the all cells. Each ribosome is composed of two subunits, large subunit (LSU) and small subunit (SSU). In yeast, 60S LSU is composed of 25S, 5.8S and 5S rRNAs and 46 r-proteins. whereas, 40S SSU is composed of 18S rRNA and 33 r-proteins.

Ribosome assembly is a complicated and highly regulated process. In yeast, assembly begins with the transcription of ribosomal RNA in the nucleus and binding of ribosomal proteins. Nearly 200 assembly factors and 76 small nucleolar RNAs are transiently associated with for proper assembly of ribosomes. In near future, we would like to illustrate the molecular mechanism of these factors in ribosome assembly.

3. Methods for cryo- EM

One of the major bottleneck to achieve high resolution in cryo- EM is to get evenly distributions of particle with optimum ice thickness on the cryo-

EM grid. We are planning to screen different compound and grids made up of different metals, to see the effect on particle distribution and ice thickness.

LABORATORY OF TRANSCRIPTION

Mechanism of transcription termination and antitermination in *Escherichia coli*.

Faculty	Ranjan Sen	Staff Scientist
Ph.D. Students	Gairika Ghosh	Senior Research Fellow
	Md. Hafeezunnisha	Senior Research Fellow
	Passong Immanual	Junior Research Fellow
	Ajay Khatri	Junior Research Fellow
	Saddam Ansari	Junior Research Fellow (since Feb. 2018)
Other Members	Shweta Singh	DST SERB Young Scientist
	Shreyans Jain	Postdoctoral fellow
	Sushmit Shambhare	Postdoctoral Fellow (till April 2017)
	Amit Kumar	Postdoctoral Fellow
	T Pragna Lakshmi	Postdoctoral Fellow
	KVS Rammohan	Project Assistant
	Sapna Godavarthi	Technical Officer
	Krishna	Lab Attendant
Collaborator	Dr. Jayanta Mukhopadhyay	Bose Institute, Kolkata
	Prof. Akira Ishihama	Hosei University, Japan.

Objectives.

Fundamental questions in the area of mechanism of transcription termination and antitermination processes in bacteria is still not very clear, and we are interested to understand these questions. In my laboratory, following studies are underway. 1) Mechanism of action of transcription termination factor, Rho both *in vivo* and *in vitro*. 2) Molecular basis of Rho-NusG interaction. 3) Designing peptide inhibitors of Rho from the bacteriophage protein, Psu. 4) Involvements of Rho in different physiological processes. 5) Isolating myco-bacteriocidal factors from the mycobacteriophages using metagenomics approaches.

Summary of the work done until the beginning of this reporting year (April 1, 2016- March 31, 2017).

We explored the anti-Rho activity of the bacteriophage protein, Psu, for the Rho proteins from different pathogens. Sequence alignment and homology modelling of Rho proteins from pathogenic bacteria revealed the conserved nature of the Psu-interacting regions in all these proteins. We chose Rho proteins from various pathogens like, *Mycobacterium smegmatis*, *Mycobacterium bovis*, *Mycobacterium tuberculosis*, *Xanthomonas campestris*, *Xanthomonas oryzae*, *Corynebacterium*

glutamicum, *Vibrio cholerae*, *Salmonella enterica* and *Pseudomonas syringae*. The purified recombinant Rho proteins of these organisms showed variable rates of ATP hydrolysis on the poly rC as substrate and were capable of releasing RNA from the *E. coli* transcription elongation complexes. Psu was capable of inhibiting these two functions of all these Rho proteins. *In vivo* pull down assays revealed direct binding of Psu with many of these Rho proteins. *In vivo* expression of Psu induced killing of *M. smegmatis*, *M. bovis*, *X.campestris*, and *S.enterica* indicating Psu-induced inhibition of Rho proteins of these strains under physiological conditions. We propose that the “universal” inhibitory function of the Psu protein against the Rho proteins from both the gram-negative and gram-positive bacteria could be useful for designing peptides having antimicrobial functions, and these peptides could be a part of synergistic antibiotic treatment of the pathogens through compromising the Rho functions (*J. Bact.*, 2018).

In bacteria, the transcription-coupled DNA repair (TCR) initiates after the Mfd protein removes the RNA polymerases (RNAP) stalled at the DNA lesions. The RNA helicase, Rho, is a transcription termination protein of bacteria that releases the elongation complexes. We hypothesized that Rho might dislodge the stalled RNAPs at the DNA lesions and initiate the TCR. We show that

Rho mutants are synthetically lethal with the TCR as well as the base excision repair (BER) genes, strains carrying these mutants were susceptible to the TCR-inducing agents and were inefficient in repairing UV-damaged DNA *in vivo*. In the *in vitro* transcription reactions, Rho functioned like Mfd and the anti-backtracking agent, GreB, facilitated this process. We concluded that the Rho-dependent termination is a component of the TCR and the Rho competes with or augments the Mfd function or initiate TCR in the absence of the latter, thereby moonlights as a DNA repair protein (*Nature Comm, under revision, 2018*).

Details of the progress in the current reporting year (April 1, 2017- March 31, 2018).

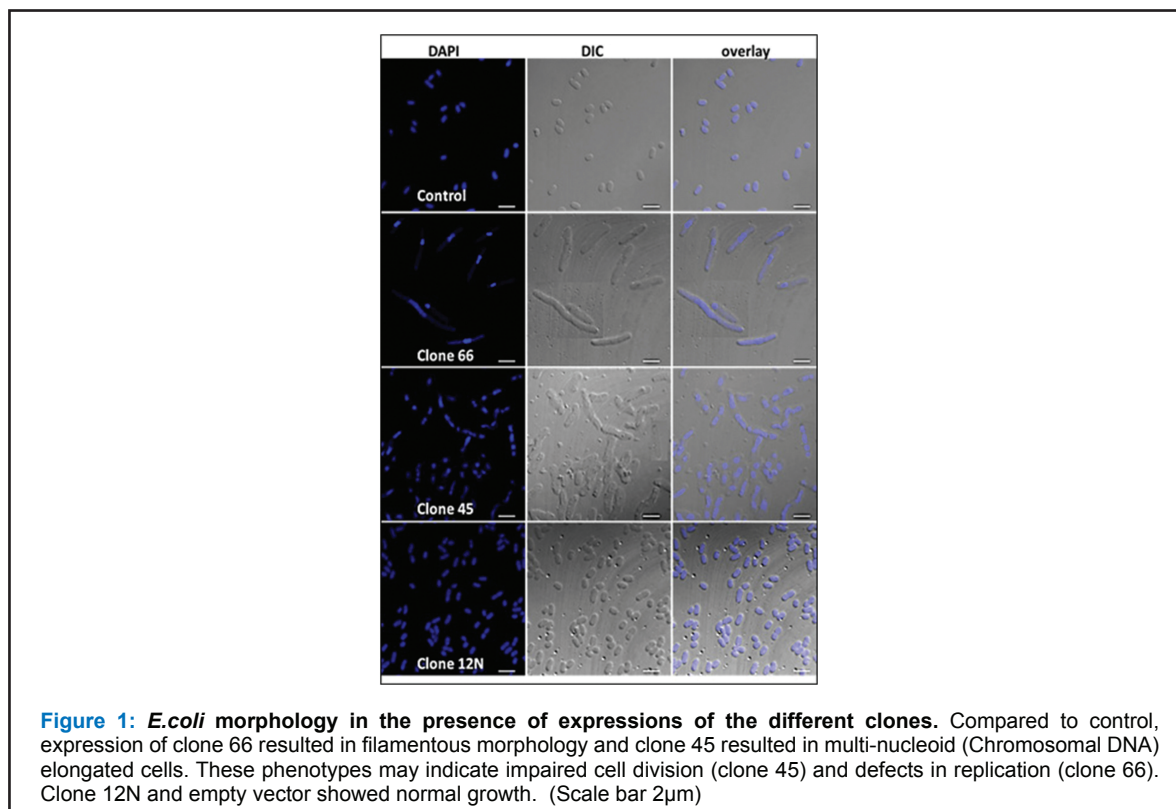
A) Exploring mycobacteriophage genomes to identify novel mycobactericidal agents

Bacteriophages are the viruses that invade bacterial cells. They code numerous protein factors capable of modulating host machineries for their own growth advantages. Mycobacteriophages infect mycobacteria, including *M. smegmatis* and *M. tuberculosis* etc. So far, about 1500 mycobacteriophages were sequenced (<http://phagesdb.org>). Functions of majority of the gene products of these phages are not known. The comparative genomic analysis of the mycobacteriophages reveals their highly

diverse nature, and the presence of unique proteins coding genes. Here we investigate mycobacteriophage derived molecules that impair the growth of the mycobacterial host.

We prepared a random library of mycobacteriophage genome fragments from 7 species and screened for the genome fragments capable of killing *M. Smegmatis*. The expressions from the protein factors from the clone 45 (from Che12), clone 66 (from Che12), clone 85 (from D29) and 12N (from D29) killed *M. Smegmatis* as well as *M. Bovis*. Many of these clones induced elongation defects and bulges in the cells. Among these clones, clone 45 and 66 induced lethality to *E.coli MC1061* strain. DAPI staining showed that the *E. coli* cells overexpressing the clone 45 were often multi-nucleoid (Chromosomal DNA) and elongated as compare to the control cells (Figure 1), whereas the expression of clone 66 resulted in filamentous morphology with single nucleoid (Figure 1). These phenotypes indicate impaired cell division (by clone 45 expression) and defect in replication process (by clone 66 expression).

Protein sequencing confirmed that the clone 45 expressed the gp34 protein and the clone 66 expressed gp49 of phage Che12. Attempts are being made to purify these proteins.



B) Rho dependent transcription termination regulates antibiotic sensitivity in *E. coli*.

We have discovered that upon compromising the Rho-dependent transcription termination function, the *E. coli* MG1655 strains exhibit a broad-spectrum antibiotic sensitivity. This indicates that the antibiotic efflux system was malfunctioned under this condition. Consistent with the aforementioned hypothesis, we found that the efflux of Ethidium Bromide, a dye effluxed by TolC-efflux pump, was significantly affected in the strains expressing Rho mutants, and the defect was comparable with what is usually observed in the strains having deletions in the efflux-pump genes, *acrA*, *acrB* and *tolC*. We also show that the MG1655 *DtoC* expressing Rho mutants fail to withstand a combination of antibiotic pressure even in the presence of antibiotic resistant genes. However, it is to be noted that this defect in efflux pump system was not due to the down-regulation of the efflux genes as was evident from the RT-qPCR. Since efflux systems pump out other toxic metabolites, bile salts, toxins like hemolysin and colicins, a defect in this pumping system

should accumulate these metabolites. A primary metabolite analysis showed higher accumulation of metabolites in the Rho mutants. Rho mutants were also capable of utilizing unusual di-peptides through the expression of *dpp* operon. These altered physiological conditions in the Rho mutants could cause high metabolic load in the cell, which in turn could “overwhelm” the limited number of TolC pumps and make them to function less efficiently. Finally, the Rho mutants showed increased expression of *pga* operon in the log phase resulting into the increased level of N-acetyl glucosamine, which caused the unusually high stickiness of the Rho mutants on the polystyrene culture plates. It is likely that the cells with compromised Rho function could have altered poly-glucosamine structure on the cell surface. We concluded that the high level of metabolite as well as altered outer-membrane structure could be the reasons for the less efficient efflux system in the Rho mutants giving rise to high antibiotic sensitivity. We propose that the antibiotic treatment regime, especially for the multi-drug resistance pathogens, should include ways to suppress Rho functions also (Figure 2).

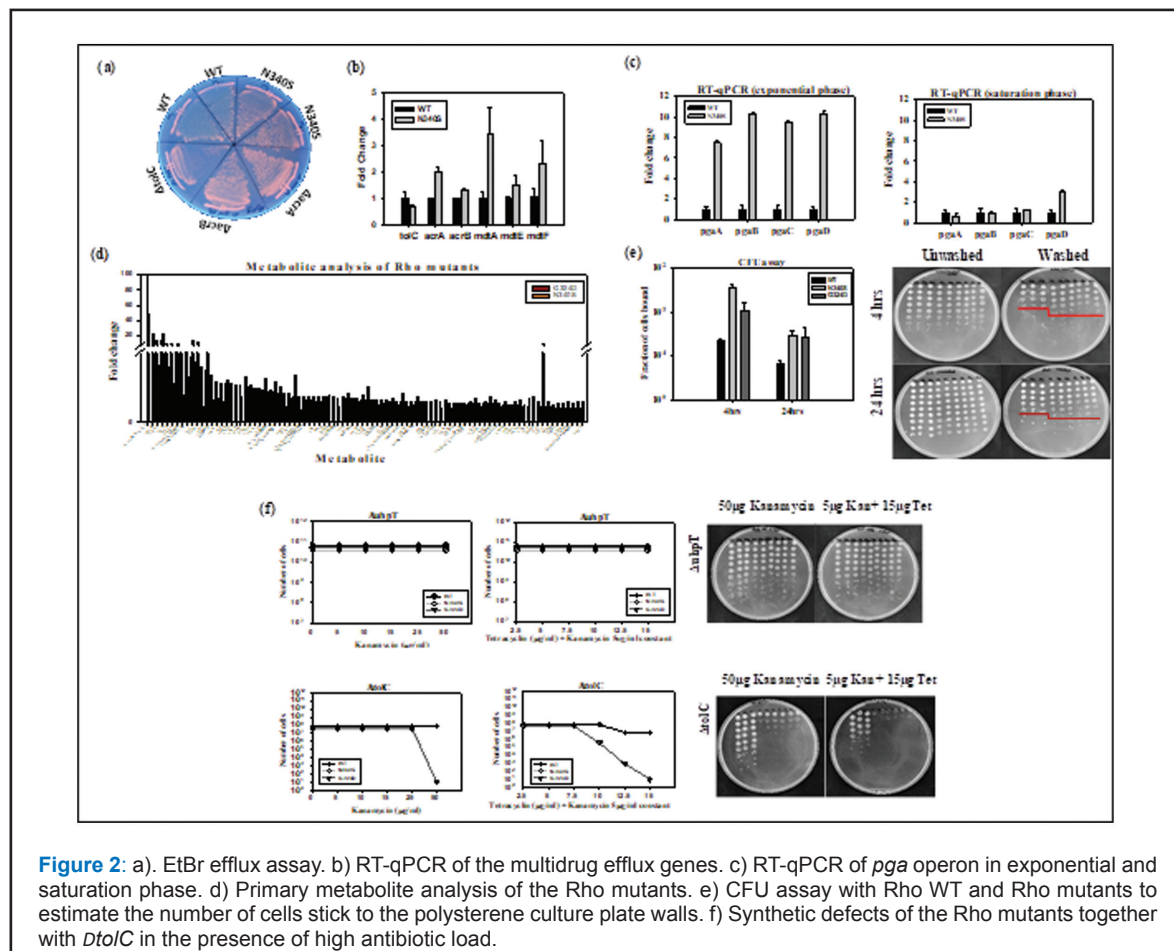
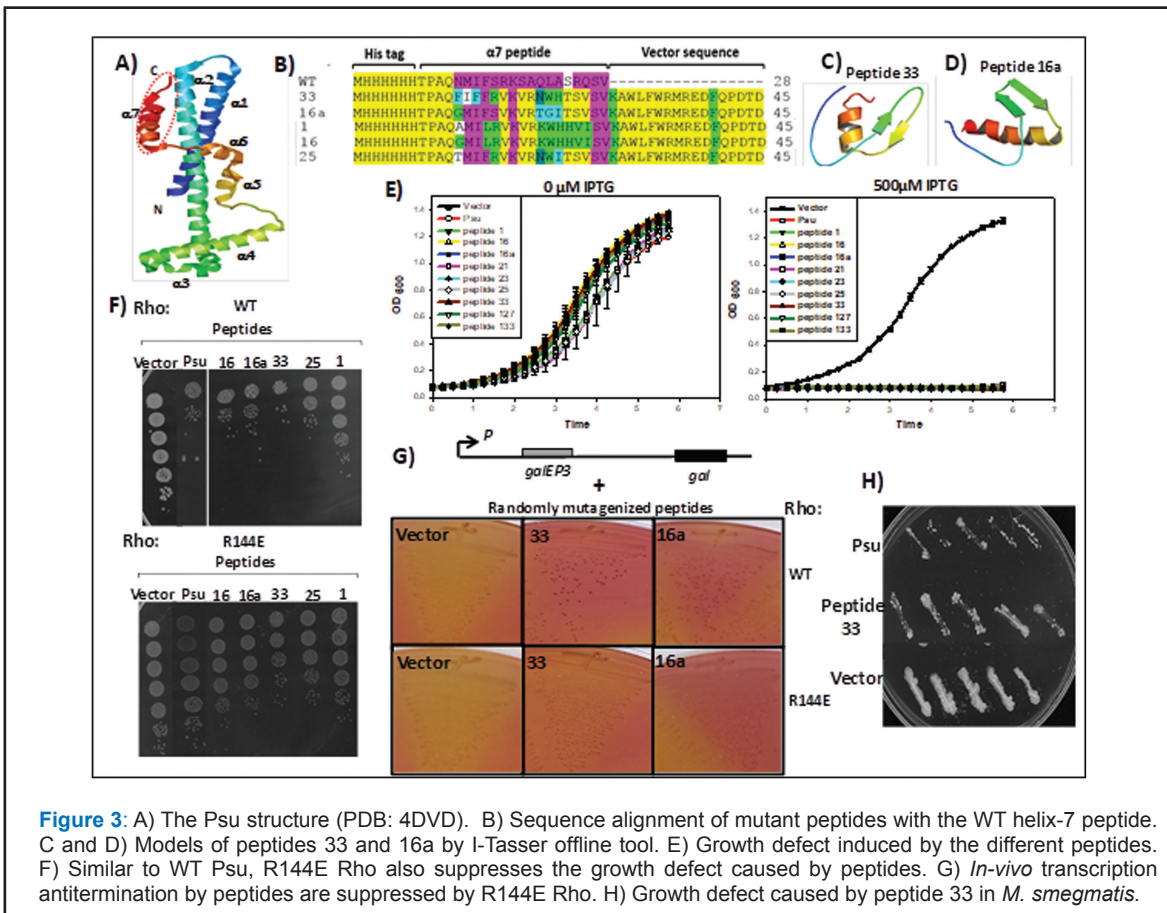


Figure 2: a). EtBr efflux assay. b) RT-qPCR of the multidrug efflux genes. c) RT-qPCR of *pga* operon in exponential and saturation phase. d) Primary metabolite analysis of the Rho mutants. e) CFU assay with Rho WT and Rho mutants to estimate the number of cells stick to the polyesterene culture plate walls. f) Synthetic defects of the Rho mutants together with *DtoC* in the presence of high antibiotic load.

C) Design of antimicrobial peptides from the bacteriophage *Psu* protein, an antagonist of transcription terminator, Rho.

Rho-dependent transcription termination in bacteria is an essential and crucial step in the regulation of gene expression. The unique multifunctional physiological properties of Rho make it a widespread drug target. *Psu* is a distinctive bacteriophage P4 capsid protein that moonlights as an inhibitor of *E. coli* Rho by antagonizing its ATPase and translocase activity. Here, we report the generation of peptide inhibitors from the functional domain of *Psu*. The *Psu* c-terminal helices 6 and 7 comprise the region that are instrumental in direct interactions with Rho. A library of peptides is prepared by randomly mutagenizing the sequence of the helix-7 and they are cloned in an expression

vector. Gain of function mutant peptides capable of performing anti-termination through a Rho-dependent terminator in a similar way as the WT *Psu* protein were selected by genetic screening. These peptides when overexpressed in *E. coli* showed severe growth defect. The gain of function peptides not only had the mutagenized peptide sequence from the helix-7 of *Psu* but also their C-terminal region extended into the vector sequence resulting into 45 mer peptides. The antitermination and the lethal effects of the peptides were suppressed by a specific Rho mutant, R144E, defective for the *Psu*-binding, strongly indicated that these lethal effects were due to the specific binding to the Rho protein. Hence, these set of novel antimicrobial peptides functioning against Rho can be further developed for antimicrobial treatment (Figure 3).



D) Identification of NusG dependent terminators

NusG, a transcription elongation factor, is capable of binding to RNAP and Rho. NusG has been observed to facilitate Rho-dependent termination both *in vivo* and *in vitro*. We have

earlier shown that NusG increases the rate of isomerization of open to close complex formation by Rho at the Rho-loading sites or terminators. NusG exerts its influence on a subset of Rho-dependent terminators. A characterization of such sub category of terminators is essential.

Four strains expressing NusG mutants, G146D, L158Q, V160N and I164A that are defective for Rho binding were used to perform a microarray study. Microarray analyses shown that about one third of the Rho-dependent terminators are NusG dependent (Figure 4A). We tested many of these predicted terminators and validated them by qRT-PCR (Figure 4B). Among the 15 genes that showed up-regulation belong to 14 different regulatory systems. From the 14 operon

checked, 6 possible terminators were narrowed down to *gfcC*, *ytfI*, *ycfJ*, *sugE*, *yagL* and *yfjJ*. The software mFold was used to identify the possible terminator sequences. Two predicted terminators were cloned upstream of a reporter cassette *lacZ* to assess the *in vivo* functions and it was observed that the terminators were dependent on NusG *in vivo* (Figures 4C and 4D). Further *in vitro* and *in vivo* characterizations are in progress.

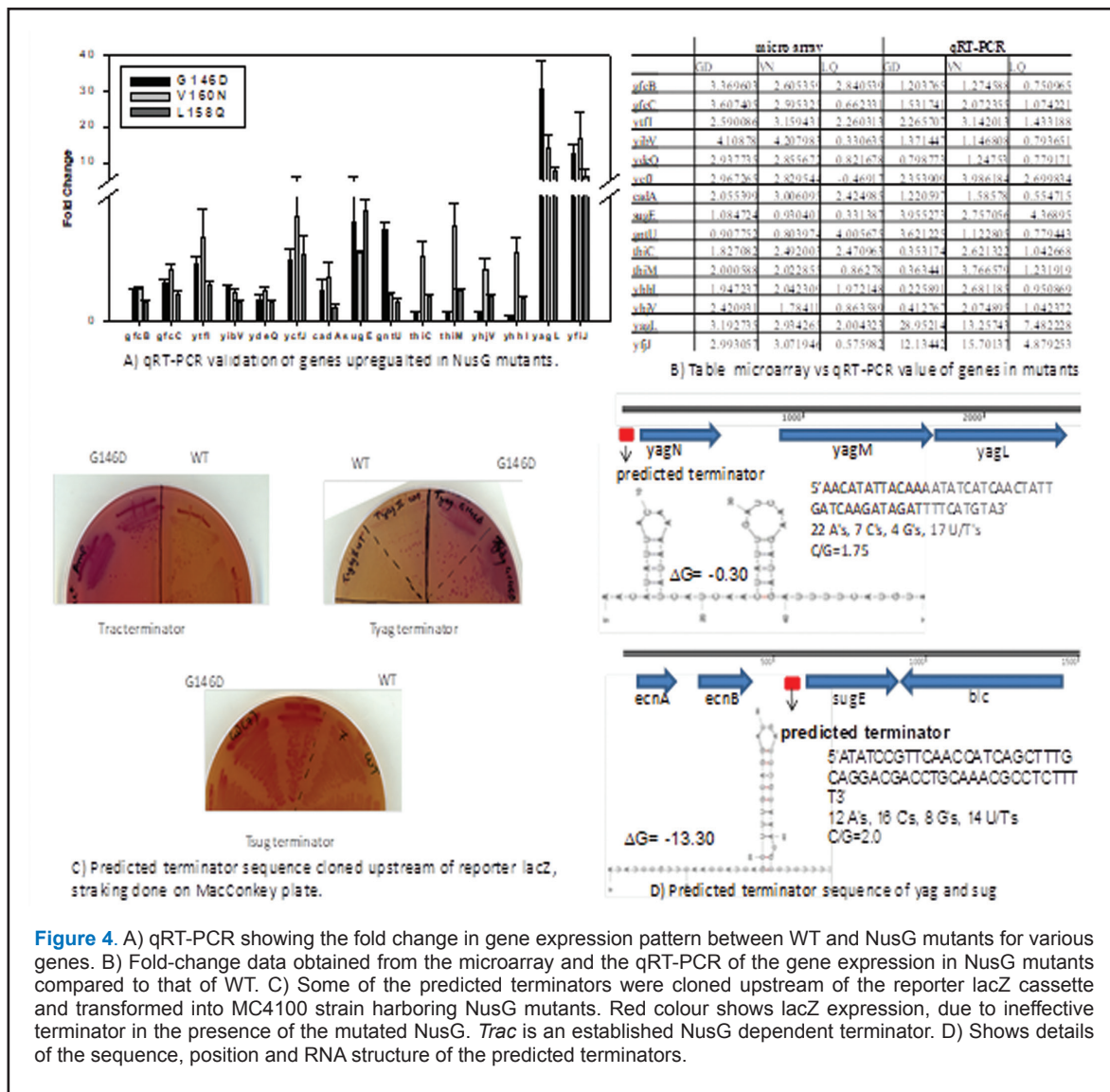


Figure 4. A) qRT-PCR showing the fold change in gene expression pattern between WT and NusG mutants for various genes. B) Fold-change data obtained from the microarray and the qRT-PCR of the gene expression in NusG mutants compared to that of WT. C) Some of the predicted terminators of the reporter *lacZ* cassette were cloned upstream of a reporter cassette and transformed into MC4100 strain harboring NusG mutants. Red colour shows *lacZ* expression, due to ineffective terminator in the presence of the mutated NusG. *Trac* is an established NusG dependent terminator. D) Shows details of the sequence, position and RNA structure of the predicted terminators.

Future Plans/directions:

The following projects, being pursued in my lab, are in different stages of completion. 1) Involvement of Rho in controlling the antibiotic sensitivity and the toxin-anti-toxin system, ii) design of peptide-inhibitors from

Psu, iii) characterization of different myco-bacteriocidal factors from mycobacteriophages, iv) characterization of the Rho-RNAP interaction during the transcription and identification of the NusG terminators as well as mode of interactions with the Rho.

Publications in 2017 – 2018.

- 1) Ghosh, G., Reddy, J. Sambhare, S. and Sen, R. (2018) A bacteriophage capsid protein is an inhibitor of a conserved transcription terminator of various bacterial pathogens. **Journal of Bacteriology**, 200(1): e00380-17, 1-16.
- 2) Mitra, P., Ghosh, G., Hafeezunnisa, M. and Sen, R. (2017). Rho protein: mechanism and

action. **Annual Review of Microbiology**, 71, 687-709.

In press 2018:

- 1) Chhakchhuak, P.I.R., Khatri, A. and Sen, R. (2018) Mechanism of action of bacterial transcription terminator Rho. **Proceedings of Indian Science Academy**.

अन्य वैज्ञानिक सेवाएँ / सुविधाएँ
other scientific services / facilities

LABORATORY ANIMAL FACILITY

Faculty Coordinators	Murali D Bashyam Rashna Bhandari	Staff Scientist (since Feb 2018) Staff Scientist (since Feb 2018)
Research Facility Manager	Raghavendrachar Jois	Staff Scientist
Other Members	Hole Jayant Pundalikrao S. Harinarayana Rao Pranjali Pore Sridhar Kavela Navitha Bedarakota	Officer In-Charge (till May 2017) Officer In-Charge (since Oct 2017) Veterinarian (since March 2018) Technical Officer Technical Assistant

Objectives

1. The main objective of the Laboratory Animal Facility (LAF) is to breed, maintain and supply laboratory animals to institutional scientists. Breeding and experimentation of all strains of mice is undertaken in individually ventilated caging systems;
2. To support research programmes that promote the health and well being of people and animals by facilitating high quality and scientifically sound research with animals;
3. To comply with regulatory government body (CPCSEA) requirements for animal experimentation and breeding; and
4. To maintain a stable and contained environment for animals and personnel working in the facility, to ensure consistent animal quality and reduce operational costs.

Summary of work done until the beginning of this reporting year (up to March 31, 2017)

The CDFD LAF started its activities on July 1, 2011, within the premises of M/s Vimta Labs Limited, located at Genome Valley, Shameerpet, Hyderabad. Infrastructure was established to

house mice in individually ventilated cages (IVCs), and conduct standard experimental procedures. All procedures conducted on animals housed in this facility are approved by the Institutional Animal Ethics Committee (IAEC) constituted by the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), Ministry of Environment, Forest and Climate Change (MoEF & CC), Govt. of India, at M/s Vimta Labs Ltd. Until March 2017, the facility housed approximately 1200 mice of five different strains, and in 2016-17, users were supplied with 749 mice for IAEC approved experimentation.

Details of the progress made in the current reporting year (April 1, 2017 - March 31, 2018)

During this reporting year, the CDFD LAF has housed five inbred mouse strains, including *Ip6k1*, *Nnat*, C57BL/6, *FoxN1^{nu}* and BALB/c. Mice were bred to expand the colonies and meet CDFD users' requirements. Currently this facility has 583 adult and 79 newborn mice housed in 461 IVC cages (Table 1). During the year, 937 mice were supplied to users for IAEC approved experimentation.

Strains	Total (Male + Female)	Under Breeding (Male + Female)	Supplied during 2017-18
<i>Ip6k1</i>	99+77	05+10	36
<i>Nnat</i> ΔNEO/ΔI ²	113+109	06+06	90
BALB/c	35+28	08+16	653
C57BL/6	47+34	06+12	86
<i>Foxn1^{nu}</i>	26+15	10+20	72

Table 1. Strain-wise break up of adult mice housed at LAF as on March 31, 2018, and supplied to users during 2017-18.

Routine IAEC approved procedures conducted on these animals include blood collection for measurement of biochemical parameters, embryo collection for the preparation of embryonic fibroblasts, tail biopsies for genotyping analysis and necropsy for histopathological analysis. Some of the experiments conducted during 2017-18 are highlighted below:

- ❖ 253 BALB/c mice were injected intravenously with *Candida glabrata* for studies on comparative bio-burden of different *Candida* strains.
- ❖ 100 BALB/c mice were injected with the non-pathogenic mycobacteria, *M. smegmatis*, expressing some candidate *Mtb* proteins, to study the *in vivo* immunomodulatory role of these proteins.

- ❖ 90 *Nnat* mice were used for measurement of biochemical parameters.
- ❖ 86 C57BL/6 and 43 BALB/c mice were injected with thioglycolate by intra-peritoneal route for the generation of macrophages.
- ❖ 76 BALB/c mice were used to study the *in vivo* anti-inflammatory roles of recombinantly purified PPE2 and PPE18 proteins of *Mycobacterium tuberculosis*.
- ❖ 72 *FoxN1^{nu}* athymic mice were injected with oncogenic cell lines to study tumour progression and metastasis.
- ❖ 60 BALB/c mice were used to study the effect of *Mycobacterium tuberculosis* protein PPE18-coated nanoparticles on microbial sepsis.

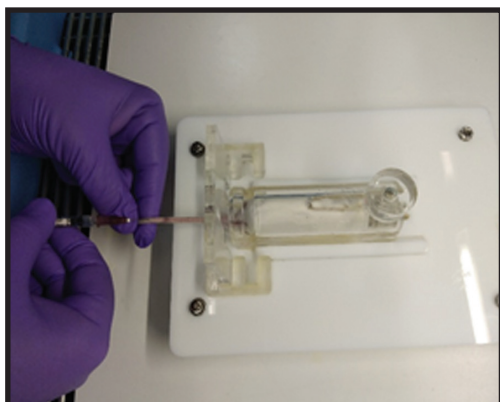


Figure - 1

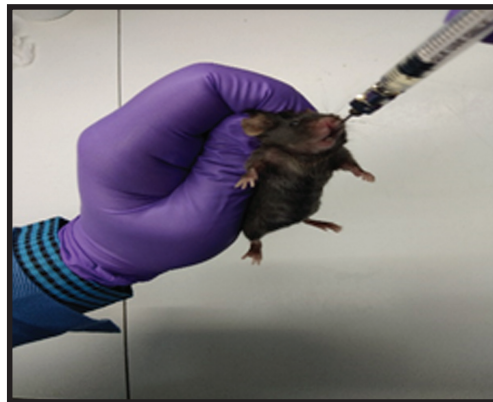


Figure - 2

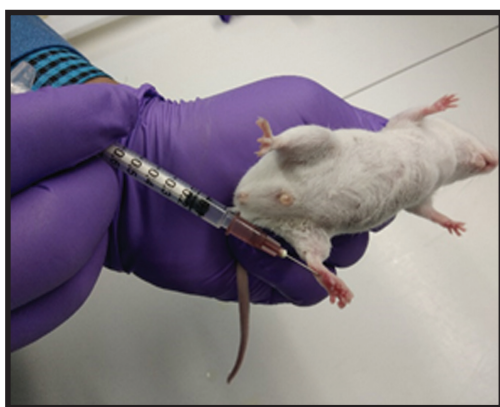


Figure - 3



Figure - 4

Figure 1. Tail vein injection of *Candida glabrata* into female Balb/c mice. **Figure 2.** Oral administration of dextrin to *Ip6k1* mice. **Figure 3.** Subplantar injection of carrageenan in hind paw of Balb/c mice to induce oedema. **Figure 4.** *FoxN1^{nu}* athymic nude mice bred successfully at the CDFD Laboratory Animal Facility.

- ❖ 60 BALB/c mice were used to study the effect of *Mycobacterium tuberculosis* protein PPE18 on caecal ligation and puncture induced sepsis.
- ❖ 43 BALB/c mice were injected subcutaneously with protein antigens and polyclonal antibodies were generated successfully.
- ❖ 36 *Ip6k1* mice were used for histopathological and physiological analyses of testes and gastrointestinal tract.
- ❖ 18 BALB/c mice were used to study the toxicity of novel phytochemicals from medicinal plants.

The IAEC approved projects in progress during this reporting year are mentioned in Table 2.

S. No	Projects in progress
1	Functional analysis of Neuronatin's second intron by knock out strategy
2	Establishment and histopathological characterization of <i>Ip6k1</i> knockout mice – version 2
3	Signal transduction pathway in immune cells regulating their innate and effector functions during oxidative stress
4	Protocol for comparative bio-burden study of fifteen strains of <i>Candida glabrata</i> in BALB/c mice
5	Immunization of BALB/c mice for generation of antibodies against few purified recombinant mycobacterial proteins
6	Studying the effect of PPE18 (Rv1196) on LPS induced endotoxaemia in mice
7	Use of nude mice in the study of tumorigenesis
8	Protocol for generation of mouse /rat polyclonal antibodies - version 2
9	Isolation of macrophages from BALB/c mice
10	Studying the immunomodulatory role of some candidate recombinantly purified proteins of mycobacteria
11	Studying the <i>in vivo</i> immunomodulatory role of some candidate PE/PPE proteins of <i>Mycobacterium tuberculosis</i> recombinantly over-expressed in the non pathogenic mycobacterial strain of <i>M. smegmatis</i>
12	Studying the <i>in vivo</i> epigenetic role of some candidate proteins of <i>Mycobacterium tuberculosis</i> recombinantly over expressed in the non pathogenic mycobacterial strain of <i>M. smegmatis</i>
13	Protocol for testing tumorigenic and metastatic potential in nude mice
14	Investigating potential of <i>Mycobacterium tuberculosis</i> protein PPE18 coated nanoparticles as therapy for microbial sepsis
15	Protocol for comparative vaginal bio-burden analysis of <i>Candida glabrata</i> strains in BALB/c mice
16	Protocol for comparative bio-burden analysis of <i>Candida glabrata</i> strains in C57BL/6 mice
17	Protocol for testing tumorigenic and metastatic potential of novel cancer related genes in nude mice
18	Protocol for studying tumorigenic potential of novel genes associated with pancreatic cancer in nude mice
19	Protocol for studying tumorigenic and metastatic potential of Head and Neck cancer and Esophageal cancer genes in nude mice
20	Identification and molecular characterization of the CgHog1 kinase interactome: impact on iron homeostasis and <i>Candida</i> pathogenesis
21	Studying the <i>in vivo</i> anti-inflammatory roles of recombinantly purified PPE2 and PPE18 proteins of <i>Mycobacterium tuberculosis</i>
22	Protocol for screening of novel phytochemical(s) from medicinal plants identified on the basis of indigenous knowledge for chemo-prevention and anti-cancer efficacy against Lung cancer
23	Protocol for screening of novel phytochemical(s) from medicinal plants identified on the basis of indigenous knowledge for anti-cancer efficacy against A549 cell induced lung cancer xenograft model

Table 2. IAEC approved projects proposed by various groups at CDFD, in progress during 2017-18.

We are close to the completion of CDFD's own Experimental Animal Facility which is under construction in the CDFD campus at Uppal, Hyderabad. We are working towards the completion of the facility, to ensure its compliance and registration with CPCSEA, and enable the start of operations.

Future direction

Once the CDFD Experimental Animal Facility is operational, we plan to expand our breeding

colonies, and house rabbits, rats, and additional transgenic mouse strains to add to the repertoire of experimental animal research being conducted at CDFD. We also aim to develop cryopreservation, archiving and retrieval of transgenic mouse strains for future use.

BIOINFORMATICS

In-charge

M Kavita Rao

Staff Scientist

Members

R Chandra Mohan
Prashanthi Katta
S Vijay Kumar

Technical Officer
Junior Assistant
Technical Assistant (On Contract)

Objectives

1. To maintain various servers, workstations, PCs, printers and other peripheral devices;
2. To maintain CDFD website, to provide web based services and e-mail services;
3. To maintain Institute-wide LAN/WAN as well as the internet connectivity;
4. To secure CDFD network from security threats;
5. To integrate Institute's network into National and International grid computing networks;
6. To coordinate the procurement process of servers, workstations, PCs, laptops, printers, other peripheral devices and software required.

Summary of work done until the beginning of this reporting year (upto March 31, 2017)

- ❖ Activities related to installation, administration and maintenance of servers which provide various services, databases and computational jobs were undertaken.
- ❖ Internet, web, email and intranet services have been provided with enhanced functionalities.
- ❖ Initiated procurement of high-end server.
- ❖ High-end PCs, laptops, scanners and printers were procured and installed.
- ❖ Initiated the process of bulk procurement of Desktop Computers.
- ❖ Shifting of 4 Mbps P2P leased line from Gruhakalpa campus to Uppal Hostel Campus.
- ❖ Initiated the procurement of high-end network equipment including fibre and PoE switches for permanent campus.

- ❖ AMC of high-end servers to OEMs.

Details of progress made in the current reporting year (April 1, 2017 - March 31, 2018)

- ❖ Successfully retrofitted the data centre high-end rack mounted servers, racks, firewall and switches including the internet leased lines of NKN and BSNL ISP from Tuljaguda Campus to permanent campus and configured successfully with no data loss and minimal downtime.
- ❖ Actively coordinated the campus shifting activity from both the erstwhile interim campuses located at CDFD Hostel & Residential complex, Uppal and at Tuljaguda to permanent campus at Uppal by shifting all the PCs, peripheral items and network switches.
- ❖ Procured high-end network switches and wi-fi network equipment for permanent campus at Uppal and configured successfully.
- ❖ Activities related to installation, administration and maintenance of high-end servers which provide various services, databases and computational jobs were undertaken.
- ❖ Internet, web, email and other intranet services are being provided with enhanced functionalities.
- ❖ High-end Server was procured for upgrading the existing email server.
- ❖ High-end PCs (30 Nos); laptops; scanners and printers were procured and successfully installed.
- ❖ Owing to the expiry of the PC Annual Maintenance Contract with M/s Accel Frontline Limited, new tender was processed and offered to a new AMC service provider,

INSTRUMENTATION

Head	Raghavendrachar J	Staff Scientist
Other Members	Mr. R N Mishra	Technical Officer
	Mrs S D Varalaxmi	Technical Officer
	Mr M Laxman	Technical Officer
	Mr Satyanarayana RMK	Technical Officer
	Mr T Ramakrishna Reddy	Tech. Assistant

Objectives

To maintain repair and service all the equipment in laboratory. To provide pre-installation requirements for new instruments and to coordinate with the manufacturers / their agents in Installation and warranty service of the new instruments. Also to provide the reports on the newly arrived instruments and to follow up with the suppliers for short shipped items.

Summary of work done until the beginning of this reporting year (April 1, 2016 – March 31, 2017) :

During the year 2016-17, we have installed 25 new equipments like Shimadzu HPLC Prominence I LC 2030C, AB 3500 Genetic Analyzer HD, Spectromax M5 multimode reader, etc. and we have also completed 269 work orders for repair & maintenance of various laboratory equipments.

We are involved in coordinating the operations of sophisticated instruments in CDFD through an Equipment operations outsourcing agency, M/s Sandor Life Sciences. We are also involved in coordinating with M/s Vimta Labs for CDFD animal experimentation facility in their facility at Shameerpet.

Details of progress made in the current reporting year (April 1, 2017 – March 31, 2018)

During the year 2017-18, we have installed 56 new equipments like Nano ITC, Chemidocs,

Water Bath Shakers, Refrigerated Table Top Centrifuges, Non refrigerated centrifuges, PCR Machines, Refrigerated Incubators, Refrigerated Incubator Shakers, Power supplies, etc. and we have also completed 289 work orders for repair & maintenance of various laboratory equipments.

Further, we were involved in installation of 2 nos of Heavy duty double door automated autoclaves, Necropsy table, RO water systems, IVC Cages, etc in the newly constructed Experimental Animal Facility. We were also involved in shifting of the interim CDFD laboratory from Tuljaguda complex, Nampally to Uppal campus. To facilitate the shifting, we got Laboratory benches installed in Uppal campus and got them to useable condition. After shifting we have installed all the equipment at various locations.

We are involved in coordinating the operations of sophisticated instruments in CDFD. We are also involved in coordinating with M/s Vimta Labs for CDFD animal experimentation facility in their facility at Shameerpet.

In addition, we were involved in organizing the audio & visual requirements for presentations in various seminars, lectures and workshops, CDFD Foundation day lecture at IICT auditorium. We have maintained most of the equipment with maximum uptime in the Laboratory. Most of the Instruments are maintained by our Instrumentation staff, thereby saving on the expensive AMCs and with very little downtime of the equipment.

प्रकाशन
Publications

RESEARCH PAPERS

A. Publications during the year 2017

1. Abraham PR, Pathak N, Pradhan G, Sumanlatha G and Mukhopadhyay S (2017). The N-terminal domain of Mycobacterium tuberculosis PPE17 (Rv1168c) protein plays a dominant role in inducing antibody responses in active TB patients. *PLoS One*, 12(6): e0179965.
2. Alber M, Kalscheuer VM, Marco E, Sherr E, Lesca G, Till M, Gradek G, Wiesener A, Korenke C, Mercier S, Becker F, Yamamoto T, Scherer SW, Marshall CR, Walker S, Dutta UR, Dalal AB, Suckow V, Jamali P, Kahrizi K, Najmabadi H, Minassian BA (2017). ARHGEF9 disease: Phenotype clarification and genotype-phenotype correlation. *Neurology Genetics* 26;3(3):e148.
3. Ali A and Tyagi S (2017). Diverse roles of WDR5-RbBP5-ASH2L-DPY30 (WRAD) complex in the functions of the SET1 histone methyltransferase family. *Journal of Biosciences*, 42 (1): 155-159.
4. Ali A, Sailaja NV, Chinchole A and Tyagi S (2017). MLL/WDR5 Complex Regulates Kif2A Localization to Ensure Chromosome Congression and Proper Spindle Assembly during Mitosis. *Developmental Cell*, 41 (6). 605-622.e7.
5. Aparna Y, Kumari MD, Prasanthi S, Jha A, Satyavathi VV and Anitha M (2017). Effect of polyamines on mechanical and structural properties of Bombyx mori silk. *Biopolymers*, 107 (1): 20-27.
6. Basu Baul TS, Dutta D, Duthie A, Guchhait N, Rocha BGM, Guedes da Silva MFC, Mokhamatam RB, Raviprakash N, Manna SK (2017) New dibutyltin(IV) ladders: Syntheses, structures and, optimization and evaluation of cytotoxic potential employing A375 (melanoma) and HCT116 (colon carcinoma) cell lines in vitro. *Journal of Inorganic Biochemistry* 166: 34-48.
7. Basu Baul TS, Kehie P, Duthie A, Guchhait N, Raviprakash N, Mokhamatam RB, Manna SK, Armata N, Scopelliti M, Wang R, Englert U (2017) Synthesis, photophysical properties and structures of organotin- Schiff bases utilizing aromatic amino acid from the chiral pool and evaluation of the biological perspective of a triphenyltin compound. *Journal of Inorganic Biochemistry* 168: 76-89.
8. Jagadisan B, Ranganath P (2017). Glycogen storage disease type VI with a novel mutation in the PYGL gene. *Indian Pediatrics* 54(9):775-776.
9. Bhat KH, Srivastava S, Kumar KS, Ghosh S and Mukhopadhyay S (2017). The PPE2 protein of Mycobacterium tuberculosis translocates to host nucleus and inhibits nitric oxide production. *Scientific Reports*, 7: 39706.
10. Chaudhary AK, Mohapatra R, Nagarajaram HA, Ranganath P, Dalal A, Dutta A, Danda S, Girisha KM and Bashyam MD (2017). The novel EDARp.L397H missense mutation causes autosomal dominant hypohidrotic ectodermal dysplasia. *Journal of the European Academy of Dermatology and Venereology*, 31(1): e17-e20.
11. Cheng T, Wu J, Wu Y, Chilukuri RV, Huang L, Yamamoto K, Feng L, Li W, Chen Z, Guo H, Liu J, Li S, Wang X, Peng L, Liu D, Guo Y, Fu B, Li Z, Liu C, Chen Y, Tomar A, Hilliou F, Montagné N, Jacquin-Joly E, d'Alençon E, Seth RK, Bhatnagar RK, Jouraku A, Shiotsuki T, Kadono-Okuda K, Promboon A, Smagghe G, Arunkumar KP, Kishino H, Goldsmith MR, Feng Q, Xia Q, Mita K (2017). Genomic adaptation to polyphagy and insecticides in a major East Asian noctuid pest. *Nature Ecology & Evolution*, 1(11): 1747-1756.
12. Das Bhowmik A, Gupta N, Dalal A and Kabra M (2017). Whole exome sequencing identifies a homozygous nonsense variation in ALMS1 gene in a patient with syndromic obesity. *Obesity Research & Clinical Practice*, 11(2): 241-246.
13. Deborah DA, Vemireddy LR, Roja V, Patil S, Choudhary GP, Noor S, Srividhya A, Kaliappan A, Sandhya Rani B, Satyavathi VV, Anuradha G, Radhika K, Yamini KN, Gopalakrishna MK, Ranjith Kumar N, Siddiq EA and Nagaraju J (2017). Molecular dissection of QTL governing grain size traits employing association and linkage mapping in Basmati rice. *Molecular Breeding*, 37(6): 77.
14. Dev RR, Ganji R, Singh SP, Mahalingam S, Banerjee S, Khosla S (2017) Cytosine methylation by DNMT2 facilitates stability

- and survival of HIV-1 RNA in the host cell during infection. *Biochemical Journal* 474: 2009-2026.
15. Dutta U, Bahal A, Vineeth VS, Vasantha S, Ranganath P and Dalal A (2017). A novel mosaic complex supernumerary marker chromosome in a girl with seizures: Systematic characterization of the complex marker. *Gene Reports*, 8: 128-133.
 16. Francis F, Bhat V, Balachander B, Khare C, Bethou A, Dalal A, Ponnala R (2017). Look Up to Diagnose Down! *Indian Journal of Pediatrics* 84(12):961-962.
 17. Gangula NR, Maddika S (2017). Interplay between the phosphatase PHLPP1 and E3 ligase RNF41 stimulates proper kinetochore assembly via the outer-kinetochore protein SGT1. *J Biol Chem*. 292(34): 13947-13958.
 18. Ghosh A, Sengupta A, Pavan Kumar SG, Ali N, Rama Rao EVVS, Bung N, Gopalakrishnan B, Pal M and Halder D (2017). A novel SIRT1 inhibitor, 4bb induces apoptosis in HCT116 human colon carcinoma cells partially by activating p53. *Biochemical and Biophysical Research Communications*, 488(3): 562-569.
 19. Ghosh G, Reddy J, Sambhare S and Sen R (2017). A Bacteriophage Capsid Protein Is an Inhibitor of a Conserved Transcription Terminator of Various Bacterial Pathogens. *Journal of Bacteriology*, 200 (1). e00380-17.
 20. Gopinath, G. and Srikeerthana, K. and Tomar, A. and Sekhar, S.M.C. and Arunkumar, K.P. (2017) RNA sequencing reveals a complete but an unconventional type of dosage compensation in the domestic silkworm *Bombyx mori*. *Royal Society Open Science*, 4 (7). p. 170261.
 21. Gouin A, Bretaudeau A, Nam K, Gimenez S, Aury JM, Duvic B, Hilliou F, Durand N, Montagne N, Darboux I, Kuwar S, Chertemps T, Siaussat D, Bretschneider A, Mone Y, Ahn SJ, Hanniger S, Grenet ASG, Neunemann D, Maumus F, Luyten I, Labadie K, Xu W, Koutroumpa F, Escoubas JM, Llopis A, Maibeche-Coisne M, Salasc F, Tomar A, Anderson AR, Khan SA, Dumas P, Orsucci M, Guy J, Belser C, Alberti A, Noel B, Couloux A, Mercier J, Nidelet S, Dubois E, Liu NY, Boulogne I, Mirabeau O, Le Goff G, Gordon K, Oakeshott J, Consoli FL, Volkoff AN, Fescemyer HW, Marden JH, Luthe DS, Herrero S, Heckel DG, Wincker P, Kergoat GJ, Amselem J, Quesneville H, Groot AT, Jacquin-Joly E, Negre N, Lemaitre C, Legeai F, d'Alencon E, Fournier P (2017). Two genomes of highly polyphagous lepidopteran pests (*Spodoptera frugiperda*, Noctuidae) with different host-plant ranges. *Scientific Reports*, 7: 11816.
 22. Guo H, Cheng T, Chen Z, Jiang L, Guo Y, Liu J, Li S, Tanai K, Asaoka K, Kadono-Okuda K, Arunkumar KP, Wu J, Kishino H, Zhang H, Seth RK, Gopinathan KP, Montagné N, Jacquin-Joly E, Goldsmith MR, Xia Q and Mita K (2017). Expression map of a complete set of gustatory receptor genes in chemosensory organs of *Bombyx mori*. *Insect Biochemistry and Molecular Biology*, 82: 74–82.
 23. Harms FL, Girisha KM, Hardigan AA, Kortüm F, Shukla A, Alawi M, Dalal A, Brady L, Tarnopolsky M, Bird LM, Ceulemans S, Bebin M, Bowling KM, Hiatt SM, Lose EJ, Primiano M, Chung WK, Juusola J, Akdemir ZC, Bainbridge M, Charng WL, Drummond-Borg M, Eldomery MK, El-Hattab AW, Saleh MAM, Bézieau S, Cogné B, Isidor B, Küry S, Lupski JR, Myers RM, Cooper GM and Kutsche K (2017). Mutations in *EBF3* Disturb Transcriptional Profiles and Cause Intellectual Disability, Ataxia, and Facial Dysmorphism. *American Journal of Human Genetics*, 100 (1): 117-127.
 24. Himabindu P and Anupama K (2017). Decreased expression of stable RNA can alleviate the lethality associated with RNase E deficiency in *Escherichia coli*. *Journal of Bacteriology*, 199 (8): e00724-16.
 25. Kasbekar DP (2017). Ascus dysgenesis in hybrid crosses of *Neurospora* and *Sordaria* (Sordariaceae). *Journal of Genetics*, 96 (3): 457-463.
 26. Kasbekar DP and Rekha S (2017). *Neurospora tetrasperma* crosses heterozygous for hybrid translocation strains produce rare eight-spored asci-bearing heterokaryotic ascospores. *Journal of Biosciences*, 42 (1): 15-21.
 27. Khandelwal R, Sipani R, Govinda Rajan S, Kumar R, Joshi R (2017). Combinatorial action of Grainyhead, Extradenticle and Notch in regulating Hox mediated apoptosis in *Drosophila* larval CNS. *PLoS Genetics* 13(10): e1007043.

28. Kumar P and Maddika S (2017). Cellular Dynamics Controlled by Phosphatases. **Journal of the Indian Institute of Science**, 97 (1): 129-145.
29. Kumar P, Munnangi P, Chowdary KR, Shah VJ, Shinde SR, Kolli NR, Halehalli RR, Nagarajaram HA, Maddika S (2017). A human tyrosine phosphatase interactome mapped by proteomic profiling. **J Proteome Res**. 16(8): 2789-2801.
30. Kumar R and Bashyam MD (2017). Multiple oncogenic roles of nuclear β -catenin. **Journal of Biosciences**, 42 (4): 695-707.
31. Malla AB and Bhandari R (2017). IP6K1 is essential for chromatoid body formation and temporal regulation of TNP2 and PRM2 expression in mouse spermatids. **Journal of Cell Science**, 130 (17): 2854-2866.
32. Malla AB and Bhandari R (2017). IP6K1 is indispensable for the temporal regulation of mouse spermiogenic proteins. **Cell Biology Newsletter, published by Indian Society of Cell Biology** 36: 38-39.
33. Mitra P, Ghosh G, Hafeezunnisa Md and Sen R (2017). Rho Protein: Roles and Mechanisms. **Annual Review of Microbiology**, 71 (1): 687-709.
34. Mukhopadhyay S, Ghosh S (2017). Mycobacterium tuberculosis: what is the role of PPE2 during infection? **Future Microbiology**, 12(6): 457-460.
35. Pandey SS, Patnana PK, Rai R and Chatterjee S (2017). Xanthoferrin, the α -hydroxy carboxylate type siderophore of *Xanthomonas campestris* pv. *campestris* required for optimum virulence and growth inside cabbage. **Molecular Plant Pathology**, 18 (7): 949–962.
36. Pandey SS, Singh P, Samal B, Verma RK and Chatterjee S (2017). Xanthoferrin Siderophore Estimation from the Cell-free Culture Supernatants of Different *Xanthomonas* Strains by HPLC. **Bio-Protocol**, 7 (14): e2410.
37. Rao S and Nandineni MR (2017). Genome sequencing and comparative genomics reveal a repertoire of putative pathogenicity genes in chilli anthracnose fungus *Colletotrichum truncatum*. **PLoS One**, 12 (8). e0183567.
38. Tallapaka KB, Ranganath P, Dalal A (2017). Variable Expressivity and Response to Bisphosphonate Therapy in a Family with Osteoporosis Pseudoglioma Syndrome. **Indian Pediatrics**, 54(8):681-683.
39. Saranathan R, Sudhakar P, Sawant AR, Tomar A, Madhangi M, Sah S, Annapurna S, Arunkumar KP and Prashanth K (2017). Disruption of tetR type regulator adeN by mobile genetic element confers elevated virulence in *Acinetobacter baumannii*. **Virulence**, 8 (7): 1316-1334.
40. Sarkar A and Nandineni MR (2017). Development of a SNP-based panel for human identification for Indian populations. **Forensic Science International: Genetics**, 27: 58-66.
41. Sarkar A, Stoneking M and Nandineni MR (2017). Unraveling the human salivary microbiome diversity in Indian populations. **PLoS One**, 12 (9): e0184515.
42. Satyavathi VV, Ghosh R and Srividya S. (2017). Long Non-Coding RNAs Regulating Immunity in Insects. **Non-Coding RNA**, 3 (1): e14.
43. Shah A, Ganguli S, Sen J and Bhandari R (2017). Inositol Pyrophosphates: Energetic, Omnipresent and Versatile Signalling Molecules. **Journal of the Indian Institute of Science**, 97 (1): 23-40.
44. Shinde SR, Maddika S (2017). PTEN regulates glucose transporter recycling by impairing SNX27 retromer assembly. **Cell Reports**, 21 (6): 1655-1666.
45. Singh M and Nandineni MR (2017). Population genetic analyses and evaluation of 22 autosomal STRs in Indian populations. **International Journal of Legal Medicine**, 131 (4): 971-973.
46. Taware, R; Taunk, K; Pereira, JAM; Dhakne, R; Kannan, N; Soneji, D; Camara, JS; Nagarajaram, HA; Rapole, S (2017). Investigation of urinary volatile alterations in head and neck cancer: a non-invasive approach towards diagnosis and prognosis. **Metabolomics**, 13: 111.
47. Uttarilli A, Pasumarthi D, Ranganath P and Dalal AB (2017). Functional characterization of arylsulfatase B mutations in Indian patients with Maroteaux-Lamy syndrome (mucopolysaccharidosis type VI). **Gene**, 599: 19-27.
48. Verma N and Manna SK (2017). Advanced Glycation End Products (AGE) Potentiates

Cell Death in p53 Negative Cells via Upregulation of NF- κ B and Impairment of Autophagy. *Journal of Cellular Physiology*, 232(12): 3598-3610.

B. Publications in 2018 (Till March 31, 2018)

49. Aggarwal S, Tandon A, Das Bhowmik A and Dalal A (2018). Autopsy findings in EPG5-related Vici syndrome with antenatal onset: Additional report of Focal cortical microdysgenesis in a second trimester fetus. *American Journal of Medical Genetics Part A*, 176: 499-501.
50. Aggarwal S, Tandon A, Das Bhowmik A, Jain JMN and Dalal A (2018). A Dymorphology Based Systematic Approach Toward Perinatal Genetic Diagnosis in a Fetal Autopsy Series. *Fetal and Pediatric Pathology*, 37(1): 49-68.
51. Angara RK, Yousuf S, Gupta SK and Ranjan A (2018). An I κ B like protein from mycobacteria regulates leuCD operon and induces dormancy-like growth arrest in Mycobacterium smegmatis. *Tuberculosis*, 108: 83-92.
52. Ansari MZ, Kumar A, Ahari D, Priyadarshi A, Padmavathi L, Bhandari R and Swaminathan R (2018). Protein Charge Transfer Absorption Spectra: An Intrinsic Probe to Monitor Structural and Oligomeric Transitions in Proteins. *Faraday Discuss*, 114 (3). 586 a.
53. Anwar T, Sen B, Aggarwal S, Nath R, Pathak N, Katoch A, Aiyaz M, Trehanpati N, Khosla S, Ramakrishna G. (2018) Differentially regulated gene expression in quiescence versus senescence and identification of ARID5A as a quiescence associated marker. *Journal of Cell Physiology*, 233:3695-3712.
54. Das Bhowmik A, Vijayalakshmi SR and Dalal A (2018). Tarsal-carpal coalition syndrome: Report of a novel missense mutation in NOG gene and phenotypic delineation. *American Journal of Medical Genetics Part A* 176 (1): 219-224.
55. Dolasia K, Bisht MK, Pradhan G, Udgata A and Mukhopadhyay S (2018). TLRs/NLRs: Shaping the landscape of host immunity. *International Reviews of Immunology*, 37(1): 3-19.
56. Ghosh DK, Roy A and Ranjan A (2018). Aggregation-prone regions in HYPK help it to form sequestration complex for toxic protein aggregates. *Journal of Molecular Biology*, 430(7): 963-986.
57. Ghosh, DK, Roy A, Ranjan A (2018). Disordered nanostructure in Huntingtin Interacting Protein K acts as a stabilizing switch to prevent protein aggregation. *Biochemistry*, 53(13): 2009-2023.
58. Javvadi S, Pandey SS, Mishra A, Pradhan BB and Chatterjee S (2018). Bacterial cyclic β -(1,2)-glucans sequester iron to protect against iron-induced toxicity. *EMBO Reports*, 19: 172-186.
59. Kar A, Phadke SR, Das Bhowmik A, and Dalal A (2018). Whole exome sequencing reveals a mutation in ARMC9 as a cause of mental retardation, ptosis, and polydactyly. *American Journal of Medical Genetics Part A* 176 (1): 34-40.
60. Kumar R, Raman R, Kotapalli V, Gowrishankar S, Pyne S, Pollack JR and Bashyam MD (2018). Ca²⁺/nuclear factor of activated T cells signaling is enriched in early-onset rectal tumors devoid of canonical Wnt activation. *Journal of Molecular Medicine*, 96 (2): 135-146.
61. Narayanan DL, Deshpande D, Das Bhowmik A, Varma DR and Dalal A (2018). Familial choreoathetosis due to novel heterozygous mutation in PDE10A. *American Journal of Medical Genetics Part A*, 176(1): 146–150.
62. Puri RD, Kapoor S, Kishnani PS, Dalal A, Gupta N, Muranjan M, Phadke SR, Sachdeva A, Verma IC and Mistry PK (2018). Diagnosis and Management of Gaucher Disease in India - Consensus Guidelines of the Gaucher Disease Task Force of the Society for Indian Academy of Medical Genetics and the Indian Academy of Pediatrics. *Indian Pediatrics*, 55 (2): 143-153.
63. Raghavendra V and Haldar D (2018). Mammalian target of rapamycin complex 2 (mTORC2) controls glycolytic gene expression by regulating Histone H3 Lysine 56 acetylation. *Cell Cycle*, 17(1): 110-123.
64. Ram BM, Jayashree D, Kulkarni R, Usha R, Usha B, Usha Rani P, Islam M, Trehanpati N and Ramakrishna G (2018). Human papillomavirus (HPV) oncoprotein E6 facilitates Calcineurin-Nuclear factor for activated T cells 2 (NFAT2) signaling to promote cellular proliferation in cervical cell carcinoma. *Experimental Cell Research*, 362 (1): 132-141.
65. Rameshwaram NR, Shrivastava R, Pradhan G, Singh P and Mukhopadhyay S.

- Phagosome-lysosome fusion hijack - An art of intracellular bacteria. (2017). **Proceedings of the Indian National Academy of Sciences** 83: 533-548.
66. Sarkar A, Nandineni MR (2018). Association of common genetic variants with human skin color variation in Indian populations. **American Journal of Human Biology**, 30 (1): e23068.
 67. Sengupta A and Haldar D (2018). Human sirtuin 3 (SIRT3) deacetylates histone H3 lysine 56 to promote nonhomologous end joining repair. **DNA Repair**, 61: 1-16.
 68. Shinde SR, Maddika S (2018). Post-translational modifications of Rab GTPases. **Small GTPases**. 9(1-2): 49-56.
 69. Shukla A, Das Bhowmik A, Hebbar M, Rajagopal KV, Girisha KM, Gupta N and Dalal A (2018). Homozygosity for a nonsense variant in AIMP2 is associated with a progressive neurodevelopmental disorder with microcephaly, seizures, and spastic quadriplegia. **Journal of Human Genetics**, 63(1): 19-25.
 70. Yaseen I, Choudhury M, Sritharan M and Khosla S (2018). Histone methyltransferase SUV39H1 participates in host defense by methylating mycobacterial histone-like protein HupB. **The EMBO Journal**, 37 (2): 183-200.
 71. Zargar ZU, Rao MK and Tyagi S (2018). Dynamic site-specific recruitment of RBP2 by pocket protein p130 modulates H3K4 methylation on E2F-responsive promoters. **Nucleic Acids Research**, 46(1): 174-188.
- C. Publications in press (as on 31st March 2018)**
72. Ahmed A, Dolasia K and Mukhopadhyay S. (2018). Mycobacterium tuberculosis PPE18 protein reduces inflammation and increases survival in animal model of sepsis. **Journal of Immunology**.
 73. Bakshi A and Joshi R (2018). Understanding the regulation of neural stem cell proliferation in Drosophila central nervous system. **J Neuroscience Research**.
 74. Behera S, Kapadia B, Kain V, Alamuru-Yellapragada NP, Murunikkara V, Kumar ST, Babu PP, Seshadri S, Shivaridraiah P, Hiriyani J, Gangula NR, Maddika S, Misra P, Parsa KVL (2018). ERK1/2 activated PHLPP1 induces skeletal muscle ER stress through the inhibition of a novel substrate AMPK. **Biochim Biophys Acta**.
 75. Chhakchhuak, P.I.R., Khatri, A. and Sen, R. (2018) Mechanism of action of bacterial transcription terminator Rho. **Proceedings of Indian Science Academy**.
 76. Das Bhowmik A, Patil SJ, Deshpande DV, Bhat V, Dalal A (2018). Novel splice-site variant of UCHL1 in an Indian family with autosomal recessive spastic paraplegia-79. **Journal of Human Genetics**.
 77. Aggarwal S, Das Bhowmik A, Tandon A, Dalal A (2018). Exome sequencing reveals blended phenotype of double heterozygous FBN1 and FBN2 variants in a fetus. **European Journal of Medical Genetics**.
 78. Godbole KG, Ramachandran A, Karkamkar AS, Dalal AB (2018). Compound Heterozygosity for Hb Alpertion (HBB: c.407C>T) and IVS-I-5 (G>C) (HBB: c.92+5G>C) Mutations Presenting as a Moderate Anemia in an Indian Family. **Hemoglobin**.
 79. Gupta P, Zaidi AH, Manna SK. (2018) Suppression of IKK, but not activation of p53 is responsible for cell death mediated by naturally occurring oxidized tetranortriterpenoid. **Journal of Cellular Biochemistry** 2018 May 8. doi: 10.1002/jcb.26879. [Epub ahead of print] PMID: 29738082.
 80. Kumar R, Kotapalli V, Naz A, Gowrishankar S, Rao S, Pollack JR, and Bashyam MD (2018). XPNPEP3 is a novel transcriptional target of canonical Wnt/ β -catenin signalling. **Genes Chromosomes & Cancer**.
 81. Li S, Ajimura M, Chen Z, Liu J, Chen E, Guo H, Tadapatri V, Reddy CG, Zhang J, Kishino H, Abe H, Xia Q, Arunkumar KP, Mita K (2018). A new approach for comprehensively describing heterogametic sex chromosomes. **DNA Res**.
 82. Pandey S.S, Patnana P.K, Padhi Y, and Chatterjee S. (2018). Low-iron conditions induces the hypersensitive reaction and pathogenicity hrp genes expression in Xanthomonas and is involved in modulation of hypersensitive response and virulence. **Environmental Microbiology Reports**.
 83. Patil SJ, Das Bhowmik A, Bhat V, Satidevi Vineeth V, Vasudevamurthy R, Dalal A (2018). Autosomal recessive otofaciocervical syndrome type 2 with novel homozygous

- small insertion in PAX1 gene. **American Journal of Medical Genetics.**
84. Raghunathan, N., Kapshikar RM, Leela JK, Mallikarjun J, Bouloc P, and Gowrishankar J. (2018). Genome-wide relationship between R-loop formation and antisense transcription in Escherichia coli. **Nucleic Acids Research.**
 85. Rasheed, M., Battu A. and Kaur, R. (2018) Aspartyl proteases in Candida glabrata are required for suppression of the host innate immune response. **Journal of Biological Chemistry.**
 86. Shah VJ, Maddika S (2018). CRL7SMU1 E3 ligase complex-driven H2B ubiquitylation functions in sister chromatid cohesion by regulating SMC1 expression. **J Cell Sci.**
 87. Singh P, Rameshwaram NR, Ghosh S and Mukhopadhyay S. (2018). Cell envelope lipids in the pathophysiology of Mycobacterium tuberculosis. **Future Microbiology.**
 88. Tallapaka K, Venugopal V, Dalal A, Aggarwal S (2018). Novel RSPO1 mutation causing 46, XX testicular disorder of sex development with palmoplantar keratoderma: A review of literature and expansion of clinical phenotype. **American Journal of Medical Genetics.**
- D. Other Publications**
1. Aggarwal S (2017). Counseling for Fetal Central Nervous System Defects. **Journal of Fetal Medicine** 4(2): 65–73.
 2. Aggarwal S (2017). Fetal Dysmorphology: An Indispensable Tool for Synthesis of Perinatal Diagnosis. **Genetic clinics** 10(2):11-19.
 3. Kasbekar DP (2017). Pushpa Mittra Bhargava (1928–2017). **Current Science**, 113 (4): 807-808.
 4. Kasbekar DP (2017). Sherlock Holmes, David Perkins, and the missing Neurospora inversions. **Journal of Biosciences**, 42 (1): 5-10.
 5. Kasbekar, D. P. (2017). The science underlying use of DNA evidence to solve crime. Oct 26, LiveLaw.in (<http://www.livelaw.in/science-underlying-use-dna-evidence-solve-crime/>).
 6. Kasbekar, D. P. (2018). Series. A cross-eyed geneticist's view 1. Making sense of the lamin B receptor, a chimeric protein. **J. Biosci.** 43: 235-237.
- E. Patents filed/granted : NIL**

मानव संसाधन विकास
Human Resource Development

PhD Program

For the PhD program, CDFD invites applications from highly motivated candidates willing to take up challenges in modern biology, usually in the month of March. Keeping in view the interdisciplinary nature of modern biology, the Centre especially encourages persons from different scientific disciplines to take up challenges in these areas. Those admitted as Junior Research Fellows (JRFs) are encouraged to take admission in the PhD program of Manipal University or University of Hyderabad.

The eligibility for the program is Masters degree in any branch of Science, Technology or Agriculture from a recognized University or Institute or MBBS. Candidates must have cleared National Eligibility Test (NET) with valid CSIR-JRF or UGC-JRF or DBT-JRF or ICMR-JRF or INSPIRE-PhD. As the number of applicants outnumbers the seats available each year by a ratio of 1:40 or more, eligible candidates are invited for a written examination followed by interviews of short-listed candidates.

As of March 31, 2018 the Centre has 93 Research Scholars working for their doctorates in different areas of research. In the reporting year, 15 Research Scholars have completed PhD and are pursuing careers in science elsewhere in India or abroad.

Postdoctoral Program

In addition to the JRF program, the Centre also carries out training at the post-doctoral level. The post-doctoral fellows are funded through the extramural grants that CDFD receives. Some post-doctoral fellows are also selected competitively by the DST fast track young scientist scheme or the DST N-PDF program or the DBT post-doctoral fellowship program.

Summer Training Program

CDFD provides admissions to summer training program to those students who are supported either by the Indian Academy of Science, Bangalore or Jawaharlal Nehru Centre for Advanced Scientific Research, Bangalore or the Kishore Vigyanik Protsahan Yojna, New Delhi. In the reporting year 20 students received summer training at the Centre.

Training for students from BITS, Pilani

CDFD has an agreement with BITS, Pilani to provide project training to their M.Sc. students. Under this programme, the students spend 6 months to 1 year at CDFD and work on active projects being carried out here. The project work helps the students in gaining hands-on experience in modern biology. In the reporting year, 3 students were given the opportunity to avail training under this programme.

SECTION 'A'
Students Conferred with Ph.D. Degree Duing 01.04.2017 - 31.03.2018

Sl. No.	Name of the Scholar	Supervisor from CDFD	Date of viva voce examination	Title of thesis
1	Ms. Vandana Sharma	Dr. Rupinder Kaur	04.05.2017	"A Study of molecular mechanism(s) underlying interaction of Candida glabrata with mammalian host cells"
2	Ms. C Venkata Lakshmi Manasa	Dr. Rashna Bhandari	23.05.2017	"Investiating the Cellular role of inositol Pyrophosphates in mammals"
3	Mr. Tarique Anwar	Dr. Gayatri Ramakrishna	12.06.2017	"Evaluation of Molecular signatures unique to growth arrest conditions of quiescence and senescence"
4	Mr. Imtiyaz Yaseen	Dr. Sanjeev Khosla	12.06.2017	"Identification and characterization of Mycobacterial proteins interacting with host cell epigenetic circuitry"
5	Mr. Aamir Ali	Dr. Shweta Tyagi	15.06.2017	"Regulation of Cell - cyle Progression by MLL H3K4 HMT complex"
6	Mr. Ajit Roy	Dr. Akash Ranjan	13.06.2017	"Studies on HOSA mediated molecular interactions involved in pathophysiology of Escherichia Coll"
7	Ms. Rachana Roshan Dev	Dr. Sanjeev Khosla	14.06.2017	"Identificatin and characterization of cellular function of mammalian methyltransferase DNMT2"
8	Mr. Sheo Shankar Pandey	Dr. Subhadeep Chatterjee	07.07.2017	"Role of Iron and secreted virulence functions in Xanthomonas Virulence"
9	Ms. Gangua Narmadha Reddy	Dr. M V Subba Reddy	24.08.2017	"Identification and characterization of PHLPP associated proteins in regulation of cell survival"
10	Ms. Parul Singh	Dr. Sangita Mukhopadhyay	06.10.2017	"Molecular and Immunological characterizaion of the Mycobacterium tuberculosis PE protein RV1169C"
11	Ms. Asha Minz	Dr. K P Arun Kumar	24.10.2017	"Insect immunity: Functional characterization of new Candidate genes"
12	Mr. Anujit Sarkar	Dr. N Madhusudan Reddy	08.12.2017	"Human genetic variation studies in indian populations employing DNA - based markers and its application in forensic human identification Purposes"
13	Ms. Lahiri Konada	Dr. Devyani Haldar	26.02.2018	"Understaind the functions of fission yeast Sirtuin Hst4 in DNA replication"
14	Mr. Gourango Pradhan	Dr. Sangita Mukhopadhyay	27.03.2018	"Understanding the role of Secretory Protein PKnG of mycobacterium tuberculosis in the modulation of macrophage - effector functions"
15	Ms. Amrita Sen Gupta	Dr. Devyani Haldar	28.03.2018	"To investigate and Understand the nuclear functions of Mammalian Sirtuin 3"

पुरस्कार एवं सम्मान
Awards and Honours

AWARDS & HONOURS

Dr. Debashis Mitra	Awarded the prestigious J.C. Bose National Fellowship.
Dr. Sangita Mukhopadhyay	ICMR Chaturvedi Ghanshyam Das Jaigopal Memorial Award – 2015
Dr. Subhadeep Chatterjee	<ol style="list-style-type: none"> 1. Appreciation certificate from American Phytopathology Society in recognition for outstanding service as an associate editor for the journal. 2. Elected as a member of Guha Research Conference
Dr Murali D Bashyam	Selected for Senior Scientist award of Association of Biotechnology and Pharmacy during the Annual Meeting to be held at Hyd Univ in December 2017.
Dr. M Subba Reddy	B.M. Birla Science Prize in Biology for the year-2016
Dr. Rashna Bhandari and Dr. R Harinarayanan	3 rd Prize winners of 6 th Inter-Lab Quiz Competition held at CCMB, Hyderabad on 30.11.2017
Dr. Ranjan Sen	<ol style="list-style-type: none"> 1. Elected as a fellow of Indian National Science Academy at the Annual General meeting on 14.10.2018. 2. Elected as a member of Indian Academy of Sciences in its meeting held in December 2017. 3. Elected as fellow of the Telangana Academy of Sciences (FTAS) for the year 2017.
PHD STUDENTS & PROJECT PERSONNEL	
Mr. Debasish Kumar Ghosh	Third Prize in poster papers & Springer award for best poster in International Conference on Electron Microscopy & Allied Techniques EMSI-2017 held at Mahabalipuram, Tamilnadu during (July 17-19, 2017).
Mr. Akshay kumar Shrikondawar	Second Prize in metallography contest (other microscopy) in International Conference on Electron Microscopy & Allied Techniques EMSI-2017 held at Mahabalipuram, Tamilnadu during (July 17-19, 2017).
Ms. Pratyusha Bala	Best Poster Award at NextGen Genomics Biology, Bioinformatics and Technologies conference (NGBT) held at Bhubaneswar during (October 2-4, 2017).
Dr. R Nagender Rao	EMBO short-term fellowship for a period of 3 months at University of Gothenburg, Sweden from 25.09.2017 to 24.12.2017.
Mr. Sriyans Jain and Ms. Richa Gupta	1 st position for Poster Presentation in International Conference "Microbiology in New Millennium: from Molecules to Communities" held at Bose Institute, Kolkata during 27-29 October 2017
Ms. Dipti Deshpande	Best Poster Award at the 86 th SBC (I) Annual Meeting of the Society held at Jawaharlal Nehru University, New Delhi during November 16-19, 2017.
Mr. Rohan Mishra and Mr. V A Ramesh	1 st Prize winners of 6 th Inter-Lab Quiz Competition held at CCMB, Hyderabad on 30.11.2017
Ms. Komal Dolasia	Awarded Dr. G.P. Talwar Young Scientist Award at the 44 th Annual Conference of Indian Immunology Society (IMMUNOCON – 2017) held at Nirma University, Ahmedabad, Gujarat during December 14-16, 2017
Ms. Ashmala Naz	Awarded for Travel grant from IACR to attend the IACR-2018 meeting in Kolkata
Ms. Akruiti Shah	Awarded the CCMB Prize for the Best Poster Presentation at the 2018 International Congress of Cell Biology (ICCB) held in Hyderabad during January 27-31, 2018
Mr. Raju Kumar	DST-SERB support for travel to attend the AACR Annual meeting in Chicago during 14-18 April 2018
Ms. Shalini A	Springer Nature best poster award at INDO-US conference held at Indian Institute of Science, Bangalore during 6-10 March 2018

**व्याख्यान, बैठक, कार्यशाला व
अन्य महत्वपूर्ण कार्यक्रम**

**Lectures, Meetings, Workshops
and Important Events**

LECTURES

Visitor	Title of Lecture	Date
Dr. Swarup Roy Chowdhury Donald Danforth Plant Science Center St. Louis, Missouri, 63132, USA	Heterotrimeric G protein signaling in plants	09.05.2017
Dr. Varsha Singh, faculty Department of Molecular Reproduction, Development & Genetics Indian Institute of Science Bangalore	Of Worms and Bugs: A tale of Sensing and Survival	17.05.2017
Dr. Aravind Rengan Assistant Professor Department of Biomedical Engineering IIT, Hyd.	Theranostics –To be or not to be ?!	07.06.2017
Dr. Supreet Saini Indian Institute of Technology Mumbai	Dynamics of intrinsic antibiotic resistance in <i>E.coli</i> via MAR-SOX-ROB regulon	12.06.2017
Dr. Edward L. Trimble Director Centre for Global Health (CGH) National Cancer Institute, NIH	Priorities for Global Cancer Research	11.07.2017
Dr. Pratibha Boga-Kamat Senior Programme Manager Entrepreneurship, C-CAMP Bangalore	Centre for Cellular And Molecular Platforms (C-CAMP) and it's efforts to promote innovation and entrepreneurship	02.08.2017
Dr. Babu Reddy JN University of California Irvine CA, USA	Molecular mechanisms behind load induced force adaptation of intracellular lipid droplets	10.08.2017
Dr. Kushagra Bansal Dept. of Microbiology and Immunology Harvard Medical School, Boston MA, USA	Thymic induction of immunological self-tolerance: the role of AIRE and its partners	16.08.2017
Dr. Sabarinathan Radhakrishnan Institute for Research in Biomedicine (IRB Barcelona) Spain	Landscape of mutational processes and driver mutations in cancer whole genomes	17.08.2017
Dr. Sathish Kumar Mungamuri Assistant Professor Department of Oncological Sciences Icahn School of Medicine at Mount Sinai, Mount Sinai School of Medicine New York, NY, USA	Understanding the role of heterochromatin in the progression and therapy of p53 wild type tumors	22.08.2017
Dr. Madhu Sudhan Ravindran Post-Doctoral Fellow University of Michigan Medical School USA	Molecular chaperones and motors: From proteostasis to pathogenesis	01.09.2017

Visitor	Title of Lecture	Date
Dr. Ramkumar Sambasivan Institute for Stem Cell Biology and Regenerative Medicine (InStem) Bangalore	Recapitulating developmental cues guide pluripotent stem cell differentiation into dual potential muscle / heart progenitors	17.10.2017
Dr. Sreeramaiah N. Gangappa Postdoctoral Scientist Department of Cell & Developmental Biology John Innes Centre Norwich NR4 7UH United Kingdom	Temperature mediated regulation of plant growth and defense responses	23.10.2017
Dr. Shubhakar K.P. Forensic Physician NHS Tayside & Police Scotland, Dundee, UK	Confronting rape and sexual violence	07.11.2017
Dr. Yogesh S Shouche National Centre for Cell Science (NCCS), Pune	Human microbiome in health and disease: Indian perspective	08.12.2017
Prof. Gabriel Schaaf Department of Plant Nutrition University of Bonn, Germany	Grow or Fight? How inositol pyrophosphates regulate plant defense	08.12.2017
Dr. Prem Kaushal Centre for DNA Finger Printing and Diagnostics (CDFD), Hyderabad	The Nobel Prize in Chemistry 2017- for developing cryo-electron microscopy (cryo- EM) for the high-resolution structure determination of biomolecules in solution	11.12.2017
Dr. Manoj K Bhat Scientist G and Dean (Academics) National Centre for Cell Science (NCCS), Pune	Cancer and metabolic disorders: An overview of ongoing work	08.02.2018
Dr. Sambasivarao Damaraju Professor, University of Alberta, Canada	The long-ignored germline DNA is making a comeback in diagnostics and prognostics for polygenic diseases	23.03.2018

LECTURE SERIES “LEARN FROM THE MASTER”

Visitor	Title of Lecture	Date
Dr Jyotsna Dhawan CSIR Center for Cellular & Molecular Biology, Hyderabad & Institute for Stem Cell Biology & Regenerative Medicine, Bangalore	The quiescent genome: Balancing options, anticipating change	26.04.2017
Dr Roop Mallik Associate Professor Department of Biological Sciences TIFR-Mumbai	A fat story	17.05.2017
Dr Partha Majumder National Institute of Biomedical Genomics Kolkata	In search of the drivers	12.09.2017

IMPORTANT EVENTS

Event	Date
International Yoga Day	21.06.2017
Independence Day	15.08.2017
Observance of Sadbhavana Diwas	18.08.2017
19 th RAP-SAC meeting	29.08.2017 & 30.08.2017
Hindi workshop on Unicode Software	04.09.2017
Hindi Day (Pakwada)	14.09.2017
IISF-2017 celebrations	13.10.2017
36th CDFD Finance Committee Meeting at NII Conference Hall, New Delhi	24.10.2017
Observance Of Vigilance Awareness Week, 2017	30.10.2017
CDFD Building Committee Meeting in DBT, New Delhi	06.11.2017
43rd Meeting of CDFD Governing Council in DBT, New Delhi	07.11.2017
Republic Day Celebrations	26.01.2018
Observance of Martyrs' Day	30.01.2018
National Productivity Week Celebrations	12-18 Feb 2018
Memorandum of Understanding between CDFD, Hyderabad and SVP National Police Academy, Hyderabad.	22.03.2018
27 th CDFD Building Committee meeting held in CDFD, Uppal	23.03.2018
37 th CDFD Finance Committee meeting held in CDFD, Uppal	23.03.2018

**सी डी एफ डी कर्मचारियों की
विदेशों में प्रतिनियुक्ति
Deputations Abroad of
CDFD Personnel**

**List of Staff Members who had been Abroad on Deputation
During the Period from
1st April 2017 to 31st March 2018**

Name of the Employee & Designation	Duration of visit	Place & purpose of visit
Dr. Ranjan Sen Staff Scientist – VI & I/c – Director	25.06.2017 to 30.06.2017	USA: To visit USA during 25-30 June, 2017 (excluding travel period) to attend the FASEB Summer Conference on “Mechanisms & Regulation of Prokaryotic Transcription” held at Vermont Academy, Saxtons River, Vermont, US
Dr. Murali Dharan Bashyam Staff Scientist – VI	12.04.2018 to 24.04.2018	Chicago: (i) To visit his collaborator at the Northwestern University, Chicago on 13.04.2018. (ii) To attend and present his work at “American Association for Cancer Research (AACR) Annual Meeting 2018” during 14-18 April, 2018 at Chicago. (iii) To visit his collaborator at the University of Illinois at Urbana - Champaign during 19-23 April, 2018. (iv) To visit University of Pennsylvania, Philadelphia during 19-23 April, 2018 in place of University of Illinois.
Dr. Sanjeev Khosla Staff Scientist – VI	10.09.2017 to 15.09.2017	Japan: To give a talk at the “12 th Asian Epigenomics Meeting – 2017” during 12-13 September, 2017 at Tomakomai, Hokkaido, Japan
Dr. Akash Ranjan Staff Scientist – VI	15.11.2017 to 21.11.2017	Portugal: To visit Portugal during 15-21 November, 2017 to participate at Proteostasis EMBO Workshop during 17-21 November, 2017 at Erinceira, Portugal.
Dr. Rupinder Kaur Staff Scientist – VI	22.04.2017 to 27.04.2017	Spain : To visit Spain during 22-27 April, 2017 to attend and make a presentation in the “ <i>Young Scientist Networking (YSN) 2017 – Understanding Life</i> ” Conference during 24-25 April, 2017 at CRG, Barcelona, Spain.

Dr. Ashwin B Dalal Staff Scientist –VI	17.05.2017	21.05.2017	Russia: To visit Russia during 17-21 May, 2017 to attend 1 st International Workshop on “Recent advances in rare diseases as a model “(RARD 2017) during 18-20 May, 2017.
Dr. Devyani Halder Staff Scientist – V	31.03.2017	12.04.2017	USA: (i) To attend the Keystone Symposium on Genetic instability and DNA Repair joint with the meeting on DNA Replication and Recombination during 02-06 April, 2017 at Santa Fe, New Mexico, USA. (ii) To deliver a lecture at NICHD, NIH, Bethesda on 10.04.2017.
Dr. N Madhusudan Reddy Staff Scientist – V	17.09.2017	19.07.2017	Thailand: To attend the Asia-Pacific Medico-Legal Association (APMLA) Annual Meeting and International Committee of the Red Cross (ICRC) Regional Workshop on the “Forensic Issues in Human Migration” held at The Central Institute of Forensic Science (CIFS), Ministry of Justice, Pattaya, Thailand.
	23.10.2017	31.10.2017	Germany: To visit Germany from 23.10.2017 to 31.10.2017 to conduct research as Guest Scientist in the laboratory of Prof. Mark Stoneking, Department of Evolutionary Genetics, Max Planck Institute for Evolutionary Anthropology (MPI-EVA), Leipzig, Germany against his seventh visit to Prof. Mark Stoneking’s Laboratory as a part of the “Max Planck Partner Group Programme” (MPPGP) between CDFD and MPI-EVA awarded by the Max Planck Society, Germany

Dr. Rohit Joshi, Staff Scientist – IV	22.06.2017	09.07.2017	USA: To meet Dr. Santanu Banerjee at Syracuse University during 23-24 June, 2017 for scientific interaction and to explore areas of future scientific collaboration.
			To attend Gordon Research Conference in Developmental Biology themed “Generation and Regeneration of Biological Tissues through Cellular Diversification and Cooperation” scheduled to be held during 25-30 June, 2017 at Mount Holyoke College, South Hadley, MA, USA.
			To meet Dr. Pranav Sharma at the Scripps Research Institute, San Diego, CA during 01-03 July, 2017 to discuss on advance data analysis for confocal microscopy and to explore potential of STORM and Light Sheet microscopy in thick tissues imaging like in case of Drosophila brain.
			To meet Dr. Gary Struhl and his group and to give a talk on 05.07.2017 for scientific interactions to explore scientific collaborations.
			To meet Dr. Richard Mann at Columbia University, New York on 06.07.2017.
			To attend Gordon Research Conference in Developmental Biology themed “Generation and Regeneration of Biological Tissues through Cellular Diversification and Cooperation” scheduled to be held during 25-30 June, 2017 at Mount Holyoke College, South Hadley, MA, USA.
			To meet Dr. Pranav Sharma at the Scripps Research Institute, San Diego, CA during 01-03 July, 2017 to discuss on advance data analysis for confocal microscopy and to explore potential of STORM and Light Sheet microscopy in thick tissues imaging like in case of Drosophila brain.
			To meet Dr. Gary Struhl and his group and to give a talk on 05.07.2017 for scientific interactions to explore scientific collaborations.
Dr. R Harinarayanan Staff Scientist – III	06.08.2017	12.08.2017	USA: To attend a conference titled “Molecular Genetics of Bacteria and Phages” scheduled to be held during 07-11 August, 2017 at Madison, Wisconsin, USA.

Details of Foreign visit (01.04.2017 - 31.03.2018)

Sl. No.	Name of the Research Scholar	Group Head	Name of the Conference	Place of Visit	Period of Visit	Expenditure of the visit
1	Mr. Debashish Kumar Ghosh	Dr. Akash Ranjan	EMBO Conference - Proteostasis	Ericeira, Portugal	17.11.2017 to 21.11.2017	1) Travel expenditure met out of EMBO Travel Award of Euros. 500 and 2) rest of the expenditure borne by CDFD
2	Mr. Raviranjana Kumar	Dr. Rohit Joshi	EMBO Workshop on "Neural Development"	Taipei, Taiwan	02.03.2018 to 06.03.2018	No financial liability on CDFD

सीडीएफडी के संकाय एवं अधिकारी
Faculty and Officers of CDFD

SCIENTIFIC GROUP LEADERS (FACULTY)

Dr. Debashis Mitra
Dr. Ranjan Sen
Dr. Sangita Mukhopadhyay
Dr. M D Bashyam
Dr. Sanjeev Khosla
Dr. Sunil Kumar Manna
Dr. Akash Ranjan
Dr. Rupinder Kaur
Dr. Ashwin B Dalal
Dr. Rashna Bhandari
Dr. Devyani Halder
Dr. N Madhusudan Reddy
Dr. Shweta Tyagi
Dr. M V Subba Reddy
Dr. Subhadeep Chatterjee
Dr. Sardesai Abhijit Ajit
Dr. Rohit Joshi
Dr. Prem Singh Kaushal
Dr. R Harinarayanan

ADJUNCT FACULTY

Dr. E A Siddiq
Prof. T Ramasarma
Prof. Anuradha Lohia
Dr. Renu Wadhwa
Dr. Prajnya Ranganath
Dr. Shagun Aggarwal

OTHER GROUP LEADERS

Mr. Raghavendrachar J
Ms. Varsha
Ms. M Kavita Rao

SENIOR ADMINISTRATIVE STAFF

Mr. J Sanjeev Rao

केन्द्र की समितियाँ
(31.03.2018 तक)
Committees of the Centre
(As on 31.03.2018)

MEMBERS OF CDFD SOCIETY

Hon'ble Dr. Harsh Vardhan Hon'ble Minister for Science & Technology and Earth Sciences	-	President
Dr Renu Swarup Secretary, DBT, New Delhi	-	Member (Ex-officio)
Director General CSIR, New Delhi	-	Member (Ex-officio)
Director General Bureau of Police Research and Development (BPR&D) Ministry of Home Affairs, New Delhi	-	Member (Ex-officio)
Joint Secretary & FA DBT, New Delhi	-	Member (Ex-officio)
Joint Secretary (PM) Ministry of Home Affairs, New Delhi	-	Member (Ex-officio)
Joint Secretary & Legal Advisor Ministry of Law & Justice, New Delhi	-	Member (Ex-officio)
Prof Partha P Majumder Director, NIBMG, West Bengal Chairman of Scientific Advisory Committee, CDFD	-	Member (Ex-officio)
Dr A K Rawat Director, DBT, New Delhi	-	Member (Ex-officio)
Prof V S Chauhan ICGEB, New Delhi	-	Member
Prof Dipankar Chatterji Indian Institute of Science (IISc), Bangalore	-	Member
Dr Rakesh K Mishra Director, CCMB, Hyderabad	-	Member
Dr Debashis Mitra Director, CDFD, Hyderabad	-	Member-Secretary

MEMBERS OF CDFD GOVERNING COUNCIL

Dr. Renu Swarup Secretary, DBT, New Delhi	-	Chairperson
Director General CSIR, New Delhi	-	Member (Ex-officio)
Director General Bureau of Police Research and Development (BPR&D) Ministry of Home Affairs, New Delhi	-	Member (Ex-officio)
Prof Partha P Majumder Director, NIBMG, West Bengal Chairman of Scientific Advisory Committee, CDFD	-	Member (Ex-officio)
Mr. B Anand, IAS Addl. Secretary & FA, DBT, New Delhi	-	Member (Ex-officio)
Mr. CP Goyal Joint Secretary (Administration), DBT, New Delhi	-	Member (Ex-officio)
Joint Secretary (PM) Ministry of Home Affairs, New Delhi	-	Member (Ex-officio)
Joint Secretary & Legal Advisor Ministry of Law & Justice, New Delhi	-	Member (Ex-officio)
Dr. A K Rawat Director, DBT, New Delhi	-	Member (Ex-officio)
Prof V S Chauhan ICGEB, New Delhi	-	Member
Prof Dipankar Chatterji Indian Institute of Science (IISc), Bangalore	-	Member
Dr. Rakesh K Mishra Director, CCMB, Hyderabad	-	Member
Dr. Debashis Mitra Director, CDFD, Hyderabad Member-Secretary	-	Member

MEMBERS OF CDFD RESEARCH AREA PANELS – SCIENTIFIC ADVISORY COMMITTEE

Dr. Partha P Majumder NIBG, West Bengal	-	Chairman
Dr. Arun Kumar Rawat DBT, New Delhi (DBT Representative)	-	Member
Mr. K P S Kartha CFSL, Hyderabad (MHA Nominee)	-	Member
Dr. Manisha Madkaikar Natl Inst. of Immunohaematology, Mumbai (ICMR representative)	-	Member
Dr. K V Bhat NBPGR, Delhi (ICAR representative)	-	Member
Dr. Thangaraj CCMB, Hyderabad (CCMB representative)	-	Member
Prof. Sriram Ramaswamy IISc, Bangalore	-	Member
Prof. B K Thelma University of Delhi (South Campus), New Delhi	-	Member
Prof. Dr Seyed E Hasnain Jamia Hamdard, New Delhi	-	Member
Dr. Saman Habib CDRI, Lucknow	-	Member
Dr. Krishanu Ray TIFR, Mumbai	-	Member
Prof. Tapas Kundu JNCASR, Bangalore	-	Member
Dr. Anurag Agrawal IGIB, New Delhi	-	Member
Dr. Debasisa Mohanty NII, New Delhi	-	Member
Dr. Sankaranarayanan CCMB, Hyderabad	-	Member
Prof. Umesh Varshney IISc., Bangalore	-	Member
Dr. Jaya Sivaswami Tyagi AIIMS, New Delhi	-	Member
Dr. Usha Vijayraghavan IISc., Bangalore	-	Member
Dr. Shantanu Chowdhury IGIB, New Delhi	-	Member
Prof. V Nagaraja JNCASR, Bangalore	-	Member
Dr. Ranjan Sen Incharge-Director, CDFD, Hyderabad	-	Member Secretary

COMPOSITION OF FINANCE COMMITTEE

Prof. V S Chauhan , Visiting Scientist, International Centre for Genetic Engineering & Biotechnology (ICGEB), ICGEB Campus, Aruna Asaf Ali Marg, New delhi-110067	-	Chairman
Dr. Dipankar Chatterji , Hon. Professor Molecular Biophysics Unit, Indian Institute of science, Banglore-560012	-	Member
AS & FA , Dept. of Biotechnology, Ministry of Science & Technology, Block-2, 7 th Floor, CGO Complex, Lodi Road, New Delhi-110003	-	Member
Dr. A K Rawat , Director, Dept. of Biotechnology, Ministry of Science & Technology, Block-2, 6 th Floor, CGO Complex, Lodi Road, New Delhi-110003	-	Member
Shri A P Rao , FAO, CCMB, Hyderabad	-	Member
Dr. Debashis mitra , Director, CDFD, Hyderabad	-	Member
Mr E V Rao , Incharge F&A, CDFD, Hyderabad	-	Member Convener
Mr T Abhishek , Accounts Officer, CDFD, Hyderabad	-	Special Invitee

MEMBERS OF THE INSTITUTIONAL BIO-SAFETY COMMITTEE (IBSC)

Dr. Sangita Mukhopadhyay Staff Scientist - VI, CDFD	-	Chairperson
Dr. Arvind Kumar Principal Scientist, CCMB	-	DBT Nominee
Dr. Rashna Bhandari Staff Scientist – V, CDFD	-	Member Secretary
Dr. Krishnaveni Mishra Asso. Professor	-	Outside Expert Department of Biochemistry, SLS, University of Hyderabad,
Dr. Ashwin B Dalal Staff Scientist – VI, CDFD	-	Biosafety Officer
Dr. M D Bashyam Staff Scientist – VI, CDFD	-	Internal Expert
Dr. Sanjeev Khosla Staff Scientist – VI, CDFD	-	Internal Expert
Dr. Rupinder Kaur Staff Scientist – VI, CDFD	-	Internal Expert

MEMBERS OF SEXUAL HARASSMENT COMPLAINTS COMMITTEE

Dr. Sangita Mukhopadhyay SS – VI	-	Chairperson
Dr. Rupinder Kaur Staff Scientist – VI	-	Member
Mr. J Sanjeev Rao Head – Administration	-	Member
Ms. V Naga Sailaja TO – II	-	Member
Ms. M V Sukanya TO – II	-	Member
Mr. M S A Zaman Khan Section Officer	-	Member
Ms. P Jamuna Gramya Resource Centre for Women (Representing as NGO)	-	Member

MEMBERS OF INSTITUTIONAL BIO-ETHICS COMMITTEE

Prof. G B Reddy University College of Law, OU, Hyderabad	-	Chairperson
Prof. Sheela Prasad Associate Professor, Centre for Regional Studies, School of Social Sciences, University of Hyderabad	-	Member
Dr. Mahtab S Bamji Emeritus Scientist Dangoria Charitable Trust, Hyderabad	-	Member
Dr. Amita Kasbekar VP, Deloitte Consulting India Pvt. Ltd., RMZ, Hitech City, Hyderabad	-	Member
Dr. M D Bashyam Staff Scientist – VI, CDFD	-	Member
Dr. Sanjeev Khosla Staff Scientist – VI, CDFD	-	Member
Dr. Ashwin B Dalal Staff Scientist – VI, CDFD	-	Member Secretary

MEMBERS OF CDFD BUILDING COMMITTEE

Prof VS Chauhan JC Bose Fellow (DST), Distinguished Biotechnology, Research Professor & Chairman, UGC, New Delhi.	-	Chairman
Joint Secretary (Admin.) DBT, New Delhi	-	Member
Dr. Debashis Mitra Director, CDFD, Hyderabad	-	Member
Mr. J Sanjeev Rao Head-Administration, CDFD, Hyderabad	-	Member
Mr. E.V. Rao Account Officer, CDFD, Hyderabad	-	Member
Mr. V.H. Rao Ex-Sr.Consultant, NIAB, Hyderabad	-	Member
Mr. Raghavendrachar Jois In-charge Engineering, CDFD, Hyderabad	-	Member-Convenor

MEMBERS OF CDFD MANAGEMENT COMMITTEE

Director CDFD, Hyderabad	-	Chairman
Dr. Ranjan Sen SS – VII	-	Member
Dr. Sangita Mukhopadhyay SS – VI	-	Member (for a 2 years period)
Dr. Abhijit A Sardesai SS – IV	-	Member (for a 2 years period)
I/c – Finance & Accounts	-	Member
Head – Administration	-	Member – Convenor

सूचना अधिकार अधिनियम, 2005 का परिपालन
Implementation of RTI Act, 2005

IMPLEMENTATION OF RIGHT TO INFORMATION (RTI) ACT, 2005

Appellate Authority : Dr M D Bashyam

Central Public Information Officer : Ms Varsha

Details about the RTI applications and appeals received in CDFD

As received under RTI Act 2005	Opening Balance as on 1.4.2017	Received during the year 2017-18			Disposed of during the year 2017-18			Closing Balance as on 31.3.2018
		Received directly	Received as transfer from other Public Authorities [u/s 6(3) of Act]	Total	Decisions where applications accepted/ appeals upheld	Decisions where applications accepted/ appeals rejected	Transferred to other Public Authorities [U/s 6(3) of Act]	
Applications	7	22	10	39	33	1	0	34
Appeals	0	02	Not applicable	02	02	Nil	Not applicable	2
								5+
								Nil

3 applications were not disposed for receipt of additional payment. RA-31, RA-42, and RA-47.

बजट एवं वित्त
Budget and Finance

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS HYDERABAD

Budget & Finance 2017-18

Sources of Funds

The Financial resources of the Centre are the Core Plan Grant-in Aid provided by the Department of Biotechnology, Government of India as against Annual Budgetary projections made by the Institute. Other resources are in the form of Research Grants provided by various National and International agencies and also from Services rendered by CDFD. The components of the core grants are Plan (Recurring) essentially for meeting expenditures on salaries, Operating expenses etc., and Plan (Non-Recurring) for meeting expenses on account of Equipments, Infrastructure and Furnishing etc.,

Receipts during the year 2017-18

Particulars	Amount in Lakhs	Percentage - %
Plan Grant in Aid	4390.00	78.71
Sponsored Projects	863.26	15.48
CDFD Services	60.19	1.08
Misc Receipts	263.43	4.73
Total	5576.88	100.00

I. Application of Funds during 2017-18 (Plan Grant in Aid)

S No	Particulars	Amount in Lakhs	Percentage- %
1	Recurring		
	GIA- Salaries	1536.84	34.48
	GIA-General	1869.00	41.93
	Total	3405.84	76.41
2	Non-Recurring		
	GIA- Capital	1051.77	23.59
	Total	1051.77	23.59
	Grand Total	4457.61	100.00

II. Application of Funds during 2017-18 (Extra Mural Projects)

S No	Particulars	Amount in Lakhs	Percentage- %
1	Recurring		
	Salaries	270.61	36.87
	General	395.51	53.89
	Total	666.12	90.76
2	Non-Recurring		
	Capital	67.86	9.24
	Total	67.86	9.24
	Grand Total	733.98	100.00

लेखा परिक्षक की रिपोर्ट
Auditor's Report

B Purushottam & Co

Chartered Accountants

AUDITOR'S REPORT

Date: 10-10-2018

The Director,
Centre for DNA Fingerprinting and Diagnostics,
Uppal, Hyderabad – 500 039

We have audited the attached Balance Sheet of **CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS**, Hyderabad, as at 31st March 2018 and also the Income & Expenditure Account for the year ended on that date annexed there to. These Financial Statements are the responsibility of the organization management. Our responsibility is to express an opinion on these Financial Statements based on our audit.

We report that:

1. We have obtained all the information and explanations, which to the best of our knowledge and belief were necessary for the purpose of our audit.
2. In our opinion, the organization has kept proper books of account as required by law so far, as appears from our examination of these books.
3. The Balance Sheet and Income & Expenditure account dealt with by this report is in agreement with the books of account.
4. (a) The centre has maintained accounts on accrual basis.
(b) The Centre receives extra mural grants from various National & International agencies for specific research activities. The Centre has a policy of allocating the overheads and transfer of expenditure of CDFD to different projects at the end of the financial year after taking into account the amount of maximum permissible limit of overheads and also based on the approved budget estimates and expenditure of the respective projects during the financial year.
5. In our opinion and to the best of our information and according to the explanations given to us, the said Balance Sheet and the Income & Expenditure account read together with the notes thereon gives the required information in the manner so required and gives a true and fair view.
 - a) In so far it relates to the Balance Sheet as at 31st March 2018 and
 - b) In so far as it relates to the Income & Expenditure account excess of income over expenditure for the year ended on 31st March 2018.

for **B Purushottam & Co.,**
Chartered Accountants
Reg. No.002808S

[CH SATYANARAYANA]
M.No.19092

Place: Hyderabad

Date: 10/10/2018

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD				
INCOME & EXPENDITURE FOR THE YEAR ENDED 31st MARCH 2018				
INCOME	Schedule	Current Year	Previous Year	(Amount - Rs.)
Income from Sales/Services	12	6019186	7071528	
Grants/Subsides	13	379000000	300000000	
Fees/Subscriptions	14	0	0	
Income from Investments	15	24311928	5685649	
Income from Royalty, Publications etc.	16	0	0	
Interest Earned	17	1265453	1785882	
Other Income	18	891583	4788491	
Increase/(decrease) in stock of Finished goods and works-in-progress	19	0	0	
TOTAL (A)		411488150	319331550	
EXPENDITURE				
Establishment Expenses	20	145105060	122420108	
Administrative Expenses	21	162653452	164271394	
Expenditure on Grants, Subsides etc.	22	0	0	
Interest	23	0	0	
Depreciation (Net Total at the year-end -corresponding to Schedule 8)		60247615	67006639	
Less: Transferred to Grants-in-Aid		60247615	67006639	
Provision For Salaries		7668375	8264377	
TOTAL (B)		315426887	294955879	

Balance being excess of Income over Expenditure (A-B)				96061263		24375671
Transfer to Special Reserve (Specify each)						
Transfer to/from General Reserve				6019186		9506144
BALANCE BEING SURPLUS/(DEFLECT) CARRIED TO CORPUS/CAPITAL FUND	24			90042077		14869527
SIGNIFICANT ACCOUNTING POLICIES	25					
CONTINGENT LIABILITIES AND NOTES ON ACCOUNTS						
DIRECTOR CDFD	For B.PURUSHOTTAM & CO. CHARTERED ACCOUNTANTS (B.PURUSHOTTAM)			I/c - FINANCE & ACCOUNTS CDFD		

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD				
RECEIPTS AND PAYMENTS ACCOUNT FOR THE YEAR ENDED 31st MARCH 2018				
				(Amount - Rs.)
RECEIPTS	Current Year	Previous Year	PAYMENTS	Current Year
1. Opening Balances				
a) Cash in hand	0	0	1. Expenses	
b) Bank Balances			a) Establishment Expenses (corresponding to Schedule 20)	153369437
i) In current accounts	17665452	27660890	b) Administrative Expenses (corresponding to Schedule 21)	176041112
ii) In deposit accounts	291098273	0	c) Schedule 22	0
iii) Savings accounts	9699923	11145109		
2. Grants Received				
a) From Government of India	439000000	600000000	2. Payments made against funds for various projects	
b) From State government			(Name of the fund or project should be shown along with the particulars of payments made for each project)	
c) From other sources (details)			Projects (Annexure F)	73398198
(Grants for capital & revenue exp. To be shown separately)			CSIR(Stipend)	5641420
Research Associates - DBT(Stipend)	1233187	6712089	DBT(Stipend)	5836035
Research Associates - DST(Stipend)	186895	9882757	DST(Stipend)	1176895
Research Associates - ICMR(Stipend)	1678583	1922748	ICMR(Stipend)	1761842
Research Associates - IISC(Stipend)	1440426	106012	IISC(Stipend)	708543
Research Associates - UGC(Stipend)	1742857	4127705	IISC(Stipend)	2033572
3. Investments and deposits made				
Projects (Annexure - C)	86416491	90196329	a) Out of Earmarked/Endowment funds	0
			b) Out of Own Funds (Investments-Others)	0
3. Income on Investments from				
a) Earmarked/Endow. Funds	24300554	5504248	4. Expenditure on Fixed Assets & Capital Work-in-Progress	
DIRECTOR				
For B.PURUSHOTTAM & CO.				
CHARTERED ACCOUNTANTS				
(B.PURUSHOTTAM)				
I/c - FINANCE & ACCOUNTS				
CDFD				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
RECEIPTS AND PAYMENTS ACCOUNT FOR THE YEAR ENDED 31st MARCH 2018					
					(Amount - Rs.)
RECEIPTS	Current Year	Previous Year	PAYMENTS	Current Year	Previous Year
Advance/Refunds/Recovery/Adj.(Annexure-B)	108026153	73435613	i) In current accounts	56770667	17665452
NIMS	2582360	6107113	ii) In deposit accounts	311098273	0
			iii) Savings accounts	15058466	9699923
TOTAL	1074511850	1424186319	TOTAL	1074511850	1424186319
DIRECTOR	For B.PURUSHOTTAM & CO.				
CDFD	CHARTERED ACCOUNTANTS				
	(B.PURUSHOTTAM)				
	I/c - FINANCE & ACCOUNTS				
	CDFD				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		(Amount - Rs.)	
BALANCE SHEET AS ON 31st MARCH 2018			
	Current Year		Previous Year
SCHEDULE 1 - CORPUS/CAPITAL FUND :			
Balance as at the beginning of the year		1942028103.00	1686691192.00
Add : Contribution towards Corpus/Capital Fund	60000000.00	300000000.00	
CDFD Core - Plan (Non-Recurring)	6785660.00	7474023.00	307474023.00
Capitalised portion of Capital Expenditure of projects			
Less : Depreciation For the Year 2016-2017	60247615.00	60247615.00	67006639.00
Add : Excess of Expenditure over Income	90042077.00	90042077.00	14869527.00
BALANCE AS AT THE YEAR - END		2038608225.00	1942028103.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS			(Amount - Rs.)	
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018			Current Year	Previous Year
SCHEDULE 2 -RESERVES AND SURPLUS :				
1.Capital Reserve :				
As per last Account	0.00		0.00	
Addition during the year	0.00		0.00	
Less : Deductions during the year	0.00	0.00	0.00	0.00
2.Revolution Reserve :				
As per last Account	0.00		0.00	
Addition during the year	0.00		0.00	
Less : Deductions during the year	0.00	0.00	0.00	0.00
3.Special Reserves :				
As per last Account	0.00		0.00	
Addition during the year	0.00		0.00	
Less : Deductions during the year	0.00	0.00	0.00	0.00
4.General Reserve :				
As per last Account	25990202.00		16484058.00	
Addition during the year	6019186.00		9506144.00	0.00
Less : Deductions during the year	0.00	32009388.00	0.00	25990202.00
Total		32009388.00		25990202.00

DNA Fingerprinting Receipts	3674199
Diagnosics Receipts	1910400
Sophisticated Equipment Facility Receipts	434587
Total Receipts	6019186

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS			SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		(Amount - Rs.)
	Current Year		Previous Year		
SCHEDULE 3 - EARMARKED/ENDOWMENT FUNDS :					
(Refer Annexures)					
(a) Opening balance of the Funds					
(b) Additions to the Funds :					
i. Donations /grants	86326089.66	5912597.03	90196329.00	-18029485.84	
ii. Income from investments made on account of funds	0.00		0.00		
iii. Other additions	0.00	86326089.66	0.00	90196329.00	
TOTAL (a+b)		92238686.69		72166843.16	
(c) Utilisation/Expenditure towards objective of funds					
(i) Capital Expenditure (Refer Annexures I & II)					
- Fixed Assets	6628487.00		7474023.00		
- Others	157173.00	6785660.00	0.00	7474023.00	
- Total					
(ii) Revenue Expenditure (Refer Annexures I & II)					
- Salaries, Wages and allowances etc.	27061925.00		29848272.00		
- Rent	0.00		0.00		
- Other Expenses	39550613.00	66612538.00	28931951.13	58780223.13	
Total					
TOTAL (c)		73398198.00		66254246.13	
NET BALANCE AS AT THE YEAR-END [(a + b)-c]		18840488.69		5912597.03	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS			(Amount - Rs.)	
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018			Current Year	Previous Year
SCHEDULE 4 - SCHEDULE LOANS AND BORROWINGS :				
1. Central Government				
		0		0
2. State Government (Specify)				
		0		0
3. Financial Institutions				
	a) Term Loans	0	0	
	b) Interest accrued and due	0	0	0
4. Banks :				
	a) Terms Loans	0	0	0
	- Interest accrued and due	0	0	0
	b) Other Loans	0	0	0
	- Interest accrued and due	0	0	0
5. Other Institutions and Agencies				
		0		0
6. Debentures and Bonds				
		0		0
7. Others (Specify)				
TOTAL			0	0
Note: Amount due within one year				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS			SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		(Amount - Rs.)
			Current Year		Previous Year
SCHEDULE 5 - UNSECURED LOANS AND BORROWINGS :					
1. Central Government			0		0
2. State Government (Specify)			0		0
3. Financial Institutions			0		0
4. Banks :					
a) Terms Loans			0	0	
b) Other Loans			0	0	0
5. Other Institutions and Agencies					0
6. Debentures and Bonds			0		0
7. Fixed Deposits			0		0
8. Others (Specify)			0		0
TOTAL			0		0
Note: Amount due within one year					

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		(Amount - Rs.)
		Current Year		Previous Year
SCHEDULE 6 - DEFERRED CREDIT LIABILITIES :				
a) Acceptances secured by hypothecation of capital equipment and other assets		0		0
b) Others		0		0
TOTAL		0		0
Note: Amount due within one year				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018

(Amount - Rs.)

	Current Year		Previous Year
SCHEDULE 7 - CURRENT LIABILITIES AND PROVISIONS :			
A. CURRENT LIABILITIES			
1. Acceptances		926161.00	910797.00
2. Sundry Creditors		24325.00	502477.00
3. Advances Received		1520314.00	1559790.00
4. Interest accrued but not due on:		1680631.00	360230.00
5. Statutory Liabilities:			
Income Tax			
Service Tax			
TDS			
Works Tax			
6. Other current Liabilities			
CDFD.CP Fund A/C(Annexure-G)		52520328.00	43287242.00
Contract Staff security deposit		125594.00	622172.00
Diagnostics Collaboration With NIMS		0.00	5260668.00
ECCS		0.00	163285.00
EMD		2077382.00	2303652.00
Festival Advance		450.00	0.00
GSLI		31526.00	24616.00
House Building Advance		129831.00	129831.00
Lab Security Deposit & Hostel Security Deposit		1346016.00	1294396.00
LIC		2550.00	2550.00
Others (I-Remittances)		0.00	487642.00
Out Standing Liabilities		11845456.00	11845456.00
Professional Tax		94342.00	96592.00
Public Provident Fund		391158.00	391158.00
Royalty & Consultancy		1531642.00	1531642.00
Security Deposit		5496040.00	2547185.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS			SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		(Amount - Rs.)
	Current Year		Previous Year		
SCHEDULE 7 - CURRENT LIABILITIES AND PROVISIONS :					
STAFF BENEVOLENT FUND					
TA Abroad [Advance]	42673.00		12569.00		
TA-DA-Hon within India [Advance]	79909.00	79866328.00	109576.00	65909.00	73509435.00
TOTAL (A)		79866328.00			73509435.00
B.PROVISIONS					
1. For Taxation	0.00		0.00		
2. Gratuity	0.00		0.00		
3. Superannuation/Pension	0.00		0.00		
4. Accumulated Leave Encashment	0.00		0.00		
5. Trade Warranties/Claims	0.00		0.00		
6. Others (Specify)	0.00		0.00		
TOTAL (B)		7668375.00			8264377.00
TOTAL (A+B)		87534703.00			81773812.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018

(Amount - Rs.)

	GROSS BLOCK						DEPRECIATION						NET BLOCK			
	Cost/valuation at beginning of the year	Addition during the year	Deductions during the year	Cost/valuation at the year end	As at the beginning the year	On additions during the year	On Deductions during the year	Total up to the year end	As at the current year end	As at the previous year end						
A. FIXED ASSETS:																
1. LAND:																
a) Freehold	3900000.00	0.00	0.00	3900000.00	0.00	0.00	0.00	0.00	3900000.00	3900000.00	0.00	0.00	0.00	3900000.00	0.00	0.00
b) Leasehold	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
2. BUILDINGS																
a) On Freehold Land	220052369.00	0.00	0.00	220052369.00	100930732.00	11912164.00	0.00	112842896.00	107209473.00	119121637.00	0.00	0.00	0.00	119121637.00	0.00	0.00
b) On Leasehold Land	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
c) Ownership Flats/Premises	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
d) Superstructures on Land not belongs to the entity	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
3. PLANT MACHINERY & EQUIPMENT	729171242.05	12866888.00	0.00	742038130.05	442561681.00	46545898.00	0.00	489107579.00	252930551.05	286609561.05	0.00	0.00	0.00	286609561.05	0.00	0.00
4. VEHICLES	4153026.00	0.00	0.00	4153026.00	3745076.00	61193.00	0.00	3806269.00	346757.00	407950.00	0.00	0.00	0.00	407950.00	0.00	0.00
5. FURNITURE, FIXTURES	16044396.00	4736.00	0.00	16049132.00	11815277.00	401304.00	0.00	12216581.00	3832551.00	4229119.00	0.00	0.00	0.00	4229119.00	0.00	0.00
6. OFFICE EQUIPMENT	12149882.00	5000.00	0.00	12154882.00	10004841.00	364128.00	0.00	10368969.00	1785913.00	2145041.00	0.00	0.00	0.00	2145041.00	0.00	0.00
7. COMPUTER/PERIPHERALS	132023.00	134000.00	0.00	266023.00	0.00	52809.00	0.00	52809.00	213214.00	132023.00	0.00	0.00	0.00	132023.00	0.00	0.00
8. ELECTRIC INSTALLATIONS	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
9. LIBRARY BOOKS	19585964.00	750947.00	0.00	20336911.00	19255158.00	840549.00	0.00	20095707.00	241204.00	330806.00	0.00	0.00	0.00	330806.00	0.00	0.00
10. TUBEWELLS & WATER SUPPLY	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
11. OTHER FIXED ASSETS	8857898.00	30000.00	0.00	8887898.00	8162190.00	69570.00	0.00	8231760.00	656138.00	695708.00	0.00	0.00	0.00	695708.00	0.00	0.00
Airconditioning works		0.00	0.00		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Aluminium partition work		0.00	0.00		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
DG Set		0.00	0.00		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Paintings		0.00	0.00		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Typewriters		0.00	0.00		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Miscellaneous non consumables		0.00	0.00		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Other Assets		0.00	0.00		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
EMB Net		0.00	0.00		0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
TOTAL	1014046800.05	13791571.00	0.00	1027838371.05	596474955.00	60247615.00	0.00	656722570.00	371115801.05	417571845.05	0.00	0.00	0.00	417571845.05	0.00	0.00
B. CAPITAL WORK-IN-PROGRESS	1168693555.70	46703759.00	0.00	1215397314.70	0.00	0.00	0.00	0.00	1215397314.70	1168693555.70	0.00	0.00	0.00	1168693555.70	0.00	0.00
TOTAL	2182740355.75	60495330.00	0.00	2243235685.75	596474955.00	60247615.00	0.00	656722570.00	1586513115.75	1586265400.75	0.00	0.00	0.00	1586265400.75	0.00	0.00

Equipment - Core	6238401
Equipment - Projects	6628487
Total	12866888

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 9 - INVESTMENTS FROM EARMARKED/ENDOWMENT FUNDS :		
1. In Government Securities	0.00	0.00
2. Other approved securities	0.00	0.00
3. Shares	0.00	0.00
4. Debentures and Bonds	0.00	0.00
5. Subsidiaries and Joint Ventures	0.00	0.00
6. Others (to be specified) - STDRs (Annexure-J)	0.00	0.00
TOTAL	0.00	0.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 10 - INVESTMENTS - OTHERS :		
(Annexure-K)		
1. In Government Securities	0.00	0.00
2. Other approved securities	0.00	0.00
3. Shares	0.00	0.00
4. Debentures and Bonds : UTI Bonds		
5. Subsidiaries and Joint Ventures	0.00	0.00
6. Others (to be specified) - STDRs,(CPF),CDFD CP FUND A/C	23387695.00	31870241.00
TOTAL	23387695.00	31870241.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018

(Amount - Rs.)

	Current Year		Previous Year	
	Current Year	Current Year	Previous Year	Previous Year
SCHEDULE 11 - INVESTMENTS - OTHERS :				
A. CURRENT ASSETS				
1. Inventors				
a) Stores and Spares	0.00		0.00	
b) Loose Tools	0.00		0.00	
c) Stock-in-trade				
Finished Goods	0.00		0.00	
Work-in-progress	0.00		0.00	
Raw Materials	0.00	0.00	0.00	0.00
2. Sundry Debtors:				
a) Debts Outstanding for a period exceeding six months	0.00		0.00	
b) Others-Life Membership Fees	169236.00	169236.00	169236.00	169236.00
3. Cash balances in hand (including cheques/drafts and imprest)				
4. Bank Balances:				
a) With Scheduled Banks:				
-On Current Accounts	56770666.75		17665451.85	
-On Deposit Accounts (includes margin money)	311098273.00		291098273.00	
-On Savings Accounts	15058466.30	382927406.05	9699922.91	318463647.76
b) With non-Schedules Banks:				
-On Current Accounts	0.00		0.00	
-On Deposit Accounts	0.00		0.00	
-On Savings Accounts	0.00	0.00	0.00	0.00
5. Post Office-Savings Accounts				
TOTAL (A)		383096642.05		318632883.76

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS			(Amount - Rs.)
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018			
	Current Year		Previous Year
SCHEDULE 11 - INVESTMENTS - OTHERS :			
B. LOANS, ADVANCES AND OTHER ASSETS			
1. Loans:			
a) Staff (Annexure-L)	632508.00	0.00	0.00
b) Other Entities engaged in activities/objectives similar to that of the Entity	0.00	632508.00	0.00
2. Advances and other amounts recoverable in cash or in kind or for value to be received			
a) On Capital Account (Annexure-H)	72726723.00	86818537.00	
b) Prepayments - Deposits (Annexure-I)	14528354.00	16472947.00	
c) TDS Receivable	486429.00	437792.00	
d) Others (Annexure-K)	81513863.00	0.00	
e) GST on Purchases (Schedule 21B)	14107476.00	0.00	103729276.00
3. Income Accrued:			
a) On Investments from Earmarked/Endowments Funds	0.00	0.00	
b) On Investments - Others	0.00	15206912.00	
c) On Loans and Advances	0.00	0.00	
d) Others	0.00	0.00	15206912.00
4. Claims Receivable		0.00	0.00
TOTAL (B)		183995353.00	118936188.00
TOTAL (A+B)		567091995.05	437569071.76

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 12 - INCOME FROM SALES/SERVICES :		
1) Income from sales		
a) Sale of Finished Goods	0.00	0.00
b) Sale of Raw Material	0.00	0.00
c) Sale of Scraps	0.00	0.00
2) Income from Services		
a) Labour and Processing Charges	0.00	0.00
b) Professional/Consultancy Services (Analysis Charges)	6019186.00	7071528.00
c) Agency Commission and Brokerage	0.00	0.00
d) Maintenance Services (Equipment/Property)	0.00	0.00
e) Others (Specify)	0.00	0.00
TOTAL	6019186.00	7071528.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 13 - GRANTS/SUBSIDIES :		
(Irrevocable Grants & Subsidies Received)		
1) Central Government (DBT Plan Grant-in-Aid)	379000000.00	300000000.00
2) State Government(s)	0.00	0.00
3) Government Agencies	0.00	0.00
4) Institutions/Welfare Bodies	0.00	0.00
5) International Organisations	0.00	0.00
6) Others (Specify)	0.00	0.00
TOTAL	379000000.00	300000000.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
(Amount - Rs.)		
SCHEDULE 14 - FEES/SUBSCRIPTIONS :		
1) Entrance Fees	0	0
2) Annual Fees/Subscriptions	0	0
3) Seminar/Program Fees	0	0
4) Consultancy Fees	0	0
5) Others (Specify)	0	0
TOTAL	0	0

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
(Amount - Rs.)		
SCHEDULE 15 - INCOME FROM INVESTMENTS :		
(Income on Invest from Earmarked/Endowment Funds transferred to Funds)		
1) Interest:		
a) On Govt. Securities	0.00	0.00
b) Other Bonds/Debentures	0.00	0.00
2) Dividends:		
a) On Shares	0.00	0.00
b) On Mutual Fund Securities	0.00	0.00
3) Rents	0.00	0.00
4) Others (Specify) STDRs	24311928.00	5504248.00
TOTAL	24311928.00	5504248.00
TRANSFERRED TO EARMARKED/ENDOWMENT FUNDS		

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 16 - INCOME FROM ROYALTY, PUBLICATION ETC. :		
1) Income from Royalty	0	0
2) Income from Publications	0	0
3) Others (Specify)	0	0
TOTAL	0	0

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 17 - INTEREST EARNED :		
1) On Term Deposits		
a) With Schedule Banks	0.00	1278536.00
b) With Non-Scheduled Banks	0.00	0.00
c) With Institutions	0.00	0.00
d) Others	0.00	0.00
2) On Saving Accounts		
a) With Schedule Banks	1265453.00	507346.00
b) With Non-Scheduled Banks	0.00	0.00
c) post Office Savings Accounts	0.00	0.00
d) Others	0.00	0.00
3) On Loans		
a) Employees/Staff	0.00	0.00
b) Others	0.00	0.00
4) Interest on Debtors and Other Receivables		
TOTAL	1265453.00	1785882.00
Note :- Tax deducted at source to be indicated		

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 18 - OTHER INCOME		
1) Profit on Sale/disposal of Assets:		
a) Owned assets	0.00	0.00
b) Assets acquired out of grants, or received free of cost	0.00	0.00
2) Export Incentives realized	0.00	0.00
3) Fees for Miscellaneous Services	0.00	0.00
4) Miscellaneous Receipts	0.00	0.00
5) Other Receipts		
Sundry Receipts	700519.00	4568979.00
Application Fee	750.00	15125.00
Sales Of Tender Forms	63500.00	90500.00
Licence Fee	51680.00	54880.00
Interest On Computer Advance, Conveyance Advance And HBA	22298.00	6171.00
Leave Salary-Pension Contribution	52836.00	52836.00
TOTAL	891583.00	4788491.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 19 - INCREASE/(DECREASE) IN STOCK OF FINISHED GOODS & WORK IN PROGRESS :		
a) Closing stock		
-Finished Goods	0	0
-Work-in-progress	0	0
Total (a)	0	0
b) Less: Opening stock		
-Finished Goods	0	0
-Work-in-progress	0	0
Total (b)	0	0
NET INCREASE/(DECREASE) [a-b]	0	0

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 20 - ESTABLISHMENT EXPENSES :		
a) Salaries and Wages	99673311.00	45425480.00
b) Allowances and Bonus	36170434.00	62477804.00
c) Contribution to Provident Fund	8860627.00	4407988.00
d) Contribution to Other Fund (NPS)	4151325.00	3162884.00
e) Staff Welfare Expenses - Medical charges	3090035.00	2219993.00
f) Expenses on Employees Retirement and Terminal Benefits	1318666.00	4725959.00
g) Others (specify) - Staff leased House	0.00	0.00
h) EPF Employer Contribution	105039.00	0.00
TOTAL	153369437.00	122420108.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 21 - OTHER ADMINISTRATIVE EXPENSES :		
1) Purchases	25100098.00	33249114.00
2) Electricity and power	22233159.00	22793626.00
3) Water charges	1101091.00	1662990.00
4) Insurance	125076.00	97432.00
5) Repairs and maintenance	15144297.00	16694133.00
6) Rent, Rates and Taxes	15662184.00	21280489.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 21 - OTHER ADMINISTRATIVE EXPENSES :		
7) Vehicles Running and Maintenance	2339712.00	1386497.00
8) Postage, Telephone and Communication Charges	2751048.00	2229539.00
9) Printing and Stationary	1933758.00	1344515.00
10) Travelling and Conveyance Expenses	6339244.00	5982640.38
11) Expenses on Seminar/Workshops	385663.00	78900.00
12) Subscription Expenses	397837.00	54500.00
13) Expenses on Fees	17771.00	94777.00
14) Auditors Remuneration	67260.00	39500.00
15) Hospitality Expenses	456293.00	813197.00
16) Professional Charges	1232188.00	1389456.00
17) Advertisement and Publicity	881572.00	1779225.00
18) Bank Charges	13318.61	5297.00
19) Security & Cleaning Contract Charges	26481982.00	24811357.00
20) Training Course /Symposia	9000.00	9600.00
21) Other Contingencies	4456541.00	5202138.00
22) Liveries & Blankets	7000.00	0.00
23) Other Research Expenses	15734404.00	23260806.00
24)Office Books	1708.00	11666.00
25)Over Heads	0.00	0.00
26)Contract Staff	9314458.00	0.00
27)Manpower Outsourcing(Staff)	10466789.00	0.00
TOTAL	162653451.61	164271394.38

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 22 - EXPENDITURE ON GRANTS, SUBSIDIES, ETC.		
a) Grants given to Institutions/Organisations	0.00	0.00
b) Subsidies given to Institutions/Organisations	0.00	0.00
TOTAL	0.00	0.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS		
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2018		
	Current Year	Previous Year
	(Amount - Rs.)	
SCHEDULE 23 - INTEREST		
a) On Fixed Loans	0.00	0.00
b) On Other Loans (including Bank Charges)	0.00	0.00
c) Others	0.00	0.00
TOTAL	0.00	0.00

**Schedule 24: Significant Accounting Policies & Schedule
25: Contingent Liabilities & Notes on Account
for the period ended 31/03/2018**

1. Method of Accounting:

- a. The accounting system adopted by the organization is on “accrual basis”.
- b. The organization has been getting plan Grant-In-Aid under the “Non-recurring” & “Recurring” heads.

2. Revenue recognition:

Income comprises of Grant-in-Aid, Internal Resources through services and interest from short term deposits. Income accounted on the basis of the Cash/DD/Cheques/Cr notes/ on line transfers received.

3. Fixed Assets:

- (a) Fixed assets are stated at cost. Cost includes freight, duties, and taxes etc.,
- (b) Depreciation: Depreciation Account on Fixed Assets has since been prepared at the rate prevailing to the concerned Fixed Assets as specified in the Income Tax Act, 1961 on Written Down Value Method of Depreciation.
- (c) Capital work in progress has been entered to the extent of the last running account bills paid.
- (d) Realization on sale of obsolete/surplus fixed assets which is not required for the purpose of research activities are adjusted against capital cost.

4. Inventories:

All purchases of chemicals, glassware and other consumables have been charged to consumption at the time of purchase.

5. Foreign Currency transactions:

Foreign Currency transactions are recognized in the books at the exchange rates prevailing on the date of transaction.

6. Investments:

Investments in STDR's are stated at book values.

7. Advances

It is observed from the objection book register that advances to suppliers for consumables & Equipments are to be reconciled and adjustment entries are to be passed in the books of accounts.

8. The previous year balances have been regrouped/rearranged, wherever necessary.

Director CDFD

I/c- Finance & Accounts
CDFD

for B Purushottam & Co
Chartered Accountants
Reg.No.002808S

[CH SATYANARAYANA]

M.No.019092

Place: Hyderabad

Date: 10/10/2018

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

CLARIFICATION ON NOTES ON ACCOUNTS: 2017-18

- ❖ **Notes on Accounts 1 to 2 & 4 to 6: Method of Accounting/ Revenue recognition/Fixed Asset/Inventories/ Foreign Currency transactions/Investments:**

These are all only informatory items.

- ❖ **Notes on Accounts 3: Fixed Assets:**

Depreciation has been calculated on Written down Value method and at the rates prevailing to the concerned Fixed Asset as specified on the Income Tax Act, 1961 and set off against the Grant-in-Aid (non-recurring). The details of the Depreciation on Fixed Assets are at Schedule -8 is an integral part of the financial statements

- ❖ **Notes on Accounts 7: Advances:**

The observation of the audit has been noted. The action has already been initiated to reconcile the objection book register.

E V Rao

I/C Finance & Accounts

CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

Annexure-I

Details of Closing balances of various Earmarked / Endowment Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2018

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
-9650327	COE1	COE1	-10150735
-23954089	COE2	COE2	-19673739
2028298	others	"Fellowship / others "	2450797
-630047	P-03	"Transgenesis and Genetic basis of Pathogen Resistance in the Silkworm, Bombyx Mori	-630047
244305	P-09	"NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics"	244305
-28332	P-10	"Role of upstream sequence elements in Hyper activation of transcription from Baculovirus polyhedrin gene promoter"	-28332
-576590	P-100	Effect of reactive oxygen species on T-Cell immune response: An approach to understand the molecular mechanism of immunosuppression during tuberculosis - National Bioscience Award	-576590
-27922	P-102	Understanding the role of Mycobacterium tuberculosis heat shockprotein 60 as Th1/Th2 immuno modular	-27922
-300000	P-103	National Bioscience Award - Regulation of mast cell signaling, apoptosis and surface receptors	-300000
-1289897	P-104	Virtual Centre of Excellence on Epigenetics	-1289897
-862685	P-105	Cloning, Characterization and analysis of chromosomal rearrangements in human genetic disorders	-862685
327575	P-107	IYBA Project - Mechanism and role of bacterial cell-cell signaling molecules in plant defense response	327575
-454643	P-108	Establishment of EBV transformed cell lines from families with rare genetic disorders	-454643
-362393	P-109	Molecular dissection of PI3-Kinase/Akt pathway by using proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors	-1228422
-19391	P-110	India-Japan research project title"Identification and analysis of sex determining genes in silkmoths"	-19391
-450859	P-114	Evaluating the Calcineurin-NFAT Pathway and its regulators superoxide dismutase (SOD) AND RCAN1 (regulator of Calcineurin) Down Syndrome	-450859
-1251366	P-116	DBT-India and AIST - Japan : Understanding molecular mechanisms controlling dual role of Ras, Sirtuins and CARF in relation to cellular proliferation and senescence: Novel Strategy for developing cancer therapeutics	-1251366
-2892	P-119	Analysis of DNA copy number alterations in esophageal cancer	-2892
-769484	P-120	Effect of reactive oxygen species on macrophage signalosome: impact on antigen presentation functions and T Cell priming responses	-769484
-1130866	P-121	Identification and characterization of PTEN regulators	-1130866
21124	P-122	Understanding the role of Hox genes in anterior-posterior axis determination of the central nervous system	0
1440687	P-123	Establish a Max Planck Partner Group for Genetic Diversity Studies at CDFD	1112558
-748411	P-124	Preparation and characterization of peroxometal compounds and studies and their biological significance in cellular signalling	-748411
160270	P-126	Rho-dependent transcription termination machinery: mechanism of action	160270
-158488	P-128	Mechanism of iron acquisition and iron homeostasis in an opportunistic human pathogen Candida glabrata	-158488
3947	P-13	"Programme to delineate gene functions in the post – genomics era by a systematic two gene knockout method"	3947
-142258	P-130	Comparative genetic analysis of sex chromosomes and sex determining genes in silkmoths	-142258
398632	P-131	Structural and functional studies of Acyl CoA Binding proteins from plasmodium falciparum	398632
-12199	P-132	Characterization of tumor suppressor function of ARID1B, a component of the human SWI/SNF chromatin remodelling complex	-12199
-1324223	P-133	Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster	-1324223
-77061	P-134	Exploration of wild silk moth biodiversity in Manipur and their genetic characterization using molecular markers	-77061
-1118756	P-135	Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Pathogen Interaction in TB Infection	-171539

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

Annexure-I

Details of Closing balances of various Earmarked / Endowment Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2018

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
-196001	P-136	Raf Kinase - a key target for modern-day therapy against tumors	-196001
-1451500	P-138	Co-evaluation of Dnmt3l and Genomic imprinting	-1451500
20000	P-139	Evaluating the role of Sirtuins and epigenetic changes during cellular senescence in context of p53 status	20000
-608652	P-140	Development of baculovirus resistant silkworms strains through synthetic miRNA based knockdown of essential viral genes	-608652
-125000	P-141	Evaluating the functional role of PTEN interacting proteins in cell survival signaling and tumor suppression	-125000
-81861	P-142	Identification of H3K4 TRI Demethylase involved in erasing H3K4 trimethylation marks at E2F Responsive promoters	-81861
-719139	P-143	Microarray based characterisation of squamous cell carcinoma of the tongue occurring in non smokers	-719139
122130	P-144	Tri-National Training Program for Psychiatric Genetics	122130
3222	P-145	"H3K4 HMT family regulates cell cycle progression "	3222
59533	P-146	"Role of MLL in ribosomal RNA transcription "	59533
-272874	P-147	"The Effect of Parental Education, Ethics of Research Participation and Array Comparative Genomic Hybridization in Subjects with Mental Retardation (MR) and /or Autism "	-272874
-73001	P-149	"Role of SUMOylation in the pathobiology of Candida Glabrata "	-73001
199137	P-151	"Human Exome Sequencing to Identify Novel Genes for Medelian Disorders "	199137
-1123979	P-152	"Global transcriptomics of sex specific splicing "	-572237
1161773	P-153	"An attractive and promising strategy for early cancer diagnosis through the assembly of the human cancer volatome"	1138373
-434393	P-154	"Rational design, synthetic strategies for developing organometallic anticancer compounds based on organotin and organoiron "	-476750
335194	P-155	"Studies on the cellular roles of calcium signalling proteins in Neurospora crassa "	335194
-605123	P-156	"Targeting microbial quorum sensing to demonstrate potential application of cell-cell signaling molecules from Xanthomonas group of plant pathogen in disease control	-843369
124009	P-157	"Identification of novel antifungal drug and delineation of drug resistance mechanisms in an opportunistic human fungal pathogen Candida glabrata "	0
-168374	P-158	"Modulation of host immune responses by a PPE Protein of Mycobacterium tuberculosis: Understanding its role in host - pathogen cross-talk "	-297870
-300000	P-159	"Gene Targeting of microbial isolates to demonstrate potential plant growth promoting (PGP) traits by third generation sequencing "	-498696
-147180	P-160	"Understanding the role of novel adhesins of Xanthomonas oryzae PV oryzae in Virulence and colonization in Rice "	-309972
-464167	P-162	Characterization and design of inhibitors of Mycobacterium tuberculosis transcription	-803658
1530339	P-163	Unravelling new functions for the H-NS family of proteins in Gram-negative bacterial pathogens	247176
-29200	P-164	"A Yeast based screen for discovery of novel sirtuin inhibitors as anticancer agents "	-29200
862906	P-165	"Identification and functional characterization of immune response genes in silkworms"	722877
-368609	P-166	"Sequencing analysis of transcriptome variants in early-onset sporadic rectal cancer"	-138261
780652	P-167	"To elucidate the role of MLL complex in epigenetic specification of centromeres"	0
-161318	P-168	"A Search for nucleus -limited genes in Neurospora"	-439554
-332017	P-169	"Implementation of 3 year DNB Program in Medical Genetics by Department of Biotechnology in collaboration with National Board of Examination ag SGHR, NIBMG&CDFD"	-342565
-687887	P-17	"Studies on inositol-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV" – Transfer from IMTECH, Chandigarh	-687887
-383863	P-170	"Women Scientist Scheme ""Identification and character of deregulated micro RNAs in defined sub-set of early onset sporadic rectal cancer patients using transcriptome sequencing"	-658863

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

Annexure-I

Details of Closing balances of various Earmarked / Endowment Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2018

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
-1237535	P-171	"Role of vesicle-mediated transport and chromatin remodelling in the virulence of Candida glabrata"	1596152
40020	P-172	"Molecular Characterization of early onset sporadic rectal cancer"	129248
1672130	P-173	"Development and application of a next generation sequencing approach for molecular genetic analysis of lysosomal storage disorders"	1018438
209406	P-174	"Is non-canonical Wnt signalling a major player in early-onset sporadic rectal cancer"	459319
-121669	P-175	"Multi Centri Collaborative study of the Clinical, Biochemical and Molecular Characterization of Lysosomal storage disorders in India - The initiative for research in Lysosomal Storage Disorders"	-898484
208017	P-176	International Atomic Energy Agency	139289
-119970	P-177	"Morphological and molecular taxonomy of the Phlebotomus argentipes species complex in relation to transmission of Kala-azar in India"	-119970
184199	P-178	"Understanding differential signaling via toll like receptor-2: A proteomics approach"	268252
50000	P-179	"Quality Assurance Programme for Molecular and Prenatal Diagnosis of Hemoglobin Opathies"	0
-274286	P-18	"Mapping of receptor binding site on the Eythrocyte binding of malaria parasite"	-274286
63384	P-180	"Collaborative studies on genomic diversity among bombycoid silkmths in Asia"	8621
1223096	P-181	"To conduct multilocational field trails on transgenic BmNPV resistant silkworm strains to establish their efficacy and generate data for their regulatory approval"	1691792
533274	P-182	"Ramalingaswami Fellowship "	0
-1091800	P-183	"Prevalence and predictors of vitamin B12 deficiency: genetic associations for low vitamin B12 levels-multi-center a pan India study,"	0
123065	P-184	"Computational Approaches to Understanding Peptide- Protein Interactions involved in the Regulatory Events in the Cell"	-149102
1271410	P-185	"Investigating potential of mycobacterium tuberculosis protein PPE18 encapsulated nanoparticle as therapy for microbial sepsis"	885366
449029	P-186	"In vivo corss-talks between Rho-dependent transcription termination and other biological processes"	604691
1282677	P-187	"Understanding the mechanism of induction of innate immunity in plants by the Xanthomonas Diffusible signal factor (DSF)"	1488067
832894	P-188	"Identification of Novel Genes for Intellectual Disability"	806614
17423746	P-189	"Characterization of glycosylphosphatidylinositol-linked aspartyl proteases in Candida glabrata: role in pathogenicity"	14714544
245026	P-190	"Exploring mycobacteriophages to source novel factors / regulators of bacterial transcription machinery"	234953
5718535	P-191	"Human Frontier Science Program Reseech Grant - A comprehensive approach towards the chemistry & biology of polyphosphate: the forgotten biopolymer"	0
458917	P-192	"Design of peptide inhibitor(s) for the bacterial trancription terminator Rho, a potent drug target"	1648409
1001347	P-193	"Screening for male infertility markers in the human Yq12 heterochromatic block"	77682
210034	P-194	"Mechanisms and regulation of iron transportin the pathogenic yeast Candida glabrata"	0
872204	P-195	"Molecular and biophysical characterization of the ESAT-6: 2M complex and its effect on intracellular iron concentration and macrophage anti-mycobacterial effector responses"	1475532
1164021	p-196	"Exploring the volatome of noncommunicable diseases as a promising, innovative and integrating approach for its rapid diagnostics"	0
583730	P-197	"National Post Doctoral Fellowship"	268350
2493600	P-198	"Whole Genome Sequencing for characterization of novel genes and de novo balanced chromosomal rearrangements in human genetic disorders"	-54445
4013536	P-199	"Investigating cellular processes and pathways controlled by phosphatases"	1747473
-1888111	P-20	"Genomic Micro array R&D Programmes on infectious diseases and Neurological Disorders"	-1888111
1806199	P-200	"Characterization of divergent functions of ARID1A and ARID1B: the two alternative DNA binding constituents of the human SWI/SNF chromatic remodelling complex"	288591

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

Annexure-I

Details of Closing balances of various Earmarked / Endowment Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2018

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
1241000	P-201	"Defining the functions of MLL in mitosis"	1435959
603000	P-202	"To decipher the role of MLL Complex in the process of cytokinesis"	1736697
1186706	P-203	"Investigation of a potential novel function of fission yeast sirtuin family histone deacetylase Hst4 in regulation of DNA replication"	1764289
0	P-204	"To delineate the role of MLL complex in Microtubule organizing capability of Centrosome"	144331
0	P-205	"Genetic studies of foetuses with malformations for identification of Non-chromosomal syndromes and Mendelian disorders"	630948
0	P-206	"Characterization of the genetic etiological spectrum and identification of novel genetic etiologies for non-immune fetal hydrops"	300000
0	P-207	"Genome and transcriptome analysis of chilli anthracnose fungus colletotrichum truncatum"	2114590
0	P-208	"National Post Doctoral Fellowship "	173333
0	P-209	"Dissecting the contribution and interplay of MSI and CIMP in colorectal cancer in India"	1985915
0	P-211	"A comprehensive approach towards the chemistry & biology of polyphosphate: the forgotten biopolymer"	7112780
0	P-212	"Approaching Mycobacterium tuberculosis PPE protein Rv1168c (PPE17) as a potential marker for diagnosis of Tuberculosis (TB) patients in India"	2179000
0	P-213	"Exploring an oncogenic function of p53 mutations identified in Indian squamous cell carcinoma patients"	2724000
0	P-214	"Studies on Non-Canonical functions of splicing proteins in maintaining genomic stability"	824440
0	P-215	"Understanding Homothorax independent role of Hox cofactor Extradenticle in Drosophila neuroblast apoptosis"	970000
0	P-216	"Investigating the role of mycobacterial protein Rv2966c in modulating the host epigenetic circuitry during infection"	1768000
0	P-217	"BRICS Research Project - EpiMacroTB, ""Epigenetics of macrophages during Mycobacterium tuberculosis infection"	1141600
0	P-219	"Identification and molecular characterization of the CgHog1 kinase interactome: impact on iron homeostasis and Candida pathogenesis"	1500000
-34495	P-23	"Development of PCR base assays for detection of GMO S"	-34495
-529111	P-25	"Functional studies of Human Immuno - deficiency Virus Type- 2 (HIV-2) Viral protein X (VPX)"	-529111
-79533	P-26	Occurrence of Mutations in Non dividing cells of Escherichia Coli"	-79533
-37624	P-28	Baculovirus resistance in transgenic silkworms	-37624
-310302	P-29	"Development of Hospital Surveillance system by advanced diagnostics method & Molecular DNA fingerprinting techniques"	-310302
-234000	P-33	"Molecular and Epidemiological characterisation of cryptosporidium – An enteric protozoan parasite"	-234000
26334	P-34	"Molecular analysis of lepidopteran – specific immune proteins from silkworms"	26334
-283883	P-35	"Identification, Characterization and Physical mapping of Z-Chromosome linked genes of the silk worm, Bombyx mori"	-283883
2073896	P-36	"Development of Artificial retina using Bacterio rhodospin and genetically engineered analogues "	2073896
-4058	P-40	"Antioxidants as a potential immuno adjuvant in anti tuberculosis immunotherapy"	-4058
1873605	P-41	"Construction, characterization and analysis of expressed sequences from silkworm "	1873605
-457538	P-44	"Understanding of role of Ras and NO / iNOS signalling in promotion of hepatocellular carcinomas with persistent HBV infection"	-457538
-1586965	P-47	Research cum Training for DRDO Programme	-1586965
151826	P-48	'Molecular characterization of human liver stem cells for use in the treatment of hepatic diseases'.	151826
1041952	P-49A	International Atomic Energy Agency (IAEA)	1041952
-284065	P-51	"Understanding the mechanism of doxorubicin resistance in breast cancer cell line MCF-7"	-284065
-1231118	P-52	"Nucleo Cytoplasmic transport of HIV – 1 Vpr"	-1231118

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

Annexure-I

Details of Closing balances of various Earmarked / Endowment Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2018

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
-37877	P-54	"Study of viability of Mycobacterium leprae in clinical samples and possibility of its presence in the environment using nucleic acid amplification techniques."	-37877
224	P-55	"Identification of DNA Markers for baculovirus resistance in silkworm, Bombyx mori"	224
-1231164	P-56	"Genetics of transcription-replication interplay and of stress adaptation in bacteria"	-1231164
-2215024	P-59	"An integrated Approach towards understanding the biology of Mycobacterium tuberculosis: Genetic, biochemical, immunological and structural analyses."	-2215024
482124	P-60	"National Database of Prevalent Genetic Disorders in India: Development, Curation and Services"	482124
-280000	P-61	"Dissection of a novel phenotype of lethal accumulation of potassium in Escherichia coli mutants defective in thioredoxin/thioredoxin reductase and nucleoid protein H-NS"	-280000
-278928	P-62	"HIV – 1 Pathogenesis: Role of Integrase in Reverse Transcription and Nuclear Transport of Viral Genome"	-278928
-773874	P-63	"Upgradation of the existing computing infrastructure at the Bioinformatics facility at CDFD"	-773874
-158	P-64	Biotechnology for Leather: Towards cleaner processing phase-II	-158
-582647	P-65	"Molecular, genetic and functional analysis of the chromosomal plasticity region of the gastric pathogen Helicobacter pylori"	-582647
23733305	P-65A	APEDA-CDFD Centre for Basmati DNA Analysis	24524727
-681246	P-66	Human Epigenome Variation: Analysis of CpG island methylation in chromosomes 18 and Y, and in some Hox, insulin signaling and chromatin reprogramming genes	-681246
-113545	P-67	Identification of novel Esophageal Squamous cell carcinoma (ESCC) genes by using a combination of array-based CGH and gene expression micro arrays	-113545
-59874	P-68	Identification of High risk individual with pre-cancerous states of esophageal cancer.	-59874
-21336	P-70	Identification of disease causing mutations in familial hypertrophic cardiomyopathy (FHC) patients from Andhra Pradesh	-21336
-1421653	P-72	Nuances of non coding DNA near insulin-responsive genes.	-1421653
-857136	P-73	Identification and characterization of pancreatic cancer genes located within novel localized cpy number alterations	-857136
-10840	P-75	Preparing blueprint for the macromolecular crystallography beamline at Indus-II synchrotron source	-10840
-50234	P-76	A study of molecular markers in childhood autism with special references to nuclear factors - α APPA B	-50234
124277	P-77	Functional characterization of Mycobacterium tuberculosis PE/PPE proteins having SH3 binding domain : Understanding their role in modulating macrophage functions	124277
1304	P-78	Task force- IMD Newborn screening for Congenital Hypothyroidism & Congenital Adrenal Hyperplasia: A multicentric study	1304
-105086	P-79	Understanding the role of AGE proteins in inducing inflammatory responses and its regulation	-105086
-608222	P-80	Referral centre for detection of genetically modified foods employing DNA-based markets	-608222
143470	P-81	Reconstructing Cellular Networks: Two-component regulatory systems	143470
850453	P-81A	Financial assistance for award of J C Bose Fellowship to Dr J Gowrishankar	-60000
-369021	P-82	Functional genomic analysis of Candida Glabrata-macrophage	-369021
-1155594	P-83	Prokaryotic Transcription termination factor, Rho: Mechanism of Action and Biology	-1155594
-1150	P-84	Preparing for vaccine efficacy trials: Baseline epidemiology, improved diagnosis, markers of protection and phase I/II trials	-1150
-106479	P-84A	Human epigenetic to the rescue of human identification process: Enriching human DNA from DNA mixture employing antibodies directed against 5-methylcytosine followed by whole genome amplification	-106479
-1118755	P-85	IdeR associated gene regulatory network in mycobacteria	-1118755
-65698	P-87	Comparative genomics of wild silkworms	-65698
-636286	P-90	Role of Yapsins in the Pathobiology of Candida Glabrata	-636286
-1098900	P-91	DMMT3L: epigenetic correlation with cancer	-1098900

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

Annexure-I

Details of Closing balances of various Earmarked / Endowment Funds (Refer Sch-3)

For the Year Ended 31st MARCH 2018

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
268823	P-92	Swarnajayanti fellowship proj on "Designing transcription anti-terminators: a novel approach for making new inhibitors of gene expression"	268823
-611833	P-93/A1	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against tuberculosis	-611833
-3228626	P-93/A2	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis	-3228626
837745	P-93B2 (II)	Evaluation of peptides / small molecules targeting ESAT-6:B2M interaction and PPE18-TLR2 interaction as potent anti tuberculosis therapeutics	952280
-276552	P-97	Proteome-wide Analysis of Serine pyrophosphorylation by inositol pyrophosphates	-276552
-236042	P-98	Role of cell - cell signaling mediated by Diffusible signaling factor (DSF) in Xanthomonas virulence	-236042
-567516	P-99	Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosomae biogenesis	-567516
5912597			18840488

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
Details of Fixed Assets Fund (Capitalised portion of Project Grants)
For the Year Ended 31st MARCH 2018

Annexure-II

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
11713327	COE-I	COE for Genetics and Genomics of silkworms	11713327
12465940	COE-II	DBT Centre of Excellence for Microbial Biology	12773150
600000	P-03	"Transgenesis and Genetic basis of Pathogen Resistance in the Silkworm, Bombyx Mori	600000
329289	P-07	"Collection of well characterised clinical samples and strains of Mycobacterium tuberculosis and development of molecular techniques for detection of drug resistant strains – Multi Centric Project"	329289
588400	P-09	"NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics"	588400
47400	P-10	"Role of upstream sequence elements in Hyper activation of transcription from Baculovirus polyhedrin gene promoter"	47400
17784	P-100	Effect of reactive oxygen species on T-Cell immune response: An approach to understand the molecular mechanism of immunosuppression during tuberculosis - National Bioscience Award	17784
14378004	P-101	Role of inositol pyrophosphates in cell physiology: Investigating the biochemical significance of protein pyrophosphorylation - Senior Fellowship	14378004
698550	P-102	Understanding the role of Mycobacterium tuberculosis heat shockprotein 60 as Th1/Th2 immuno modular	698550
1000000	P-107	IYBA Project - Mechanism and role of bacterial cell-cell signaling molecules in plant defense response	1000000
3911516	P-109	Molecular dissection of PI3-Kinase/Akt pathway by using proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors	3911516
206800	P-111	Ramalingaswami Fellowship - Refractoriness mechanism in Mosquito: cracking molecular codes at genomic scale	206800
670095	P-113	Clinical and molecular genetic analysis of squamous cell carcinoma of the tongue	670095
475900	P-114	Evaluating the Calcineurin-NFAT Pathway and its regulators superoxide dismutase (SOD) AND RCAN1 (regulator of Calcineurin) Down Syndrome	475900
4580214	P-115	Setting up of the National Institute of Animal Biotechnology	4580214
800000	P-116	DBT-India and AIST - Japan : Understanding molecular mechanisms controlling dual role of Ras, Sirtuins and CARF in relation to cellular proliferation and senescence: Novel Strategy for developing cancer therapeutics	800000
183443	P-118	Construction of regulatory networks in Mycobacterium tuberculosis through analysis of gene expression data and transcription regulation predictions. (MOU with Russian Foundation)	183443
529750	P-12	Molecular genetics and Functional genomics of M.Tuberculosis patient isolates in India	529750
13632420	P-122	Understanding the role of Hox genes in anterior-posterior axis determination of the central nervous system	13963785
1674539	P-123	Establish a Max Planck Partner Group for Genetic Diversity Studies at CDFD	1932552
758900	P-126	Rho-dependent transcription termination machinery: mechanism of action	758900
6776327	P-127	Systematic studies on the functional network of phosphatases in cell life and death	6776327
1770000	P-128	Mechanism of iron acquisition and iron homeostasis in an opportunistic human pathogen Candida glabrata	1770000
1334600	P-13	"Programme to delineate gene functions in the post – genomics era by a systematic two gene knockout method"	1334600
1008000	P-130	Comparative genetic analysis of sex chromosomes and sex determining genes in silkworms	1008000
1054297	P-133	Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster	1054297
5500000	P-135	Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Pathogen Interaction in TB Infection	5500000
900000	P-137	Signaling pathways involved in down regulation of proinflammatory responses by PPE18 protein of Mycobacterium tuberculosis: Implication of PPE18 as therapeutics	900000
700000	P-138	Co-evaluation of Dnmt3l and Genomic imprinting	700000
500000	P-139	Evaluating the role of Sirtuins and epigenetic changes during cellular senescence in context of p53 status	500000
5163243	P-14	"Comparative and functional genomics approaches for the identification and characterization of genes responsible for multi drug resistance of mycobacterium tuberculosis"	5163243

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
Details of Fixed Assets Fund (Capitalised portion of Project Grants)

Annexure-II

For the Year Ended 31st MARCH 2018

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
500000	P-140	Development of baculovirus resistant silkworms strains through synthetic miRNA based knockdown of essential viral genes	500000
650000	P-142	Identification of H3K4 TRI Demethylase involved in erasing H3K4 trimethylation marks at E2F Responsive promoters	650000
1868000	P-145	"H3K4 HMT family regulates cell cycle progression"	1868000
1000000	P-146	"Role of MLL in ribosomal RNA transcription"	1000000
469000	P-149	"Role of SUMOylation in the pathobiology of Candida Glabrata"	469000
6000000	P-15	"The Helicobacter Pylori genome programme – Genome sequencing, functional analysis and comparative genomics of the strains obtained from Indian patients"	6000000
17421	P-152	Global transcriptomics of sex specific splicing	17421
3000000	P-153	"An attractive and promising strategy for early cancer diagnosis through the assembly of the human cancer volatome"	3000000
132495	P-154	"Rational design, synthetic strategies for developing organometallic anticancer compounds based on organotin and organoiron"	132495
-4634	P-156	"Targeting microbial quorum sensing to demonstrate potential application of cell-cell signaling molecules from Xanthomonas group of plant pathogen in disease control"	-4634
992265	P-157	"Identification of novel antifungal drug and delineation of drug resistance mechanisms in an opportunistic human fungal pathogen Candida glabrata"	1081661
343121	P-158	"Modulation of host immune responses by a PPE Protein of Mycobacterium tuberculosis: Understanding its role in host - pathogen cross-talk"	343121
1814901	P-16	NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics	1814901
160082	P-165	Identification and functional characterization of immune response genes in silkworms	160082
2000000	P-166	Sequencing analysis of transcriptome variants in early-onset sporadic rectal cancer	2000000
560757	P-167	"To elucidate the role of MLL complex in epigenetic specification of centromeres"	488000
396000	P-168	"A Search for nucleus -limited genes in Neurospora"	396000
295560	P-171	Role of vesicle-mediated transport and chromatin remodelling in the virulence of Candida glabrata	406301
1500000	P-172	Molecular Characterization of early onset sporadic rectal cancer	1500000
166729	P-184	Computational Approaches to Understanding Peptide- Protein Interactions involved in the Regulatory Events in the Cell"	166729
84421	P-185	Investigating potential of mycobacterium tuberculosis protein PPE18 encapsulated nanoparticle as therapy for microbial sepsis	264191
2180896	P-186	In vivo cross-talks between Rho-dependent transcription termination and other biological processes	2180896
0	P-188	Identification of Novel Genes for Intellectual Disability	109430
600000	P-189	Characterization of glycosylphosphatidylinositol-linked aspartyl proteases in Candida glabrata: role in pathogenicity	1648275
50000	P-190	Exploring mycobacteriophages to source novel factors / regulators of bacterial transcription machinery	50000
39060	P-191	"Human Frontier Science Program Research Grant - A comprehensive approach towards the chemistry & biology of polyphosphate: the forgotten biopolymer	1451177
2000000	P-192	Design of peptide inhibitor(s) for the bacterial transcription terminator Rho, a potent drug target	2157173
289966	P-194	Mechanisms and regulation of iron transport in the pathogenic yeast Candida glabrata	500000
0	P-197	National Post Doctoral Fellowship	123841
0	P-198	Whole Genome Sequencing for characterization of novel genes and de novo balanced chromosomal rearrangements in human genetic disorders"	193695
0	P-199	Investigating cellular processes and pathways controlled by phosphatases	1408161
0	P-201	Defining the functions of MLL in mitosis	194313
0	P-202	To decipher the role of MLL Complex in the process of cytokinesis	256738

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Annexure-II

Details of Fixed Assets Fund (Capitalised portion of Project Grants)

For the Year Ended 31st MARCH 2018

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
0	P-203	Investigation of a potential novel function of fission yeast sirtuin family histone deacetylase Hst4 in regulation of DNA replication	80645
0	P-209	Dissecting the contribution and interplay of MSI and CIMP in colorectal cancer in India	387500
244400	P-17	"Studies on inositol-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV" – Transfer from IMTECH, Chandigarh	244400
344020	P-18	"Mapping of receptor binding site on the Erythrocyte binding of malaria parasite"	344020
7246511	P-19	"Construction of Integrated RAPD, RFLP and Microsatellite linkage map of the Silkworm, Bombyx mori and its correlation with the Phenotypic linkage Map"	7246511
27331134	P-20	"Genomic Micro array R&D Programmes on infectious diseases and Neurological Disorders"	27331134
5300000	P-21	Development of Versatile, portable software for Bio-informatics	5300000
603747	P-22	"Biotechnology for leather – towards cleaner processing"	603747
375999	P-23	"Development of PCR base assays for detection of GMO S"	375999
0	P-24	Establishing a central facility on "Aerosol challenge in a containment facility"	0
600000	P-25	"Functional studies of Human Immuno - deficiency Virus Type- 2 (HIV-2) Viral protein X (VPX)"	600000
500000	P-26	Occurrence of Mutations in Non dividing cells of Escherichia Coli"	500000
260367	P-29	"Development of Hospital Surveillance system by advanced diagnostics method & Molecular DNA fingerprinting techniques"	260367
3746538	P-30	Transcription termination and anti termination in E-coli	3746538
3131006	P-31	Role of K-ras in Lung type II epithelial cells	3131006
4857938	P-36	"Development of Artificial retina using Bacterio rhodospin and genetically engineered analogues "	4857938
358470	P-39	"Computational analysis and functional characterization of mycobacterial protein(s) interacting with macrophage effector – APC functions – an approach to understand the molecular basis of pathogenesis of M. tuberculosis"	358470
49738	P-40	"Antioxidants as a potential immuno adjuvant in anti tuberculosis immunotherapy"	49738
3894086	P-41	"Construction, characterization and analysis of expressed sequences from silkworm "	3894086
9500000	P-42	"Structural and functional studies on Mycobacterium tuberculosis heat shock proteins".	9500000
11970000	P-43	"A generalized mechanism of transcription termination in prokaryotes: a quest for mechanism based transcription inhibitors for microbial pathogens".	11970000
3331377	P-45	Specialized chromatin structures as epigenetic imprints to distinguish parental alleles".	3331377
416137	P-46	"Effect of reactive oxygen species (ROS) on immune response : Relevance in immunosuppression and Pathogenesis"	416137
377567	P-47	Research cum Training for DRDO Programme	377567
1413292	P-48	'Molecular characterization of human liver stem cells for use in the treatment of hepatic diseases'.	1413292
198095	P-50	"Cervical cancer prevention by multiple strategies in rural community in Andhra pradesh"	198095
401738	P-51	"Understanding the mechanism of doxorubicin resistance in breast cancer celline MCF-7"	401738
1359129	P-52	"Nucleo Cytoplasmic transport of HIV – 1 Vpr"	1359129
1114495	P-53	Collaborative research project on molecular ecology and systematics	1114495
1163764	P-56	"Genetics of transcription-replication interplay and of stress adaptation in bacteria"	1163764
2131403	P-57	Improved genome annotation through a combination of machine learning and experimental methods: Plasmodium falciparum as a case study.	2131403
63000	P-58	"Indo-Malaysian collaboration in Bioinformatics: Development and maintenance of a web-portal hosting databases and tools of common interest"	63000
32974662	P-59	"An integrated Approach towards understanding the biology of Mycobacterium tuberculosis: Genetic, biochemical, immunological and structural analyses."	32974662
5720800	P-60	"National Database of Prevalent Genetic Disorders in India: Development, Curation and Services"	5720800
4308314	P-62	"HIV – 1 Pathogenesis: Role of Integrase in Reverse Transcription and Nuclear Transport of Viral Genome"	4308314

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

Annexure-II

Details of Fixed Assets Fund (Capitalised portion of Project Grants)

For the Year Ended 31st MARCH 2018

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
9637574	P-63	"Upgradation of the existing computing infrastructure at the Bioinformatics facility at CDFD"	9637574
600585	P-64	Biotechnology for Leather: Towards cleaner processing phase-II	600585
260000	P-65	"Molecular, genetic and functional analysis of the chromosomal plasticity region of the gastric pathogen <i>Helicobacter pylori</i> "	260000
16924622	P-65A	APEDA-CDFD Centre for Basmati DNA Analysis	16924622
264430	P-66	Human Epigenome Variation: Analysis of CpG island methylation in chromosomes 18 and Y, and in some Hox, insulin signaling and chromatin reprogramming genes	264430
622747	P-67	Identification of novel Esophageal Squamous cell carcinoma (ESCC) genes by using a combination of array-based CGH and gene expression micro arrays	622747
235593	P-69	ICMR adhoc New Scheme Understanding the role of PE/PPE family of M tuberculosis in the activation of HIV virus type I long terminal repeat (HIV-ILTP)	235593
1012807	P-70	Identification of disease causing mutations in familial hypertrophic cardiomyopathy (FHC) patients from Andhra Pradesh	1012807
1573795	P-71	Referral Centre for Genetic fidelity testing of tissue culture raised plants	1573795
45653	P-72	Nuances of non coding DNA near insulin-responsive genes.	45653
1000000	P-74	Molecular basic of insect plant interactions in rice under the national fund for basic and strategic research in agriculture	1000000
33672	P-75	Preparing blueprint for the macromolecular crystallography beamline at Indus-II synchrotron source	33672
245266	P-76	A study of molecular markers in childhood autism with special references to nuclear factors - α APPA B	245266
1543605	P-77	Functional characterization of Mycobacterium tuberculosis PE/PPE proteins having SH3 binding domain : Understanding their role in modulating macrophage functions	1543605
0	P-78	Task force- IMD Newborn screening for Congenital Hypothyroidism & Congenital Adrenal Hyperplasia: A multicentric study	0
496826	P-79	Understanding the role of AGE proteins in inducing inflammatory responses and its regulation	496826
4192480	P-80	Referral centre for detection of genetically modified foods employing DNA-based markets	4192480
205073	P-81A	Financial assistance for award of J C Bose Fellowship to Dr J Gowrishankar	205073
1480220	P-82	Functional genomic analysis of <i>Candida Glabrata</i> -macrophage	1480220
912255	P-83	Prokaryotic Transcription termination factor, Rho: Mechanism of Action and Biology	912255
388583	P-83A	Understanding the mechanism of Azadirachtin-mediated cell signaling: role in anti-inflammation and anti-tumorigenesis	388583
44854	P-84	Preparing for vaccine efficacy trials: Baseline epidemiology, improved diagnosis, markers of protection and phase I/II trials	44854
1430573	P-84A	Human epigenetic to the rescue of human identification process: Enriching human DNA from DNA mixture employing antibodies directed against 5-methylcytosine followed by whole genome amplification	1430573
374630	P-89	Characterization of Mycobacterium tuberculosis transcription machinery and Bacteriophage metagenomics	374630
1376869	P-90	Role of Yapsins in the Pathobiology of <i>Candida Glabrata</i>	1376869
932151	P-91	DMMT3L: epigenetic correlation with cancer	932151
8500000	P-92	Swarnajayanti fellowship proj on "Designing transcription anti-terminators: a novel approach for making new inhibitors of gene expression"	8500000
2212534	P-93/A1	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against tuberculosis	2212534
913430	P-93/A2	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis	913430
246320	P-95	Construction of regulatory networks in prokaryotes through protein: Protein interaction predictions and transcription regulation predictions. (MOU with Russian Foundation)	246320
1000000	P-97	Proteome-wide Analysis of Serine pyrophosphorylation by inositol pyrophosphates	1000000
2816418	P-98	Role of cell - cell signaling mediated by Diffusible signaling factor (DSF) in <i>Xanthomonas virulence</i>	2816418
2963482	P-99	Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosome biogenesis	2963482
320849552			327635212

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Annexure: A Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	I-Remittances	
4910125.00	TDS	5042467.00
8974333.00	Income Tax	14145332.00
278372.00	Works Tax	1337534.00
1865076.00	LIC	1755463.00
251264.00	GSLI	173120.00
1143660.00	Public Provident Fund	0.00
506200.00	Professional Tax	442350.00
4987454.00	Service Tax	2664167.00
899765.00	Others (I-Remittances)	289925.00
462714.00	Health Insurance	129638.00
2304183.00	ECCS	3013664.00
381481.00	Contract Staff security deposit	15000.00
12569.00	STAFF BENEVOLENT FUND	30104.00
0.00	EPF	105999.00
0.00	GST	58788.00
26977196.00		29203551.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
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Annexure: B Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Advance refunds/recovery/Adjst.	
734321.00	Advance for Expenses- purchases by Staff	426739.00
6067820.00	Chemicals [Advance]	0.00
70328.00	Computer Advance [Research Fellows]	0.00
168592.00	Computer Advance [Staff]	114541.00
29685.00	Consumables, glassware and Spares [Advance]	0.00
1800.00	Conveyance [Advance]	0.00
78324.00	Conveyance Advance	79256.00
6638.00	DA [Advance]	0.00
909438.00	EMD	208000.00
5613268.00	Equipment [Advance]	2214182.00
138600.00	Festival Advance	42300.00
0.00	GDA [Others]	16000.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
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Annexure: B Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
15950.00	General Deposits And Advances	1929593.00
0.00	Human Resource Development - Training of Staff - Conferences [Advance]	0.00
55200000.00	Inter Bank Transfer	98484058.00
157200.00	Lab Security Deposit & Hostel Security Deposit	178000.00
690500.00	LTC [Advance]	417780.00
30843.00	Miscellaneous Salary [Advance]	95678.00
260129.00	Others [Advances]	0.00
53387.00	Pay of Establishment [Advance]	40821.00
456821.00	Revolving Advance	284057.00
952850.00	Security Deposit	3367370.00
199732.00	TA Abroad [Advance]	73778.00
1363959.00	TA-DA-Hon within India [Advance]	44000.00
11500.00	Trainee Security Deposit	10000.00
45000.00	Water [Advance]	0.00
178928.00	Workshop & Conference	0.00
73435613.00		108026153.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
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Annexure: C Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Projects - Receipts	
8768000.00	COE1/CORE	0.00
775000.00	COE1/P-I	0.00
643000.00	COE1/P-II	0.00
1090000.00	COE1/P-III	0.00
2100000.00	COE2-II/P-1	1489000.00
1061000.00	COE2-II/P-A	744000.00
488000.00	COE2-II/P-B	250000.00
1061000.00	COE2-II/P-C	744000.00
496000.00	COE2-II/P-D	250000.00
866000.00	COE2-II/P-E	744000.00
3447000.00	COE2-II-Core	4929000.00
442000.00	COE-I/P-IV	0.00
2028298.00	others	1136685.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2018**

Annexure: C Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
172000.00	P-110	0.00
2722184.00	P-122	3462961.00
1648000.00	P-123	0.00
663747.00	P-127	0.00
500000.00	P-133	0.00
0.00	P-135	2424800.00
662545.00	P-143	0.00
0.00	P-152	587717.00
1787000.00	P-153	0.00
1638000.00	P-157	0.00
2790992.00	P-158	0.00
699600.00	P-162	0.00
1483389.00	P-163	0.00
0.00	P-166	1359100.00
900000.00	P-167	0.00
2535600.00	P-169	3858700.00
1100000.00	P-170	0.00
0.00	P-171	3533564.00
1000000.00	P-172	800000.00
2107380.00	P-173	355990.00
0.00	P-174	500000.00
2214648.00	P-175	363913.00
207044.00	P-176	0.00
225000.00	P-177	0.00
1000000.00	P-178	900000.00
100000.00	P-179	50000.00
0.00	P-181	1164000.00
2110000.00	P-182	0.00
0.00	P-183	1091800.00
0.00	P-185	545000.00
1841600.00	P-186	1599800.00
0.00	P-187	600000.00
0.00	P-188	1250000.00
5629854.00	P-189	3605421.00

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Annexure: C Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
0.00	P-190	950000.00
7765092.00	P-191	0.00
3819000.00	P-192	1819800.00
1050000.00	P-193	0.00
500000.00	P-194	0.00
1285000.00	P-195	1285000.00
1281744.00	p-196	0.00
960000.00	P-197	559246.00
2556000.00	P-198	400000.00
4013536.00	P-199	8034427.00
1830000.00	P-200	0.00
1241000.00	P-201	2320000.00
603000.00	P-202	1800000.00
1186706.00	P-203	1538000.00
0.00	P-204	558333.00
0.00	P-205	1484600.00
0.00	P-206	300000.00
0.00	P-207	2456600.00
0.00	P-208	960000.00
0.00	P-209	3054000.00
0.00	P-211	7602399.66
0.00	P-212	2179000.00
0.00	P-213	2724000.00
0.00	P-214	854333.00
0.00	P-215	970000.00
0.00	P-216	1768000.00
0.00	P-217	1141600.00
0.00	P-219	1500000.00
1004370.00	P-65A	1001001.00
1360000.00	P-81A	0.00
737000.00	P-93B2 (II)	816700.00
90196329.00		86416490.66

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2018**

Annexure: D Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Advances	
653985.00	Advance for Expenses- purchases by Staff	545167.00
6024000.00	Chemicals [Advance]	3535441.00
48400.00	Computer Advance [Research Fellows]	0.00
60000.00	Computer Advance [Staff]	0.00
1613098.00	Consumables, glassware and Spares [Advance]	512655.00
60113.00	Conveyance Advance	0.00
463820.00	EMD	434270.00
23750711.00	Equipment [Advance]	54230746.00
81000.00	Festival Advance	0.00
0.00	GDA [Others]	1000.00
55200000.00	Inter Bank Transfer	98484058.00
135520.00	Lab Security Deposit & Hostel Security Deposit	126380.00
27849.00	Liveries & Blankets [Advance]	0.00
522400.00	LTC [Advance]	573984.00
854.00	Magzines [Advance]	0.00
407759.00	Others [Advances]	33904.00
0.00	Others [Contingencies Advance]	17453.00
0.00	Printing & Stationery [Advance]	188800.00
442756.00	Revolving Advance	287000.00
8000.00	Scientific Workshops - Symposiums - Seminars [Advance]	0.00
49140.00	Security Deposit	418515.00
0.00	Software [Advance]	375400.00
0.00	TA Abroad [Advance]	218267.00
1293660.00	TA-DA-Hon within India [Advance]	30000.00
50000.00	Telephone [Advance]	0.00
11000.00	Trainee Security Deposit	10500.00
45000.00	Water [Advance]	0.00
826689.00	Workshop & Conference	170564.00
91775754.00		160194104.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
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Annexure: E Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	I-Remittances	
321745.00	Contract Staff security deposit	511578.00
2140898.00	ECCS	3447809.00
0.00	EPF	105999.00
259987.00	GSLI	166210.00
0.00	GST on Reverse Charge	67988.00
835000.00	Health Insurance	0.00
8161043.00	Income Tax	14129968.00
1865076.00	LIC	1755463.00
708678.00	Others (I-Remittances)	860080.00
508250.00	Professional Tax	444600.00
1158742.00	Public Provident Fund	0.00
4134084.00	Service Tax	3142319.00
0.00	STAFF BENEVOLENT FUND	0.00
5271099.00	TDS	5081943.00
174000.00	Works Tax	17133.00
25538602.00		29731090.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
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Annexure: F Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Projects - Expenditure	
6942349.00	COE1/CORE	500408.00
143520.00	COE1/P-I	0.00
193527.00	COE1/P-II	0.00
225358.00	COE1/P-III	0.00
1655776.00	COE2-II/P-1	868101.00
1258535.00	COE2-II/P-A	49400.00
953955.00	COE2-II/P-B	592800.00
330000.00	COE2-II/P-C	0.00
357000.00	COE2-II/P-D	0.00
597400.00	COE2-II/P-E	388826.00
2547907.00	COE2-II-Core	2970523.00
107640.00	COE-I/P-IV	0.00
0.00	others	714186.00

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Annexure: F Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
39000.00	P-107	0.00
1130336.00	P-109	866029.00
5652169.00	P-122	3484085.00
979012.00	P-123	328129.00
49400.00	P-126	0.00
2559030.00	P-127	0.00
143127.00	P-130	0.00
1121233.00	P-133	0.00
0.00	P-134	0.00
782621.00	P-135	1477583.00
-48800.00	P-138	0.00
13084.00	P-149	0.00
176714.00	P-151	0.00
1093165.00	P-152	35975.00
560922.00	P-153	23400.00
447903.00	P-154	42357.00
845072.00	P-156	238246.00
152192.00	P-157	124009.00
384020.00	P-158	129496.00
0.00	P-159	198696.00
105513.00	P-160	162792.00
142000.00	P-162	339491.00
631710.00	P-163	1283163.00
704924.00	P-165	140029.00
404305.00	P-166	1128752.00
689135.00	P-167	780652.00
161318.00	P-168	278236.00
2884532.00	P-169	3869248.00
823996.00	P-170	275000.00
1448958.00	P-171	699877.00
1071830.00	P-172	710772.00
923203.00	P-173	1009682.00
311136.00	P-174	250087.00
903645.00	P-175	1140728.00

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Annexure: F Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
199130.00	P-176	68728.00
147576.00	P-177	0.00
815801.00	P-178	815947.00
0.00	P-179	100000.00
54502.00	P-180	54763.00
520904.00	P-181	695304.00
1299226.00	P-182	533274.00
1091800.00	P-183	0.00
834677.00	P-184	272167.00
360797.00	P-185	931044.00
3802571.00	P-186	1444138.00
85323.00	P-187	394610.00
617106.00	P-188	1276280.00
5064575.00	P-189	6314623.00
854974.00	P-190	960073.00
2046557.00	P-191	5718535.00
3360083.00	P-192	630308.00
48653.00	P-193	923665.00
289966.00	P-194	210034.00
412796.00	P-195	681672.00
117723.00	p-196	1164021.00
376270.00	P-197	874626.00
62400.00	P-198	2948045.00
0.00	P-199	10300490.00
23801.00	P-200	1517608.00
0.00	P-201	2125041.00
0.00	P-202	666303.00
0.00	P-203	960417.00
0.00	P-204	414002.00
0.00	P-205	853652.00
0.00	P-207	342010.00
0.00	P-208	786667.00
0.00	P-209	1068085.00
0.00	P-211	489619.00
0.00	P-214	29893.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2018**

Annexure: F Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
82270.00	P-65A	119178.00
	P-81	0.00
512167.00	P-81A	910453.00
190135.00	P-93/A2	0.00
383090.00	P-93B2 (II)	702165.00
66254245.00		73398198.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2018**

Annexure: G Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	CDFD C.P.F ACCOUNT	
44620022.00	Opening Balance	43287242.00
	Add:	
5192511.00	Employee subscription/ refunds	11590032.00
6986.00	Transfer from other departments	0.00
0.00	Institute contribution (inc. Projects staff)	0.00
277728.00	Interest received	325840.00
6810005.00	Less Advances/withdrawals/Transfer/Adjst	2682786.00
43287242.00		52520328.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2018**

Annexure: H Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	LOANS AND ADVANCES	
	LOANS AND ADVANCES	
190569.00	Advance for Expenses- purchases by Staff	0.00
4310.00	Advances [Previous Years]	0.00
2916312.00	Chemicals [Advance]	0.00
135445.00	Computer Advance [Research Fellows]	0.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2018**

Annexure: H Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
216786.00	Computer Advance [Staff]	0.00
13688118.00	Consumables, glassware and Spares [Advance]	0.00
165077.00	Conveyance Advance	0.00
20687459.00	Equipment [Advance]	72704023.00
41850.00	Festival Advance	0.00
793547.00	Health Insurance	0.00
158200.00	Liveries & Blankets [Advance]	0.00
2391449.00	LTC [Advance]	0.00
854.00	Magzines [Advance]	0.00
95678.00	Miscellaneous Salary	0.00
67325.00	NPS Subscription	0.00
22700.00	Office Equipment [Advance]	22700.00
5973311.00	Others [Advances]	0.00
40821.00	Pay of Establishment	0.00
304569.00	Rent [Advance]	0.00
38436883.00	Research Fellows-Associates	0.00
105642.00	Revolving Advance	0.00
8000.00	Scientific Workshops - Symposiums - Seminars [Advance]	0.00
50000.00	Telephone [Advance]	0.00
24500.00	Trainee Security Deposit	0.00
11510.00	Transport maintenance [Advance]	0.00
287622.00	Workshop & Conference	0.00
86818537.00		72726723.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2018**

Annexure: I Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	DEPOSITS	
15633520.00	General Deposits And Advances	13703927.00
839427.00	GDA[Others]	824427.00
16472947.00		14528354.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2018**

Annexure: J Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	INVESTMENT A/C	
33741214.00	Deposit with Banks	31870241.00
5062115.00	Employee subscription	11565032.00
6933088.00	Less Transfer To Bank A/C	20047578.00
31870241.00		23387695.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2018**

Annexure: K Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	LOANS AND ADVANCES	
0.00	Advances [Previous Years]	4310.00
0.00	Chemicals [Advance]	6451753.00
0.00	Consumables, glassware and Spares [Advance]	14200773.00
0.00	Diagnostics Collaboration With NIMS	220965.00
0.00	ECCS	270860.00
0.00	GST on Reverse Charge	9200.00
0.00	Health Insurance	663909.00
0.00	Liveries & Blankets [Advance]	158200.00
0.00	LTC [Advance]	2547653.00
0.00	Magzines [Advance]	854.00
0.00	Others (I-Remittances)	82513.00
0.00	Others [Advances]	6007215.00
0.00	Others [Contingencies Advance]	17453.00
0.00	Printing & Stationery [Advance]	188800.00
0.00	Rent [Advance]	304569.00
0.00	Research Fellows-Associates	49313242.00
0.00	Revolving Advance	108585.00
0.00	Scientific Workshops - Symposiums - Seminars [Advance]	8000.00
0.00	Software [Advance]	375400.00
0.00	TA Abroad [Advance]	34913.00
0.00	Telephone [Advance]	50000.00
0.00	Trainee Security Deposit	25000.00
0.00	Transport maintenance [Advance]	11510.00
0.00	Workshop & Conference	458186.00
0.00		81513863.00

**CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2018**

Annexure: L Forming part of Receipts and Payment a/c

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	LOANS AND ADVANCES	
0.00	Advance for Expenses- purchases by Staff	308997.00
0.00	Computer Advance [Research Fellows]	135445.00
0.00	Computer Advance [Staff]	102245.00
0.00	Conveyance Advance	85821.00
0.00		632508.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-03 : "Transgenesis and Genetic basis of Pathogen Resistance in the Silkworm, Bombyx Mori									
Pi:									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		630047.00		Opening Balance	630047.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			0.00		630047.00			630047.00	
630047.00		Excess of Expenditure Over Income	630047.00		0.00		Closing Balance	0.00	
630047.00			630047.00		630047.00			630047.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-09: "NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics"									
Pi: Dr Seyed E Hasnain									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
244305.00		Opening Balance	244305.00		0.00		Opening Balance	0.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
244305.00			244305.00		0.00			0.00	
0.00		Excess of Expenditure over Income	0.00		244305.00		Closing Balance	244305.00	
244305.00			244305.00		244305.00			244305.00	

<p align="center">P-10: "Role of upstream sequence elements in Hyper activation of transcription from Baculovirus polyhedrin gene promoter" CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P.I: Dr M D Bashyam Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	28332.00		0.00
28332.00	Excess of Expenditure over Income	28332.00	0.00	Closing Balance	28332.00
28332.00		28332.00	28332.00		28332.00

<p align="center">P-13: "Programme to delineate gene functions in the post - genomics era by a systematic two gene knockout method" CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P.I: Dr J Gowrishankar Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
3947.00	Opening Balance	3949.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
3947.00		3947.00	0.00		0.00
0.00	Excess of Expenditure over Income	0.00	3947.00	Closing Balance	3947.00
3947.00		3947.00	3947.00		3947.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-17: "Studies on inositol-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV" – Transfer from IMTECH, Chandigarh P.I: Dr Sekhar C Mande Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
0.00	Opening Balance	0.00	687887.00	Opening Balance	687887.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	687887.00		687887.00
687887.00	Excess of Expenditure over Income	687887.00	0.00	Closing Balance	0.00
687887.00		687887.00	687887.00		687887.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-18: "Mapping of receptor binding site on the Eythrocyte binding of malaria parasite" P.I: Dr Akash Ranjan Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
0.00	Opening Balance	0.00	274286.00	Opening Balance	274286.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	274286.00		274286.00
274286.00	Excess of Expenditure over Income	274286.00	0.00	Closing Balance	0.00
274286.00		274286.00	274286.00		274286.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-20: "Genomic Micro array R&D Programmes on infectious diseases and Neurological Disorders"					
P.I: Dr Hasnain & Dr Bashyam					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
0.00	Opening Balance	0.00	1888111.00	Opening Balance	1888111.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1888111.00		1888111.00
1888111.00	Excess of Expenditure over Income	1888111.00	0.00	Closing Balance	0.00
1888111.00		1888111.00	1888111.00		1888111.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-23: "Development of PCR base assays for detection of GMO S"					
P.I: Dr Nagaraju & Dr Niyaz Ahmed					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
0.00	Opening Balance	0.00	34495.00	Opening Balance	34495.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	34495.00		34495.00
34495.00	Excess of Expenditure over Income	34495.00	0.00	Closing Balance	0.00
34495.00		34495.00	34495.00		34495.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-25: "Functional studies of Human Immuno - deficiency Virus Type- 2 (HIV-2) Viral protien X (VPX)"							
P.I: Dr Mahalingam & Dr Mande							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	529111.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			529111.00	
529111.00		Excess of Expenditure over Income	529111.00		Closing Balance	0.00	
529111.00			529111.00			529111.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-26: Occurrence of Mutations in Non dividing cells of Escherichia Coli"							
P.I: Dr Mahalingam & Dr Mande							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	79533.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			79533.00	
79533.00		Excess of Expenditure over Income	79533.00		Closing Balance	0.00	
79533.00			79533.00			79533.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-28: Baculovirus resistance in transgenic silkworms P.I: Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		37624.00		Opening Balance	37624.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			0.00		37624.00			37624.00	
37624.00		Excess of Expenditure over Income	37624.00		0.00		Closing Balance	0.00	
37624.00			37624.00		37624.00			37624.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-29: "Development of Hospital Surveillance system by advanced diagnostics method & Molecular DNA fingerprinting techniques" P.I: Dr K Prashanth Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		310302.00		Opening Balance	310302.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			0.00		310302.00			310302.00	
310302.00		Excess of Expenditure over Income	310302.00		0.00		Closing Balance	0.00	
310302.00			310302.00		310302.00			310302.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-33: "Molecular and Epidemiological characterisation of cryptosporidium – An enteric protozoan parasite"									
P.I: Dr Radha Rama Devi									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		234000.00		Opening Balance	234000.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			0.00		234000.00			234000.00	
234000.00		Excess of Expenditure over Income	234000.00		0.00		Closing Balance	0.00	
234000.00			234000.00		234000.00			234000.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-34: "Molecular analysis of lepidopteran – specific immune proteins from silkworms"									
P.I: Dr J Nagaraju									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
26334.00		Opening Balance	26334.00		0.00		Salaries - Manpower	0.00	
0.00		Grant In Aid	0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
26334.00			26334.00		0.00			0.00	
0.00		Excess of Expenditure over Income	0.00		26334.00		Closing Balance	26334.00	
26334.00			26334.00		26334.00			26334.00	

<p align="center">P-35: "Identification, Characterization and Physical mapping of Z-Chromosome linked genes of the silk worm, Bombyx mori" Centre for DNA Fingerprinting and Diagnostics, Hyderabad P.I: Dr J Nagaraju Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	283883.00	Opening Balance	283883.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	283883.00		283883.00
283883.00	Excess of Expenditure over Income	283883.00	0.00	Closing Balance	0.00
283883.00		283883.00	283883.00		283883.00

<p align="center">P-36: "Development of Artificial retina using Bacterio rhodospin and genetically engineered analogues " Centre for DNA Fingerprinting and Diagnostics, Hyderabad P.I: Dr Sekhar C Mande Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
2073896.00	Opening Balance	2073896.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2073896.00		2073896.00	0.00		0.00
0.00	Excess of Expenditure over Income	0.00	2073896.00	Closing Balance	2073896.00
2073896.00		2073896.00	2073896.00		2073896.00

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-40: "Antioxidants as a potential immuno adjuvant in anti tuberculosis immunotherapy" P.I: Dr Sangita Mukhopadhyay Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	4058.00	Opening Balance	4058.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	4058.00		4058.00
4058.00	Excess of Expenditure over Income	4058.00	0.00	Closing Balance	0.00
4058.00		4058.00	4058.00		4058.00

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-41: "Construction, characterization and analysis of expressed sequences from silkworm " P.I: Dr J Nagaraju Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
1873605.00	Opening Balance	1873605.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1873605.00		1873605.00	0.00		0.00
0.00	Excess of Expenditure over Income	0.00	1873605.00	Closing Balance	1873605.00
1873605.00		1873605.00	1873605.00		1873605.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-44: "Understanding of role of Ras and NO / iNOS signalling in promotion of hepatocellular carcinomas with persistent HBV infection" P.I: Dr Gayatri Ramakrishna Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	457538.00	Opening Balance	457538.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	457538.00		457538.00
457538.00	Excess of Expenditure over Income	457538.00	0.00	Closing Balance	0.00
457538.00		457538.00	457538.00		457538.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-47: Research cum Training for DRDO Programme P.I: Dr Gowrishankar, Dr Mahalingam, Dr Mande, Dr Nagaraju, Dr Ni Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	1586965.00	Opening Balance	1586965.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1586965.00		1586965.00
1586965.00	Excess of Expenditure over Income	1586965.00	0.00	Closing Balance	0.00
1586965.00		1586965.00	1586965.00		1586965.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-48: 'Molecular characterization of human liver stem cells for use in the treatment of hepatic diseases'. P.I: Dr Sanjeev Khosla Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
151826.00	Opening Balance	151826.00	0.00	Salaries - Manpower	0.00
0.00	Grant In Aid	0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
151826.00		151826.00	0.00		0.00
0.00	Excess of Expenditure over Income	0.00	151826.00	Closing Balance	151826.00
151826.00		151826.00	151826.00		151826.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-49A: International Atomic Energy Agency (IAEA) P.I: J Nagaraju Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
1041952.00	Opening Balance	1041952.00	0.00	Salaries - Manpower	0.00
0.00	Grant In Aid	0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1041952.00		1041952.00	0.00		0.00
0.00	Excess of Expenditure Over Income	0.00	1041952.00	Closing Balance	1041952.00
1041952.00		1041952.00	1041952.00		1041952.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-51: "Understanding the mechanism of doxorubicin resistance in breast cancer cellline MCF-7"					
P.I: Dr Sunil Kumar Manna					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	284065.00	Opening Balance	284065.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	284065.00		284065.00
284065.00	Excess of Expenditure over Income	284065.00	0.00	Closing Balance	0.00
284065.00		284065.00	284065.00		284065.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-52: "Nucleo Cytoplasmic transport of HIV - 1 Vpr"					
P.I: Dr Mahalingam & Dr Manna					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	1231118.00	Opening Balance	1231118.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1231118.00		1231118.00
1231118.00	Excess of Expenditure over Income	1231118.00	0.00	Closing Balance	0.00
1231118.00		1231118.00	1231118.00		1231118.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-54: "Study of viability of Mycobacterium leprae in clinical samples and possibility of its presence in the environment using nucleic acid amplification techniques."					
P.I: Dr Niyaz Ahmed					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
0.00	Opening Balance	0.00	37877.00	Opening Balance	37877.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	37877.00		37877.00
37877.00	Excess of Expenditure over Income	37877.00	0.00	Closing Balance	0.00
37877.00		37877.00	37877.00		37877.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-55: "Identification of DNA Markers for baculovirus resistance in silkworm, Bombyx mori"					
P.I: Dr J Nagaraju					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
224.00	Opening Balance	224.00	0.00	Salaries - Manpower	0.00
0.00	Grant In Aid	0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
224.00		224.00	0.00		0.00
0.00	Excess of Expenditure over Income	0.00	224.00	Closing Balance	224.00
224.00		224.00	224.00		224.00

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-56: "Genetics of transcription-replication interplay and of stress adaptation in bacteria" P.I: Dr Gowrishankar & Dr K Anupama Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>						
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount	
Rs		Rs.	Rs		Rs	
0.00	Opening Balance	0.00	1231164.00	Opening Balance	1231164.00	
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00	
0.00		0.00	0.00	Consumables	0.00	
0.00		0.00	0.00	Contingencies	0.00	
0.00		0.00	0.00	Travel	0.00	
0.00		0.00	0.00	Overheads	0.00	
0.00		0.00	0.00	Equipment	0.00	
0.00		0.00	0.00	Books	0.00	
0.00		0.00	0.00	AMC	0.00	
0.00		0.00	0.00	Others	0.00	
0.00		0.00	0.00	Transfer of Funds	0.00	
0.00		0.00	1231164.00		1231164.00	
1231164.00	Excess of Expenditure over Income	1231164.00	0.00	Closing Balance	0.00	
1231164.00		1231164.00	1231164.00		1231164.00	

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-57 : Improved genome annotation through a combination of machine learning and experimental methods: Plasmodium falciparum as a case study. P.I: Dr Akash Ranjan Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>						
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount	
Rs		Rs.	Rs		Rs	
0.00	Opening Balance	0.00	0.00	Opening Balance	0.00	
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00	
0.00		0.00	0.00	Consumables	0.00	
0.00		0.00	0.00	Contingencies	0.00	
0.00		0.00	0.00	Travel	0.00	
0.00		0.00	0.00	Overheads	0.00	
0.00		0.00	0.00	Equipment	0.00	
0.00		0.00	0.00	Books	0.00	
0.00		0.00	0.00	AMC	0.00	
0.00		0.00	0.00	Others	0.00	
0.00		0.00	0.00	Transfer of Funds	0.00	
0.00		0.00	0.00		0.00	
0.00	Excess of Expenditure over Income	0.00	0.00	Closing Balance	0.00	
0.00		0.00	0.00		0.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-59: "An integrated Approach towards understanding the biology of Mycobacterium tuberculosis: Genetic, biochemical, immunological and structural analyses."					
P.I: Dr Hasnain, Dr Gowrishankar, Dr Mande, Dr Ranjan Sen					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	2215024.00	Opening Balance	2215024.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	2215024.00		2215024.00
2215024.00	Excess of Expenditure over Income	2215024.00	0.00	Closing Balance	0.00
2215024.00		2215024.00	2215024.00		2215024.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-60: "National Database of Prevalent Genetic Disorders in India: Development, Curation and Services"					
P.I: Dr H A Nagarajaram					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
482124.00	Opening Balance	482124.00	0.00	Salaries - Manpower	0.00
0.00	Grant In Aid	0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
482124.00		482124.00	0.00		0.00
0.00	Excess of Expenditure over Income	0.00	482124.00	Closing Balance	482124.00
482124.00		482124.00	482124.00		482124.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-61: "Dissection of a novel phenotype of lethal accumulation of potassium in Escherichia coli mutants defective in thioredoxin/thioredoxin reductase and nucleoid protein H-NS" PI: Dr Abhijit A Sardesai Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs	Rs	Rs	Rs	Rs	Rs
0.00	Opening Balance	0.00	280000.00	Opening Balance	280000.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	280000.00		280000.00
280000.00	Excess of Expenditure over Income	280000.00	0.00	Closing Balance	0.00
280000.00		280000.00	280000.00		280000.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-62: "HIV – 1 Pathogenesis: Role of Integrase in Reverse Transcription and Nuclear Transport of Viral Genome" PI: Dr S Mahalingam Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs	Rs	Rs	Rs	Rs	Rs
0.00	Opening Balance	0.00	278928.00	Opening Balance	278928.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	278928.00		278928.00
278928.00	Excess of Expenditure over Income	278928.00	0.00	Closing Balance	0.00
278928.00		278928.00	278928.00		278928.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-63: "Upgradation of the existing computing infrastructure at the Bioinformatics facility at CDFD"					
P.I: Dr Seyed E Hasnain					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs	Rs	Rs	Rs	Rs	Rs
0.00	Opening Balance	0.00	773874.00	Opening Balance	837574.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	773874.00		773874.00
773874.00	Excess of Expenditure over Income	773874.00	0.00	Closing Balance	0.00
773874.00		773874.00	773874.00		773874.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-64: Biotechnology for Leather: Towards cleaner processing phase-II					
P.I: Dr J Gowrishankar					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs	Rs	Rs	Rs	Rs	Rs
0.00	Opening Balance	0.00	158.00	Opening Balance	158.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	158.00		158.00
158.00	Excess of Expenditure over Income	158.00	0.00	Closing Balance	0.00
158.00		158.00	158.00		158.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-65: "Molecular, genetic and functional analysis of the chromosomal plasticity region of the gastric pathogen Helicobacter pylori"					
PI: Dr Ayesha Alvi					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	582647.00	Opening Balance	582647.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	582647.00		582647.00
582647.00	Excess of Expenditure over Income	582647.00	0.00	Closing Balance	0.00
582647.00		582647.00	582647.00		582647.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-65A: APEDA-CDFD Centre for Basmati DNA Analysis					
PI: Dr J Nagaraju					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
22811205.00	Opening Balance	23733305.00	69445.00	Opening Balance	0.00
1004370.00	Grant In Aid	910600.00	12825.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	119178.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
23815575.00		24643905.00	82270.00		119178.00
0.00	Excess of Expenditure Over Income	0.00	23733305.00	Closing Balance	24524727.00
23815575.00		24643905.00	23815575.00		24643905.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-66: Human Epigenome Variation: Analysis of CpG island methylation in chromosomes 18 and Y, and in some Hox, insulin signaling and chromatin reprogramming genes PI: Dr Sanjeev Khosia Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	681246.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			681246.00	
681246.00		Excess of Expenditure over Income	681246.00		Closing Balance	0.00	
681246.00			681246.00			681246.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-67: Identification of novel Esophageal Squamous cell carcinoma (ESCC) genes by using a combination of array-based CGH and gene expression micro arrays PI: Dr M D Bashyam Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	113545.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			113545.00	
113545.00		Excess of Expenditure over Income	113545.00		Closing Balance	0.00	
113545.00			113545.00			113545.00	

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-68: Identification of High risk individual with pre-cancerous states of esophageal cancer. P.I: Dr Gayatri Ramakrishna Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
0.00	Opening Balance	0.00	59874.00	Opening Balance	59874.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	59874.00		59874.00
59874.00	Excess of Expenditure over Income	59874.00	0.00	Closing Balance	0.00
59874.00		59874.00	59874.00		59874.00

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-70: Identification of disease causing mutations in familial hypertrophic cardiomyopathy (FHC) patients from Andhra Pradesh P.I: Dr M D Bashyam Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
0.00	Opening Balance	0.00	21336.00	Opening Balance	21336.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	21336.00		21336.00
21336.00	Excess of Expenditure over Income	21336.00	0.00	Closing Balance	0.00
21336.00		21336.00	21336.00		21336.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-72: Nuances of non coding DNA near insulin-responsive genes. P.I: Dr Nirmala Yabaluri Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount Rs.	Previous Year Amount Rs.	Payments	Current Year Amount Rs.
0.00	Opening Balance	0.00	1421653.00	Opening Balance	1421653.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1421653.00		1421653.00
1421653.00	Excess of Expenditure over Income	1421653.00	0.00	Closing Balance	0.00
1421653.00		1421653.00	1421653.00		1421653.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-73: Identification and characterization of pancreatic cancer genes located within novel localized copy number alterations P.I: Dr M D Bashyam Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount Rs.	Previous Year Amount Rs.	Payments	Current Year Amount Rs.
0.00	Opening Balance	0.00	857136.00	Opening Balance	857136.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	857136.00		857136.00
857136.00	Excess of Expenditure over Income	857136.00	0.00	Closing Balance	0.00
857136.00		857136.00	857136.00		857136.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-75: Preparing blueprint for the macromolecular crystallography beamline at Indus-II synchrotron source					
P.I: Dr Sekhar C Mande					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	10840.00	Opening Balance	10840.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	10840.00		10840.00
10840.00	Excess of Expenditure over Income	10840.00	0.00	Closing Balance	0.00
10840.00		10840.00	10840.00		10840.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-76: A study of molecular markers in childhood autism with special references to nuclear factors - ± APPA B					
P.I: Dr S K Manna					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	50234.00	Opening Balance	50234.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	50234.00		50234.00
50234.00	Excess of Expenditure over Income	50234.00	0.00	Closing Balance	0.00
50234.00		50234.00	50234.00		50234.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-77: Functional characterization of Mycobacterium tuberculosis PE/PPE proteins having SH3 binding domain : Understanding their role in modulating macrophage functions P.I: Dr Sangita Mukhopadhyay Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
124277.00	Opening Balance	124277.00	0.00	Salaries - Manpower	0.00
0.00	Grant In Aid	0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
124277.00		124277.00	0.00		0.00
0.00	Excess of Expenditure over Income	0.00	124277.00	Closing Balance	124277.00
124277.00		124277.00	124277.00		124277.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-78: Task force- IMD Newborn screening for Congenital Hypothyroidism & Congenital Adrenal Hyperplasia: A multicentric study P.I: Dr A Radha Rama Devi Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
1304.00	Opening Balance	1304.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1304.00		1304.00	0.00		0.00
0.00	Excess of Expenditure over Income	0.00	1304.00	Closing Balance	1304.00
1304.00		1304.00	1304.00		1304.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-79: Understanding the role of AGE proteins in inducing inflammatory responses and its regulation PI: Dr S K Manna Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	105086.00	Opening Balance	105086.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	105086.00		105086.00
105086.00	Excess of Expenditure Over Income	105086.00	0.00	Closing Balance	0.00
105086.00		105086.00	105086.00		105086.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-80: Referral centre for detection of genetically modified foods employing DNA-based markets PI: Dr Madhusudan Reddy Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	608222.00	Opening Balance	608222.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
			0.00	Consumables	0.00
			0.00	Contingencies	0.00
			0.00	Travel	0.00
			0.00	Overheads	0.00
			0.00	Equipment	0.00
			0.00	Books	0.00
			0.00	AMC	0.00
			0.00	Others	0.00
			608222.00	Transfer of Funds	0.00
0.00		0.00	608222.00		608222.00
608222.00	Excess of Expenditure over Income	608222.00	0.00	Closing Balance	0.00
608222.00		608222.00	608222.00		608222.00

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-81: Reconstructing Cellular Networks: Two-component regulatory systems P.I: Dr Shekhar Mande Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
143470.00		Opening Balance	143470.00		Salaries - Manpower		0.00
0.00		Grant In Aid	0.00		Consumables		0.00
0.00			0.00		Contingencies		0.00
0.00			0.00		Travel		0.00
0.00			0.00		Overheads		0.00
0.00			0.00		Equipment		0.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
143470.00			143470.00				0.00
0.00		Excess of Expenditure over Income	0.00		Closing Balance		143470.00
143470.00			143470.00				143470.00

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-81A: Financial assistance for award of J C Bose Fellowship to Dr J Gowrishankar P.I: Dr J Gowrishankar Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
2620.00		Opening Balance	850453.00		Opening Balance		0.00
1360000.00		Grant In Aid	0.00		Salaries - Manpower		50000.00
0.00			0.00		Consumables		800453.00
0.00			0.00		Contingencies		0.00
0.00			0.00		Travel		0.00
0.00			0.00		Overheads		60000.00
0.00			0.00		Equipment		0.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
1362620.00			850453.00				910453.00
0.00		Excess of Expenditure Over Income	60000.00		Closing Balance		0.00
1362620.00			910453.00				910453.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-83: Prokaryotic Transcription termination factor, Rho: Mechanism of Action and Biology					
P.I: Dr Ranjan Sen					
Receipts and Payments Account from 01/04/2016 to 31/03/2017					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
0.00	Opening Balance	0.00	1155594.00	Opening Balance	1155594.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1155594.00		1155594.00
1155594.00	Excess of Expenditure over Income	1155594.00	0.00	Closing Balance	0.00
1155594.00		1155594.00	1155594.00		1155594.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-84: Preparing for vaccine efficacy trials: Baseline epidemiology, improved diagnosis, markers of protection and phase I/II trials					
P.I: Dr Niyaz Ahmed					
Receipts and Payments Account from 01/04/2016 to 31/03/2017					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
0.00	Opening Balance	0.00	1150.00	Opening Balance	1150.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1150.00		1150.00
1150.00	Excess of Expenditure over Income	1150.00	0.00	Closing Balance	0.00
1150.00		1150.00	1150.00		1150.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-82: Functional genomic analysis of Candida Glabrata-macrophage					
P.I: Dr Rupinder Kaur					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	367721.00	Opening Balance	369021.00
0.00	Grant In Aid	0.00	1300.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	369021.00		369021.00
369021.00	Excess of Expenditure Over Income	369021.00	0.00	Closing Balance	0.00
369021.00		369021.00	369021.00		369021.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-83 : Prokaryotic Transcription termination factor, Rho: Mechanism of Action and Biology					
P.I: Dr Ranjan Sen r					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	1155594.00	Opening Balance	1155594.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1155594.00		1155594.00
1155594.00	Excess of Expenditure Over Income	1155594.00	0.00	Closing Balance	0.00
1155594.00		1155594.00	1155594.00		1155594.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-84 : Preparing for vaccine efficacy trials: Baseline epidemiology, improved diagnosis, markers of protection and phase I/II trials P.I: Dr Niyaz Ahmed Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	1150.00	Opening Balance	1150.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1150.00		1150.00
1150.00	Excess of Expenditure Over Income	1150.00	0.00	Closing Balance	0.00
1150.00		1150.00	1150.00		1150.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-84A : Human epigenetic to the rescue of human identification process: Enriching human DNA from DNA mixture employing antibodies directed against 5-methylcytosine followed by whole genome amplification P.I: Dr Madhusudan Reddy Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	106479.00	Opening Balance	106479.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	106479.00		106479.00
106479.00	Excess of Expenditure Over Income	106479.00	0.00	Closing Balance	0.00
106479.00		106479.00	106479.00		106479.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-85: IdeR associated gene regulatory network in mycobacteria P.I: Dr Akash Ranjan Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	1118755.00	Opening Balance	1118755.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1118755.00		1118755.00
1118755.00	Excess of Expenditure Over Income	1118755.00	0.00	Closing Balance	0.00
1118755.00		1118755.00	1118755.00		1118755.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-87: Comparative genomics of wild silkmoths P.I: Dr J Nagaraju Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	65698.00	Opening Balance	65698.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	65698.00		65698.00
65698.00	Excess of Expenditure over Income	65698.00	0.00	Closing Balance	0.00
65698.00		65698.00	65698.00		65698.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-90: Role of Yapsins in the Pathobiology of Candida Glabrata P.I: Dr Rupinder Kaur Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year. Amount	Payments	Current Year Amount
0.00	Opening Balance	0.00	636286.00	Opening Balance	636286.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	636286.00		636286.00
636286.00	Excess of Expenditure over Income	636286.00	0.00	Closing Balance	0.00
636286.00		636286.00	636286.00		636286.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-91: DM1T3L: epigenetic correlation with cancer P.I: Dr Sanjeev Khosla Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year. Amount	Payments	Current Year Amount
0.00	Opening Balance	0.00	1098900.00	Opening Balance	1098900.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1098900.00		1098900.00
1098900.00	Excess of Expenditure over Income	1098900.00	0.00	Closing Balance	0.00
1098900.00		1098900.00	1098900.00		1098900.00

P-92: Swarnajayanti fellowship proj on "Designing transcription anti-terminators: a novel approach for making new inhibitors of gene expression" CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P.I.: Dr Ranjan Sen Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	268823.00		0.00		Opening Balance		0.00
268823.00		Grant In Aid	0.00		0.00		Salaries - Manpower		0.00
0.00			0.00		0.00		Consumables		0.00
0.00			0.00		0.00		Contingencies		0.00
0.00			0.00		0.00		Travel		0.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		0.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		0.00
268823.00			268823.00		0.00				0.00
0.00		Excess of Expenditure Over Income	0.00		268823.00		Closing Balance		268823.00
268823.00			268823.00		268823.00				268823.00

P-93/A1 : Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against tuberculosis CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P.I.: Dr Shekar Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		611833.00		Opening Balance		611833.00
645000.00		Grant In Aid	0.00		0.00		Salaries - Manpower		0.00
0.00			0.00		0.00		Consumables		0.00
0.00			0.00		0.00		Contingencies		0.00
0.00			0.00		0.00		Travel		0.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		0.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		0.00
0.00			0.00		611833.00				611833.00
611833.00		Excess of Expenditure Over Income	611833.00		0.00		Closing Balance		0.00
611833.00			611833.00		611833.00				611833.00

P-93/A2 : Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P.I.: Dr. Sangita Mukhopadhyay Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		3038491.00		Opening Balance	3228626.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		190135.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			0.00		3228626.00			3228626.00	
3228626.00		Excess of Expenditure Over Income	3228626.00		0.00		Closing Balance	0.00	
3228626.00			3228626.00		3228626.00			3228626.00	

P-93B2 (II) : Evaluation of peptides / small molecules targeting ESAT-6:B2M interaction and PPE18-TLR2 interaction as potent anti tuberculosis therapeutics CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P.I.: Dr Sangita Mukhopadhyay Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
483835.00		Opening Balance	837745.00		261800.00		Opening Balance	0.00	
737000.00		Grant In Aid	816700.00		67467.00		Salaries - Manpower	482236.00	
0.00			0.00		30000.00		Consumables	199127.00	
0.00			0.00		23823.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	20802.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
1220835.00			1654445.00		383090.00			702165.00	
0.00		Excess of Expenditure Over Income	0.00		837745.00		Closing Balance	952280.00	
1220835.00			1654445.00		1220835.00			1654445.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-97: Proteome-wide Analysis of Serine pyrophosphorylation by inositol pyrophosphates					
P.I: Dr Rashna Bhandari					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	276552.00	Opening Balance	276552.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	276552.00		276552.00
276552.00	Excess of Expenditure Over Income	276552.00	0.00	Closing Balance	0.00
276552.00		276552.00	276552.00		276552.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-98: Role of cell - cell signaling mediated by Diffusible signaling factor (DSF) in Xanthomonas virulence					
P.I: Dr Subhadeep Chatterjee					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	236042.00	Opening Balance	236042.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	236042.00		236042.00
236042.00	Excess of Expenditure Over Income	236042.00	0.00	Closing Balance	0.00
236042.00		236042.00	236042.00		236042.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-99: Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosome biogenesis P.I: Dr Rashna Bhandari Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs	Rs	Rs.	Rs	Rs	Rs
0.00	Opening Balance	0.00	567516.00	Opening Balance	567516.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	567516.00		567516.00
567516.00	Excess of Expenditure Over Income	567516.00	0.00	Closing Balance	0.00
567516.00		567516.00	567516.00		567516.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-100: Effect of reactive oxygen species on T-Cell immune response: An approach to understand the molecular mechanism of immunosuppression during tuberculosis - National Bioscience Award P.I: Dr Sangita Mukhopadhyay Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs	Rs	Rs.	Rs	Rs	Rs
0.00	Opening Balance	0.00	576590.00	Opening Balance	576590.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	576590.00		576590.00
576590.00	Excess of Expenditure Over Income	576590.00	0.00	Closing Balance	0.00
576590.00		576590.00	576590.00		576590.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-102: "Understanding the role of Mycobacterium tuberculosis heat shockprotein 60 as Th1/Th2 immuno modular" PI: Dr Sangita Mukhopadhyay Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	27922.00	Opening Balance	27922.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	27922.00		27922.00
27922.00	Excess of Expenditure Over Income	27922.00	0.00	Closing Balance	0.00
27922.00		27922.00	27922.00		27922.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-103: National Bioscience Award - Regulation of mast cell signaling, apoptosis and surface receptors PI: Dr Sunil Kumar Manna Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	300000.00	Opening Balance	300000.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	300000.00		300000.00
300000.00	Excess of Expenditure Over Income	300000.00	0.00	Closing Balance	0.00
300000.00		300000.00	300000.00		300000.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-104: Virtual Centre of Excellence on Epigenetics PI: Dr Sanjeev Khosla Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1289897.00		Opening Balance	1289897.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			0.00		1289897.00			1289897.00	
1289897.00		Excess of Expenditure Over Income	1289897.00		0.00		Closing Balance	0.00	
1289897.00			1289897.00		1289897.00			1289897.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-105: Cloning, Characterization and analysis of chromosomal rearrangements in human genetic disorders PI: Dr Ashwin B Dalal Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		844946.00		Opening Balance	862685.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			0.00		862685.00			862685.00	
862685.00		Excess of Expenditure Over Income	862685.00		0.00		Closing Balance	0.00	
862685.00			862685.00		862685.00			862685.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-107: DBT IYBA Project on "Mechanism and role of bacterial cell-cell signaling molecules in plant defense response"					
P.I: Dr Subhadeep Chatterjee					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
1036691.00	Opening Balance	366575.00	70166.00	Opening Balance	39000.00
0.00	Grant In Aid	0.00	589798.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	10202.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1036691.00		366575.00	670116.00		39000.00
0.00	Excess of Expenditure Over Income	0.00	366575.00	Closing Balance	327575.00
1036691.00		366575.00	1036691.00		366575.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-108: Establishment of EBV transformed cell lines from families with rare genetic disorders					
P.I: Dr Ashwin B Dalal					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	454643.00	Opening Balance	454643.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	454643.00		454643.00
454643.00	Excess of Expenditure Over Income	454643.00	0.00	Closing Balance	0.00
454643.00		454643.00	454643.00		454643.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-109: Molecular dissection of PI3-Kinase/Akt pathway by using proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors							
P.I: Dr M Subba Reddy							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
767943.00		Opening Balance	0.00		Opening Balance	362393.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	235174.00	
0.00			0.00		Consumables	630855.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.000.00	
0.00			0.00		Transfer of Funds	0.00	
767943.00			0.00			1130336.00	
362393.00		Excess of Expenditure Over Income	1228422.00		Closing Balance	0.00	
1130336.00			1228422.00			1228422.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-110: India-Japan research project title "Identification and analysis of sex determining genes in silkworms"							
P.I: Dr J Nagaraju							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	19391.00	
172000.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
172000.00			0.00			19391.00	
19391.00		Excess of Expenditure Over Income	19391.00		Closing Balance	0.00	
191391.00			19391.00			19391.00	

P-114: Evaluating the Calcineurin-NFAT Pathway and its regulators superoxide dismutase (SOD) AND RCAN1 (regular of Calcineurin) Down Syndrome CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD PI: Dr Gayatri Ramakrishna, Dr Ashwin Dalal Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	450859.00	Opening Balance	450859.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	450859.00		450859.00
450859.00	Excess of Expenditure Over Income	450859.00	0.00	Closing Balance	0.00
450859.00		450859.00	450859.00		450859.00

P-116: DBT-India and AIST - Japan : Understanding molecular mechanisms controlling dual role of Ras, Sirtuins and CARF in relation to cellular proliferation and senescence: Novel Strategy for developing cancer therapeutics CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD PI: Dr Gayatri Ramakrishna Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	1251366.00	Opening Balance	1251366.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1251366.00		1251366.00
1251366.00	Excess of Expenditure Over Income	1251366.00	0.00	Closing Balance	0.00
1251366.00		1251366.00	1251366.00		1251366.00

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-119: Analysis of DNA copy number alterations in esophageal cancer P.I: Dr M D Bashyam Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		2892.00		Opening Balance	2892.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			0.00		2892.00			2892.00	
2892.00		Excess of Expenditure Over Income	2892.00		0.00		Closing Balance	0.00	
2892.00			2892.00		2892.00			2892.00	

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-120: Effect of reactive oxygen species on macrophage signalosome: impact on antigen presentation functions and T Cell priming responses P.I: Dr Sangita Mukhopadhyay Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		769484.00		Opening Balance	769484.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			0.00		769484.00			769484.00	
769484.00		Excess of Expenditure Over Income	769484.00		0.00		Closing Balance	0.00	
769484.00			769484.00		769484.00			769484.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-121: Identification and characterization of PTEN regulators P.I: Dr M Subba Reddy Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1130866.00		Opening Balance	1130866.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			0.00		1130866.00			1130866.00	
1130866.00		Excess of Expenditure Over Income	1130866.00		0.00		Closing Balance	0.00	
1130866.00			1130866.00		1130866.00			1130866.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-122: Understanding the role of Hox genes in anterior-posterior axis determination of the central nervous system P.I: Dr Rohit Joshi Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
2951109.00		Opening Balance	21124.00		194574.00		Opening Balance	0.00	
2722184.00		Grant In Aid	3462961.00		3368228.00		Salaries - Manpower	328944.00	
0.00			0.00		3377.00		Consumables	2174165.00	
0.00			0.00		19369.00		Contingencies	0.00	
0.00			0.00		513833.00		Travel	332877.00	
0.00			0.00		1552788.00		Overheads	316734.00	
0.00			0.00		0.00		Equipment	331365.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
5673293.00			3484085.00		5652169.00			3484085.00	
0.00		Excess of Expenditure Over Income	0.00		21124.00		Closing Balance	0.00	
5673293.00			3484085.00		5673293.00			3484085.00	

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-123: Establish a Max Planck Partner Group for Genetic Diversity Studies at CDFD P.I: Dr N Madhusudan Reddy Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
771699.00	Opening Balance	1440687.00	199277.00	Opening Balance	0.00
1648000.00	Grant In Aid	0.00	428574.00	Salaries - Manpower	-151175.00
0.00		0.00	0.00	Consumables	132565.00
0.00		0.00	186183.00	Contingencies	0.00
0.00		0.00	0.00	Travel	88726.00
0.00		0.00	164978.00	Overheads	0.00
0.00		0.00	0.00	Equipment	258013.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2419699.00		1440687.00	979012.00		328129.00
0.00	Excess of Expenditure Over Income	0.00	1440687.00	Closing Balance	1112558.00
2419699.00		1440687.00	2419699.00		1440687.00

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-124: Preparation and characterization of peroxometal compounds and studies and their biological significance in cellular signalling P.I: Dr Gayatri Ramakrishna Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	748411.00	Opening Balance	748411.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	748411.00		748411.00
748411.00	Excess of Expenditure Over Income	748411.00	0.00	Closing Balance	0.00
748411.00		748411.00	748411.00		748411.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-126: Rho-dependent transcription termination machinery: mechanism of action					
Pi: Dr Ranjan Sen					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
209670.00	Opening Balance	160270.00	49400.00	Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
209670.00		160270.00	49400.00		0.00
0.00	Excess of Expenditure Over Income	0.00	160270.00	Closing Balance	160270.00
209670.00		160270.00	209670.00		160270.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-127: Systematic studies on the functional network of phosphatases in cell life and death					
Pi: Dr M Subba Reddy					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
1895283.00	Opening Balance	0.00	144000.00	Opening Balance	0.00
663747.00	Grant In Aid	0.00	2182390.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	232640.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2559030.00		0.00	2559030.00		0.00
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	0.00
2559030.00		0.00	2559030.00		0.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-128: Mechanism of iron acquisition and iron homeostasis in an opportunistic human pathogen Candida glabrata P.I: Dr Rupinder Kaur Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	158488.00	Opening Balance	158488.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	158488.00		158488.00
158488.00	Excess of Expenditure Over Income	158488.00	0.00	Closing Balance	0.00
158488.00		158488.00	158488.00		158488.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-130: Comparative genetic analysis of sex chromosomes and sex determining genes in silkworms P.I: Dr J Nagaraju Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
869.00	Opening Balance	0.00	125471.00	Opening Balance	142258.00
0.00	Grant In Aid	0.00	17656.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
869.00		0.00	143127.00		142258.00
142258.00	Excess of Expenditure Over Income	142258.00	0.00	Closing Balance	0.00
143127.00		142258.00	143127.00		142258.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-131: Structural and functional studies of Acyl CoA Binding proteins from Plasmodium falciparum					
P.I: Dr Akash Ranjan					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
398632.00	Opening Balance	398632.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
398632.00		398632.00	0.00		0.00
0.00	Excess of Expenditure Over Income	0.00	398632.00	Closing Balance	398632.00
398632.00		398632.00	398632.00		398632.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-132: Characterization of tumor suppressor function of ARID1B, a component of the human SWI/SNF chromatin remodelling complex					
P.I: Dr M D Bashyam, Dr Rohit Joshi					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	12199.00	Opening Balance	12199.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	12199.00		12199.00
12199.00	Excess of Expenditure Over Income	12199.00	0.00	Closing Balance	0.00
12199.00		12199.00	12199.00		12199.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-133: Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster PI: Dr Rohit Joshi Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	1324223.00	
500000.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
500000.00			0.00			1324223.00	
1324223.00		Excess of Expenditure Over Income	1324223.00		Closing Balance	0.00	
1824223.00			1324223.00			1324223.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-134: Exploration of wild silk moth biodiversity in Manipur and their genetic characterization using molecular markers PI: Dr K P Arun Kumar Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	77061.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			77061.00	
77061.00		Excess of Expenditure Over Income	77061.00		Closing Balance	0.00	
77061.00			77061.00			77061.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-135: Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Pathogen Interaction in TB Infection									
P.I: Dr. Sanjeev Kholisa									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		336135.00		Opening Balance	1118756.00	
0.00		Grant In Aid	2424800.00		343200.00		Salaries - Manpower	114400.00	
0.00			0.00		423237.00		Consumables	1276819.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		16184.00		Travel	86364.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			2424800.00		1118756.00			2596339.00	
1118756.00		Excess of Expenditure Over Income	171539.00		0.00		Closing Balance	0.00	
1118756.00			2596339.00		1118756.00			2596339.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-136: Raf Kinase - a key target for modern-day therapy against tumors									
P.I: Dr Sunil Kumar Manna									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		196001.00		Opening Balance	196001.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			0.00		196001.00			196001.00	
196001.00		Excess of Expenditure Over Income	196001.00		0.00		Closing Balance	0.00	
196001.00			196001.00		196001.00			196001.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-138: Co-evaluation of Dnmt31 and Genomic imprinting P.I: Dr Sanjeev Khosla Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1500300.00		Opening Balance	1451500.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		-48800.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			0.00		1451500.00			1451500.00	
1451500.00		Excess of Expenditure Over Income	1451500.00		0.00		Closing Balance	0.00	
1451500.00			1451500.00		1451500.00			1451500.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-139: Evaluating the role of Sirtuins and epigenetic changes during cellular senescence in context of p53 status P.I: Dr Gayatri Ramakrishna, Dr Sanjeev Khosla Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
20000.00		Opening Balance	20000.00		0.00		Opening Balance	0.00	
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
20000.00			20000.00		0.00			0.00	
0.00		Excess of Expenditure Over Income	0.00		20000.00		Closing Balance	20000.00	
20000.00			20000.00		20000.00			20000.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-140: Development of baculovirus resistant silkworms strains through synthetic miRNA based knockdown of essential viral genes					
P.I: Dr K P Arun Kumar					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	608652.00	Opening Balance	608652.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	608652.00		608652.00
608652.00	Excess of Expenditure Over Income	608652.00	0.00	Closing Balance	0.00
608652.00		608652.00	608652.00		608652.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-141: Evaluating the functional role of PTEN interacting proteins in cell survival signaling and tumor suppression					
P.I: Dr M Subba Reddy					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	125000.00	Opening Balance	125000.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	125000.00		125000.00
125000.00	Excess of Expenditure Over Income	125000.00	0.00	Closing Balance	0.00
125000.00		125000.00	125000.00		125000.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-142: Identification of H3K4 TRI Demethylase involved in erasing H3K4 trimethylation marks at E2F Responsive promoters					
P.I: Dr Shweta Tyagi					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	81861.00	Opening Balance	81861.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	81861.00		81861.00
81861.00	Excess of Expenditure Over Income	81861.00	0.00	Closing Balance	0.00
81861.00		81861.00	81861.00		81861.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-143: Microarray based characterisation of squamous cell carcinoma of the tongue occurring in non smokers					
P.I: Dr M D Bashyam					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	1381684.00	Opening Balance	719139.00
662545.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
662545.00		0.00	1381684.00		719139.00
719139.00	Excess of Expenditure Over Income	719139.00	0.00	Closing Balance	0.00
1381684.00		719139.00	1381684.00		719139.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-144 : Tri-National Training Program for Psychiatric Genetics Pl: Dr Ashwin B Dalal Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
122130.00	Opening Balance	122130.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
122130.00		122130.00	0.00		0.00
0.00	Excess of Expenditure Over Income	0.00	122130.00	Closing Balance	122130.00
122130.00		122130.00	122130.00		122130.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-145: H3K4 HMT family regulatescell cycle progression Pl: Dr Shweta Tyagi Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
3222.00	Opening Balance	3222.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
3222.00		3222.00	0.00		0.00
0.00	Excess of Expenditure Over Income	0.00	3222.00	Closing Balance	3222.00
3222.00		3222.00	3222.00		3222.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-146: Role of MILL in ribosomal RNA transcription P.I: Dr Shweta Tyagi Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs	Rs	Rs.	Rs	Rs	Rs
59533.00	Opening Balance	59533.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
59533.00	Excess of Expenditure Over Income	59533.00	0.00	Closing Balance	0.00
59533.00		59533.00	59533.00		59533.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-147: The Effect of Parental Education, Ethics of Research Participation and Array Comparative Genomic Hybridization in Subjects with Mental Retardation (MR) and /or Autism P.I: Dr Ashwin B Dalal Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs	Rs	Rs.	Rs	Rs	Rs
0.00	Opening Balance	0.00	272874.00	Opening Balance	272874.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00	Excess of Expenditure Over Income	0.00	272874.00	Closing Balance	272874.00
272874.00		272874.00	0.00		0.00
272874.00		272874.00	272874.00		272874.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-148 : Transcriptional regulation of novel tumor suppressor genes in Pancreatic Cancer					
P.I: Dr K Jayaprakash Narayana					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	0.00		0.00
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	0.00
0.00		0.00	0.00		0.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-149: Role of SUMOylation in the pathobiology of Candida Glabrata					
P.I: Dr Rupinder Kaur					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	59917.00	Opening Balance	73001.00
0.00	Grant In Aid	0.00	13084.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	73001.00		73001.00
73001.00	Excess of Expenditure Over Income	73001.00	0.00	Closing Balance	0.00
73001.00		73001.00	73001.00		73001.00

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-151: Human Exome Sequencing to Identify Novel Genes for Medelian Disorders PI: Dr Ashwin B Dalal Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
375851.00		Opening Balance	199137.00		28600.00		Opening Balance		0.00
0.00		Grant In Aid	0.00		148114.00		Salaries - Manpower		0.00
0.00			0.00		0.00		Consumables		0.00
0.00			0.00		0.00		Contingencies		0.00
0.00			0.00		0.00		Travel		0.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		0.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		0.00
375851.00			199137.00		176714.00				0.00
0.00		Excess of Expenditure Over Income	0.00		199137.00		Closing Balance		199137.00
375851.00			199137.00		375851.00				199137.00

<p style="text-align: center;">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-152 : Global transcriptomics of sex specific splicing PI: Dr K P Arun Kumar Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		30814.00		Opening Balance		1123979.00
0.00		Grant In Aid	587717.00		483433.00		Salaries - Manpower		0.00
0.00			0.00		592311.00		Consumables		35975.00
0.00			0.00		0.00		Contingencies		0.00
0.00			0.00		0.00		Travel		0.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		17421.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		0.00
0.00			587717.00		1123979.00				1159954.00
1123979.00		Excess of Expenditure Over Income	572237.00		0.00		Closing Balance		0.00
1123979.00			1159954.00		1123979.00				1159954.00

<p align="center">P-153: An attractive and promising strategy for early cancer diagnosis through the assembly of the human cancer volatome” Centre for DNA Fingerprinting and Diagnostics, Hyderabad P.I: Dr H A Nagarajaram Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	1161773.00		Opening Balance	64305.00	
1787000.00		Grant In Aid	0.00		Salaries - Manpower	296400.00	23400.00
0.00			0.00		Consumables	0.00	0.00
0.00			0.00		Contingencies	6049.00	0.00
0.00			0.00		Travel	52330.00	0.00
0.00			0.00		Overheads	0.00	0.00
0.00			0.00		Equipment	0.00	0.00
0.00			0.00		Books	0.00	0.00
0.00			0.00		AMC	0.00	0.00
0.00			0.00		Others	206143.00	0.00
0.00			0.00		Transfer of Funds	0.00	0.00
1787000.00			1161773.00			625227.00	23400.00
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	1161773.00	1138373.00
1787000.00			1161773.00			1787000.00	1161773.00

<p align="center">P-154 : Rational design, synthetic strategies for developing organometallic anticancer compounds based on organotin and organoiron Centre for DNA Fingerprinting and Diagnostics, Hyderabad P.I: Dr Sunil Kumar Manna Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
13510.00		Opening Balance	0.00		Opening Balance	434393.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	0.00
0.00			0.00		Consumables	432806.00	42357.00
0.00			0.00		Contingencies	0.00	0.00
0.00			0.00		Travel	0.00	0.00
0.00			0.00		Overheads	0.00	0.00
0.00			0.00		Equipment	0.00	0.00
0.00			0.00		Books	0.00	0.00
0.00			0.00		AMC	0.00	0.00
0.00			0.00		Others	0.00	0.00
0.00			0.00		Transfer of Funds	0.00	0.00
13510.00			0.00			447903.00	476750.00
434393.00		Excess of Expenditure Over Income	476750.00		Closing Balance	0.00	0.00
447903.00			476750.00			447903.00	476750.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-155: Studies on the cellular roles of calcium signalling proteins in Neurospora crassa P.I: Dr D P Kasbekar Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
335194.00	Opening Balance	335194.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
335194.00		335194.00	0.00		0.00
0.00	Excess of Expenditure Over Income	0.00	335194.00	Closing Balance	335194.00
335194.00		335194.00	335194.00		335194.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-156 : Targeting microbial quorum sensing to demonstrate potential application of cell-cell signaling molecules from Xanthomonas group of plant pathogen in disease control PI : Dr Subhadeep Chatterjee Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
239949.00	Opening Balance	0.00	82680.00	Opening Balance	605123.00
0.00	Grant In Aid	0.00	724735.00	Salaries - Manpower	0.00
0.00		0.00	24845.00	Consumables	238246.00
0.00		0.00	12812.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
239949.00		0.00	845072.00		843369.00
605123.00	Excess of Expenditure Over Income	843369.00	0.00	Closing Balance	0.00
845072.00		843369.00	845072.00		843369.00

P-157 : Identification of novel antifungal drug and delineation of drug resistance mechanisms in an opportunistic human fungal pathogen Candida glabrata PI : Dr Rupinder Kaur Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year		Receipts		Payments		Current Year	
Amount	Rs	Amount	Rs.	Amount	Rs	Amount	Rs
0.00		Opening Balance	124009.00	1361799.00	Opening Balance	0.00	0.00
1638000.00		Grant In Aid	0.00	109200.00	Salaries - Manpower	9058.00	9058.00
0.00			0.00	42992.00	Consumables	-171812.00	-171812.00
0.00			0.00	0.00	Contingencies	0.00	0.00
0.00			0.00	0.00	Travel	26.00	26.00
0.00			0.00	0.00	Overheads	0.00	0.00
0.00			0.00	0.00	Equipment	89396.00	89396.00
0.00			0.00	0.00	Books	0.00	0.00
0.00			0.00	0.00	AMC	0.00	0.00
0.00			0.00	0.00	Others	0.00	0.00
0.00			0.00	0.00	Transfer of Funds	197341.00	197341.00
1638000.00			124009.00	1513991.00		124009.00	124009.00
0.00		Excess of Expenditure Over Income	0.00	124009.00	Closing Balance	0.00	0.00
1638000.00			124009.00	1638000.00			124009.00

P-158 : Modulation of host immune responses by a PPE Protein of Mycobacterium tuberculosis: Understanding its role in host - pathogen cross-talk PI : Dr Sangita Mukhopadhyay Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year		Receipts		Payments		Current Year	
Amount	Rs	Amount	Rs.	Amount	Rs	Amount	Rs
0.00		Opening Balance	0.00	2575346.00	Opening Balance	168374.00	168374.00
2790992.00		Grant In Aid	0.00	187200.00	Salaries - Manpower	129496.00	129496.00
0.00			0.00	196820.00	Consumables	0.00	0.00
0.00			0.00	0.00	Contingencies	0.00	0.00
0.00			0.00	0.00	Travel	0.00	0.00
0.00			0.00	0.00	Overheads	0.00	0.00
0.00			0.00	0.00	Equipment	0.00	0.00
0.00			0.00	0.00	Books	0.00	0.00
0.00			0.00	0.00	AMC	0.00	0.00
0.00			0.00	0.00	Others	0.00	0.00
0.00			0.00	0.00	Transfer of Funds	0.00	0.00
2790992.00			0.00	2959366.00		297870.00	297870.00
168374.00		Excess of Expenditure Over Income	297870.00	0.00	Closing Balance	0.00	0.00
2959366.00			297870.00	2959366.00			297870.00

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-159 : Gene Targeting of microbial isolates to demonstrate potential plant growth promoting (PGP) traits by third generation sequencing PI : Dr Subhadeep Chatterjee Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	300000.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	198696.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			498696.00	
300000.00		Excess of Expenditure Over Income	498696.00		Closing Balance	0.00	
300000.00			498696.00			498696.00	

<p align="center">CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-160 : Understanding the role of novel adhesins of Xanthomonas oryzae PV oryzae in Virulence and colonization in Rice PI : Dr Subhadeep Chatterjee Receipts and Payments Account from 01/04/2017 to 31/03/2018</p>							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	147180.00	
0.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	162792.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			0.00			309972.00	
147180.00		Excess of Expenditure Over Income	309972.00		Closing Balance	0.00	
147180.00			309972.00			309972.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-162 : Characterization and design of inhibitors of Mycobacterium tuberculosis transcription					
PI : Dr Ranjan Sen					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs	Rs	Rs.	Rs	Rs	Rs
0.00	Opening Balance	0.00	1021767.00	Opening Balance	464167.00
699600.00	Grant In Aid	0.00	117000.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	339491.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	25000.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
699600.00		0.00	1163767.00		803658.00
464167.00	Excess of Expenditure Over Income	803658.00	0.00	Closing Balance	0.00
1163767.00		803658.00	1163767.00		803658.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-163 : Unravelling new functions for the H-NS family of proteins in Gram-negative bacterial pathogens					
PI : Dr J Gowrishankar					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs	Rs	Rs.	Rs	Rs	Rs
678659.00	Opening Balance	1530338.17	117000.00	Opening Balance	0.00
1483389.00	Grant In Aid	0.00	47378.00	Salaries - Manpower	230400.00
0.00		0.00	2229.00	Consumables	726570.00
0.00		0.00	465102.83	Contingencies	8000.00
0.00		0.00	0.00	Travel	318193.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2162048.00		1530338.17	631709.83		1283163.00
0.00	Excess of Expenditure Over Income	0.00	1530338.17	Closing Balance	247175.17
2162048.00		1530338.17	2162048.00		1530338.17

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-164 : A Yeast based screen for discovery of novel sirtuin inhibitors as anticancer agents PI : Dr Devyani Halder Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	29200.00	Opening Balance	29200.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	29200.00		29200.00
29200.00	Excess of Expenditure Over Income	29200.00	0.00	Closing Balance	0.00
29200.00		29200.00	29200.00		29200.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-165 : Identification and functional characterization of immune response genes in silkworms PI : Dr V V Satyavathi Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year Amount Rs	Payments	Current Year Amount Rs
1567830.00	Opening Balance	862906.00	297200.00	Opening Balance	0.00
0.00	Grant In Aid	0.00	407724.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	138041.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	1988.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1567830.00		862906.00	704924.00		140029.00
0.00	Excess of Expenditure Over Income	0.00	862906.00	Closing Balance	722877.00
1567830.00		862906.00	1567830.00		862906.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-166 : Sequencing analysis of transcriptome variants in early-onset sporadic rectal cancer					
PI : Dr M D Bashyam					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
35696.00	Opening Balance	0.00	354378.00	Opening Balance	368609.00
0.00	Grant In Aid	1359100.00	0.00	Salaries - Manpower	102658.00
0.00		0.00	0.00	Consumables	1022407.00
0.00		0.00	30850.00	Contingencies	1000.00
0.00		0.00	19077.00	Travel	2687.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
35696.00		1359100.00	404305.00		1497361.00
368609.00	Excess of Expenditure Over Income	138261.00	0.00	Closing Balance	0.00
404305.00		1497361.00	404305.00		1497361.00

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CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-167 : To elucidate the role of MLL complex in epigenetic specification of centromere					
PI : Dr Shweta Tyagi					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
569787.00	Opening Balance	780652.00	46800.00	Opening Balance	0.00
900000.00	Grant In Aid	0.00	617187.00	Salaries - Manpower	295416.00
0.00		0.00	0.00	Consumables	185016.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	25148.00	Travel	105490.00
0.00		0.00	0.00	Overheads	200000.00
0.00		0.00	0.00	Equipment	-72757.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	67487.00
1469787.00		780652.00	689135.00		780652.00
0.00	Excess of Expenditure Over Income	0.00	780652.00	Closing Balance	0.00
1469787.00		780652.00	1469787.00		780652.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-168 : A Search for nucleus -limited genes in Neurospora PI : Dr D P Kasbekar Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year. Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00		Opening Balance	161318.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	236913.00
0.00		0.00	161318.00	Consumables	23770.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	17553.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	161318.00		439554.00
161318.00	Excess of Expenditure Over Income	439554.00	0.00	Closing Balance	0.00
161318.00		439554.00	161318.00		439554.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-169 : Implementation of 3 year DNB Program in Medical Genetics by Department of Biotechnology in collaboration with National Board of Examination ag SGHR, NIBMG&CDFD PI : Dr J Gowrishankar Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year. Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
16915.00	Opening Balance	0.00		Opening Balance	332017.00
2535600.00	Grant In Aid	3858700.00	2529290.00	Salaries - Manpower	3433548.00
0.00		0.00	55242.00	Consumables	110700.00
0.00		0.00	0.00	Contingencies	25000.00
0.00		0.00	300000.00	Travel	300000.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
2552515.00		3858700.00	2884532.00		4201265.00
332017.00	Excess of Expenditure Over Income	342565.00	0.00	Closing Balance	0.00
2884532.00		4201265.00	2884532.00		4201265.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-170 : Women Scientist Scheme "Identification and character of deregulated micro RNAs in defined sub-set of early onset sporadic rectal cancer patients using transcriptome sequencing"							
PI : Dr Mithu Ray Chaudhuri							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	383863.00	
1100000.00		Grant In Aid	0.00		Salaries - Manpower	275000.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
1100000.00			0.00			658863.00	
383863.00		Excess of Expenditure Over Income	658863.00		Closing Balance	0.00	
1483863.00			658863.00			658863.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-171 : Role of vesicle-mediated transport and chromatin remodelling in the virulence of Candida glabrata							
PI : Dr Rupinder Kaur							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
211423.00		Opening Balance	0.00		Opening Balance	1237535.00	
0.00		Grant In Aid	3533564.00		Salaries - Manpower	502987.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	46000.00	
0.00			0.00		Travel	40149.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	110741.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
211423.00			3533564.00			1937412.00	
1237535.00		Excess of Expenditure Over Income	0.00		Closing Balance	1596152.00	
1448958.00			3533564.00			3533564.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-172 : Molecular Characterization of early onset sporadic rectal cancer							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
PI : Dr M D Bashyam							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
111850.00		Opening Balance	40020.00		Opening Balance		0.00
1000000.00		Grant In Aid	800000.00		Salaries - Manpower	235510.00	
0.00			0.00		Consumables	475262.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
1111850.00			840020.00			710772.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	129248.00	
1111850.00			840020.00			840020.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-173 : Development and application of a next generation sequencing approach for molecular genetic analysis of lysosomal storage disorders							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
PI : Dr Ashwin B Dalal							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
487953.00		Opening Balance	1672130.00		Opening Balance		0.00
2107380.00		Grant In Aid	355990.00		Salaries - Manpower	794702.00	
0.00			0.00		Consumables	214980.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
2595333.00			2028120.00			1009682.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	1018438.00	
2595333.00			2028120.00			2028120.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-174 : Is non-canonical Wnt signalling a major player in early-onset sporadic rectal cancer					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
PI : Dr M D Bashyam					
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments
Previous Year Amount	Rs	Previous Year Amount	Rs	Current Year Amount	Rs
520542.00	0.00	Opening Balance	209406.00	0.00	Opening Balance
0.00	0.00	Grant In Aid	500000.00	0.00	Salaries - Manpower
0.00	0.00		0.00	37716.00	Consumables
0.00	0.00		0.00	0.00	Contingencies
0.00	0.00		0.00	0.00	Travel
0.00	0.00		0.00	0.00	Overheads
0.00	0.00		0.00	0.00	Equipment
0.00	0.00		0.00	0.00	Books
0.00	0.00		0.00	0.00	AMC
0.00	0.00		0.00	0.00	Others
0.00	0.00		0.00	0.00	Transfer of Funds
520542.00	0.00		709406.00	0.00	
		Excess of Expenditure Over Income	0.00	209406.00	Closing Balance
520542.00			709406.00	520542.00	
					250087.00
					459319.00
					709406.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-175 : Multi Centri Collaborative study of the Clinical, Biochemical and Molecular Characterization of Lysosomal storage disorders in India - The initiative for research in Lysosomal Storage Disorders"					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
PI : Dr Ashwin B Dalal					
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments
Previous Year Amount	Rs	Previous Year Amount	Rs	Current Year Amount	Rs
0.00	0.00	Opening Balance	1432672.00	0.00	Opening Balance
2214648.00	0.00	Grant In Aid	541200.00	363913.00	Salaries - Manpower
0.00	0.00		345462.00	0.00	Consumables
0.00	0.00		0.00	0.00	Contingencies
0.00	0.00		16983.00	0.00	Travel
0.00	0.00		0.00	0.00	Overheads
0.00	0.00		0.00	0.00	Equipment
0.00	0.00		0.00	0.00	Books
0.00	0.00		0.00	0.00	AMC
0.00	0.00		0.00	0.00	Others
0.00	0.00		0.00	0.00	Transfer of Funds
2214648.00	0.00		2336317.00	363913.00	
121669.00	0.00	Excess of Expenditure Over Income	898484.00	0.00	Closing Balance
2336317.00			2336317.00	1262397.00	
					121669.00
					715854.00
					406187.00
					2217.00
					16470.00
					0.00
					0.00
					0.00
					0.00
					0.00
					1262397.00
					0.00
					1262397.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-176 : International Atomic Energy Agency							
PI : Dr K P Arun Kumar							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
200103.00		Opening Balance	208017.00		Opening Balance		0.00
207044.00		Grant In Aid	0.00		Salaries - Manpower		0.00
0.00			0.00		Consumables		25569.00
0.00			0.00		Contingencies		0.00
0.00			0.00	199130.00	Travel		43159.00
0.00			0.00	0.00	Overheads		0.00
0.00			0.00	0.00	Equipment		0.00
0.00			0.00	0.00	Books		0.00
0.00			0.00	0.00	AMC		0.00
0.00			0.00	0.00	Others		0.00
0.00			0.00	0.00	Transfer of Funds		0.00
407147.00			208017.00	199130.00			68728.00
0.00		Excess of Expenditure Over Income	0.00	208017.00	Closing Balance		139289.00
407147.00			208017.00	407147.00			208017.00

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CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD P-177 : Morphological and molecular taxonomy of the <i>Phlebotomus argentitipes</i> species complex in relation to transmission of Kala-azar in India”							
PI : Dr J Gowrishankar							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance		119970.00
225000.00		Grant In Aid	0.00		Salaries - Manpower		0.00
0.00			0.00	147576.00	Consumables		0.00
0.00			0.00	0.00	Contingencies		0.00
0.00			0.00	0.00	Travel		0.00
0.00			0.00	0.00	Overheads		0.00
0.00			0.00	0.00	Equipment		0.00
0.00			0.00	0.00	Books		0.00
0.00			0.00	0.00	AMC		0.00
0.00			0.00	0.00	Others		0.00
0.00			0.00	0.00	Transfer of Funds		0.00
225000.00			0.00	344970.00			119970.00
119970.00		Excess of Expenditure Over Income	119970.00	0.00	Closing Balance		0.00
344970.00			119970.00	344970.00			119970.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-178 : Understanding differential signaling via toll like receptor-2: A proteomics approach					
PI : Dr Rameshwaram Nagender Rao					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs	Rs	Rs	Rs	Rs	Rs
0.00	Opening Balance	184199.00	660000.00	Opening Balance	0.00
1000000.00	Grant In Aid	900000.00	134050.00	Salaries - Manpower	589032.00
0.00		0.00	15801.00	Consumables	194594.00
0.00		0.00	5950.00	Contingencies	26521.00
0.00		0.00	0.00	Travel	5800.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1000000.00		1084199.00	815801.00		815947.00
0.00	Excess of Expenditure Over Income	0.00	184199.00	Closing Balance	268252.00
1000000.00		1084199.00	1000000.00		1084199.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-179 : Quality Assurance Programme for Molecular and Prenatal Diagnosis of Hemoglobin Opathies					
PI : Dr Ashwin B Dalal					
Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs	Rs	Rs	Rs	Rs	Rs
0.00	Opening Balance	50000.00	500000.00	Opening Balance	0.00
1000000.00	Grant In Aid	50000.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	1000000.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1000000.00		100000.00	500000.00		100000.00
0.00	Excess of Expenditure Over Income	0.00	50000.00	Closing Balance	0.00
1000000.00		100000.00	1000000.00		100000.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-180 : Collaborative studies on genomic diversity among bombycoid silkworms in Asia									
PI : Dr K P Arun Kumar									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
117886.00		Opening Balance	63384.00				Opening Balance		
0.00		Grant In Aid	0.00		0.00		Salaries - Manpower		0.00
0.00			0.00		0.00		Consumables		0.00
0.00			0.00		4223.00		Contingencies		0.00
0.00			0.00		50279.00		Travel		54763.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		0.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		0.00
117886.00			63384.00		54502.00				54763.00
0.00		Excess of Expenditure Over Income	0.00		63384.00		Closing Balance		8621.00
117886.00			63384.00		117886.00				63384.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-181 : To conduct multilocational field trails on transgenic BmNPV resistant silkworm strains to establish their efficacy and generate data for their regulatory approval									
PI : Dr V V Satyavathi									
Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
1744000.00		Opening Balance	1223096.00				Opening Balance		
0.00		Grant In Aid	1164000.00		446512.00		Salaries - Manpower		661050.00
0.00			0.00		0.00		Consumables		0.00
0.00			0.00		0.00		Contingencies		0.00
0.00			0.00		74392.00		Travel		34254.00
0.00			0.00		0.00		Overheads		0.00
0.00			0.00		0.00		Equipment		0.00
0.00			0.00		0.00		Books		0.00
0.00			0.00		0.00		AMC		0.00
0.00			0.00		0.00		Others		0.00
0.00			0.00		0.00		Transfer of Funds		0.00
1744000.00			2387096.00		520904.00				695304.00
0.00		Excess of Expenditure Over Income	0.00		1223096.00		Closing Balance		1691792.00
1744000.00			2387096.00		1744000.00				2387096.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-182 : Ramalingaswami Fellowship									
PI : Dr Mohan C Joshi									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	533274.00		277500.00		Opening Balance		0.00
2110000.00		Grant In Aid	0.00		555000.00		Salaries - Manpower		0.00
0.00			0.00		744226.00		Consumables	136369.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	396905.00	
2110000.00			533274.00		1576726.00			533274.00	
0.00		Excess of Expenditure Over Income	0.00		533274.00		Closing Balance	0.00	
2110000.00			533274.00		2110000.00			533274.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-183 : "Prevalence and predictors of vitamin B12 deficiency: genetic associations for low vitamin B12 levels-multi-center a pan India study",									
regulatory approval									
PI : Dr G R Chandak									
Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		0.00		Opening Balance	1091800.00	
0.00		Grant In Aid	1091800.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		1091800.00		Transfer of Funds	0.00	
0.00			1091800.00		1091800.00			1091800.00	
1091800.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	0.00	
1091800.00			1091800.00		1091800.00			1091800.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-186 : In vivo corss-talks between Rho-dependent transcription termination and other biological processes							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
PI : Dr Ranjan Sen							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
2410000.00		Opening Balance	449029.00		Opening Balance		0.00
1841600.00		Grant In Aid	1599800.00		Salaries - Manpower		469678.00
0.00			0.00		Consumables		974460.00
0.00			0.00		Contingencies		0.00
0.00			0.00		Travel		0.00
0.00			0.00		Overheads		0.00
0.00			0.00		Equipment		0.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
4251600.00			2048829.00				1444138.00
0.00		Excess of Expenditure Over Income	0.00		Closing Balance		604691.00
4251600.00			2048829.00				2048829.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-187 : Understanding the mechanism of induction of innate immunity in plants by the Xanthomonas Diffusible signal factor (DSF)							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
PI : Dr Subhadeep Chatterjee							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
1368000.00		Opening Balance	1282677.00		Opening Balance		0.00
0.00		Grant In Aid	600000.00		Salaries - Manpower		338923.00
0.00			0.00		Consumables		0.00
0.00			0.00		Contingencies		12000.00
0.00			0.00		Travel		43687.00
0.00			0.00		Overheads		0.00
0.00			0.00		Equipment		0.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
1368000.00			1882677.00				394610.00
0.00		Excess of Expenditure Over Income	0.00		Closing Balance		1488067.00
1368000.00			1882677.00				1882677.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-188 : Identification of Novel Genes for Intellectual Disability									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
PI : Dr Aneek Das Bhowmik									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
1450000.00		Opening Balance	832894.00		605000.00		Opening Balance		0.00
0.00		Grant In Aid	1250000.00		0.00		Salaries - Manpower	661250.00	
0.00			0.00		0.00		Consumables	500000.00	
0.00			0.00		4620.00		Contingencies	4500.00	
0.00			0.00		7486.00		Travel	1100.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
1450000.00			2082894.00		617106.00			1276280.00	
0.00		Excess of Expenditure Over Income	0.00		832894.00		Closing Balance	806614.00	
1450000.00			2082894.00		1450000.00			2082894.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-189 : Characterization of glycosylphosphatidylinositol-linked aspartyl proteases in Candida glabrata: role in pathogenicity									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
PI : Dr Rupinder Kaur									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
16858467.00		Opening Balance	17423746.00		557793.00		Opening Balance		0.00
5629854.00		Grant In Aid	3605421.00		3352015.00		Salaries - Manpower	652270.00	
0.00			0.00		0.00		Consumables	3927919.00	
0.00			0.00		0.00		Contingencies	14778.00	
0.00			0.00		94351.00		Travel	97324.00	
0.00			0.00		460416.00		Overheads	574057.00	
0.00			0.00		600000.00		Equipment	1048275.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
22488321.00			21029167.00		5064575.00			6314623.00	
0.00		Excess of Expenditure Over Income	0.00		17423746.00		Closing Balance	14714544.00	
22488321.00			21029167.00		22488321.00			21029167.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-190 : Exploring mycobacteriophages to source novel factors / regulators of bacterial transcription machinery							
PI : Dr Shweta Singh							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
1100000.00		Opening Balance	245026.00		Opening Balance		0.00
0.00		Grant In Aid	950000.00		Salaries - Manpower	660000.00	
0.00			0.00		Consumables	2956685.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	4388.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
1100000.00			1195026.00			960073.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	234953.00	
1100000.00			1195026.00			1195026.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-191 : "Human Frontier Science Program Research Grant - A comprehensive approach towards the chemistry & biology of polyphosphate: the forgotten biopolymer							
PI : Dr Rashna Bhandari							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	5718535.00		Opening Balance		0.00
7765092.00		Grant In Aid	0.00		Salaries - Manpower	2847195.00	
0.00			0.00		Consumables	736127.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	46056.00	
0.00			0.00		Overheads	519867.00	
0.00			0.00		Equipment	1412117.00	
0.00			0.00		Books	157173.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
7765092.00			5718535.00			5718535.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	0.00	
7765092.00			5718535.00			5718535.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-192 : Design of peptide inhibitor(s) for the bacterial transcription terminator Rho, a potent drug target							
PI : Dr Ranjan Sen							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	458917.00		Opening Balance		0.00
3819000.00		Grant In Aid	1819800.00		Salaries - Manpower		436800.00
0.00			0.00		Consumables		181687.00
0.00			0.00		Contingencies		0.00
0.00			0.00		Travel		11821.00
0.00			0.00		Overheads		0.00
0.00			0.00		Equipment		0.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
3819000.00			2278717.00				630308.00
0.00		Excess of Expenditure Over Income	0.00		Closing Balance		1648409.00
3819000.00			2278717.00				2278717.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-193 : Screening for male infertility markers in the human Yq12 heterochromatic block							
PI : Dr Ashwin B Dalal							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	1001347.00		Opening Balance		0.00
1050000.00		Grant In Aid	0.00		Salaries - Manpower		197903.00
0.00			0.00		Consumables		684330.00
0.00			0.00		Contingencies		10000.00
0.00			0.00		Travel		31432.00
0.00			0.00		Overheads		0.00
0.00			0.00		Equipment		0.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
1050000.00			1001347.00				923665.00
0.00		Excess of Expenditure Over Income	0.00		Closing Balance		77682.00
1050000.00			1001347.00				1001347.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-194 : Mechanisms and regulation of iron transport in the pathogenic yeast <i>Candida glabrata</i>					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
PI : Dr Rupinder Kaur					
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments
0.00		Opening Balance	210034.00		Opening Balance
500000.00		Grant In Aid	0.00	0.00	Salaries - Manpower
0.00			0.00	0.00	Consumables
0.00			0.00	0.00	Contingencies
0.00			0.00	0.00	Travel
0.00			0.00	0.00	Overheads
0.00			0.00	289966.00	Equipment
0.00			0.00	0.00	Books
0.00			0.00	0.00	AMC
0.00			0.00	0.00	Others
0.00			0.00	0.00	Transfer of Funds
500000.00			210034.00	289966.00	
0.00		Excess of Expenditure Over Income	0.00	210034.00	Closing Balance
500000.00			210034.00	500000.00	
					210034.00
					210034.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-195 : Molecular and biophysical characterization of the ESAT-6: 2M complex and its effect on intracellular iron concentration and macrophage anti-mycobacterial effector responses"					
PI : Dr Sangita Mukhopadhyay					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments
0.00		Opening Balance	872204.00		Opening Balance
1285000.00		Grant In Aid	1285000.00	109200.00	Salaries - Manpower
0.00			0.00	288596.00	Consumables
0.00			0.00	0.00	Contingencies
0.00			0.00	15000.00	Travel
0.00			0.00	0.00	Overheads
0.00			0.00	0.00	Equipment
0.00			0.00	0.00	Books
0.00			0.00	0.00	AMC
0.00			0.00	0.00	Others
0.00			0.00	0.00	Transfer of Funds
1285000.00			2157204.00	412796.00	
0.00		Excess of Expenditure Over Income	0.00	872204.00	Closing Balance
1285000.00			2157204.00	1285000.00	
					681672.00
					1475532.00
					2157204.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
p-196 : Exploring the volatome of noncommunicable diseases as a promising, innovative and integrating approach for its rapid diagnostics"									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
PI : Dr H A Nagarajaram									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	1164020.70		0.00		Opening Balance	0.00	
1281744.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		117723.30		Travel	0.00	
0.00			0.00		0.00		Overheads	9418.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	1154603.00	
1281744.00			1164020.70		117723.30			1164021.00	
0.00		Excess of Expenditure Over Income	0.30		1164020.70		Closing Balance	0.00	
1281744.00			1164021.00		1281744.00			1164021.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-197 : National Post Doctoral Fellowship									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
PI : Dr Madhu Babu Battu									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	583730.00		330000.00		Opening Balance	0.00	
960000.00		Grant In Aid	559246.00		46270.00		Salaries - Manpower	660000.00	
0.00			0.00		0.00		Consumables	90785.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	123841.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
960000.00			1142976.00		376270.00			874626.00	
0.00		Excess of Expenditure Over Income	0.00		583730.00		Closing Balance	268350.00	
960000.00			1142976.00		960000.00			1142976.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-198 : Whole Genome Sequencing for characterization of novel genes and de novo balanced chromosomal rearrangements in human genetic disorders"							
PI : Dr Ashwin Dalal							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	2493600.00		Opening Balance		0.00
2556000.00		Grant In Aid	4000000.00		Salaries - Manpower		729600.00
0.00			0.00		Consumables		1956131.00
0.00			0.00		Contingencies		20081.00
0.00			0.00		Travel		48538.00
0.00			0.00		Overheads		0.00
0.00			0.00		Equipment		193695.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
2556000.00			2893600.00				2948045.00
0.00		Excess of Expenditure Over Income	54445.00		Closing Balance		0.00
2556000.00			2948045.00				2948045.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-199 : Investigating cellular processes and pathways controlled by phosphatases							
PI : Dr M Subba Reddy							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	4013536.00		Opening Balance		0.00
4013536.00		Grant In Aid	8034427.00		Salaries - Manpower		850787.00
0.00			0.00		Consumables		7105134.00
0.00			0.00		Contingencies		0.00
0.00			0.00		Travel		0.00
0.00			0.00		Overheads		936408.00
0.00			0.00		Equipment		1408161.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
4013536.00			12047963.00				10300490.00
0.00		Excess of Expenditure Over Income	0.00		Closing Balance		1747473.00
4013536.00			12047963.00				12047963.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-200 : Characterization of divergent functions of ARID1A and ARID1B: the two alternative DNA binding constituents of the human SWI/SNF chromatin remodelling complex							
PI : Dr M D Bashyam							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	1806199.00		Opening Balance		0.00
1830000.00		Grant In Aid	0.00		Salaries - Manpower		300887.00
0.00			0.00		Consumables		1181247.00
0.00			0.00	23801.00	Contingencies		12552.00
0.00			0.00	0.00	Travel		22922.00
0.00			0.00	0.00	Overheads		0.00
0.00			0.00	0.00	Equipment		0.00
0.00			0.00	0.00	Books		0.00
0.00			0.00	0.00	AMC		0.00
0.00			0.00	0.00	Others		0.00
0.00			0.00	0.00	Transfer of Funds		0.00
1830000.00			1806199.00	23801.00			1517608.00
0.00		Excess of Expenditure Over Income	0.00	1806199.00	Closing Balance		288591.00
1830000.00			1806199.00	1830000.00			1806199.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-201 : Defining the functions of MLL in mitosis							
PI : Dr Shweta Tyagi							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	1241000.00		Opening Balance		0.00
1241000.00		Grant In Aid	2320000.00		Salaries - Manpower		218615.00
0.00			0.00	0.00	Consumables		1686613.00
0.00			0.00	0.00	Contingencies		0.00
0.00			0.00	0.00	Travel		25500.00
0.00			0.00	0.00	Overheads		0.00
0.00			0.00	0.00	Equipment		194313.00
0.00			0.00	0.00	Books		0.00
0.00			0.00	0.00	AMC		0.00
0.00			0.00	0.00	Others		0.00
0.00			0.00	0.00	Transfer of Funds		0.00
1241000.00			3561000.00	0.00			2125041.00
0.00		Excess of Expenditure Over Income	0.00	1241000.00	Closing Balance		1435959.00
1241000.00			3561000.00	1241000.00			3561000.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD						
P-202 : To decipher the role of MLL Complex in the process of cytokinesis						
PI : Dr Shweta Tyagi						
Receipts and Payments Account from 01/04/2017 to 31/03/2018						
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount	Current Year Rs
0.00	Opening Balance	603000.00		Opening Balance		0.00
603000.00	Grant In Aid	1800000.00		Salaries - Manpower		226619.00
0.00		0.00		Consumables		139571.00
0.00		0.00		Contingencies		0.00
0.00		0.00		Travel		43375.00
0.00		0.00		Overheads		0.00
0.00		0.00		Equipment		256738.00
0.00		0.00		Books		0.00
0.00		0.00		AMC		0.00
0.00		0.00		Others		0.00
0.00		0.00		Transfer of Funds		0.00
603000.00		2403000.00	0.00			666303.00
0.00	Excess of Expenditure Over Income	0.00	603000.00	Closing Balance		1736697.00
603000.00		2403000.00	603000.00			2403000.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD						
P-203 : Investigation of a potential novel function of fission yeast sirtuin family histone deacetylase Hst4 in regulation of DNA replication						
PI : Dr Devyani Haldar						
Receipts and Payments Account from 01/04/2017 to 31/03/2018						
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount	Current Year Rs
0.00	Opening Balance	1186706.00		Opening Balance		0.00
1186706.00	Grant In Aid	1538000.00		Salaries - Manpower		278633.00
0.00		0.00		Consumables		560629.00
0.00		0.00		Contingencies		1504.00
0.00		0.00		Travel		39006.00
0.00		0.00		Overheads		0.00
0.00		0.00		Equipment		80645.00
0.00		0.00		Books		0.00
0.00		0.00		AMC		0.00
0.00		0.00		Others		0.00
0.00		0.00		Transfer of Funds		0.00
1186706.00		2724706.00	0.00			960417.00
0.00	Excess of Expenditure Over Income	0.00	1186706.00	Closing Balance		1764289.00
1186706.00		2724706.00	1186706.00			2724706.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-204 : To delineate the role of MLL complex in Microtubule organizing capability of Centrosome					
PI : PI : Dr Shweta Tyagi					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	558333.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	414002.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		558333.00	0.00		414002.00
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	144331.00
0.00		558333.00	0.00		558333.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
P-205 : Genetic studies of foetuses with malformations for identification of Non-chromosomal syndromes and Mendelian disorders					
PI : Dr Ashwin B Dalal					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	0.00	Opening Balance	0.00
0.00	Grant In Aid	1484600.00	0.00	Salaries - Manpower	327600.00
0.00		0.00	0.00	Consumables	497632.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	28420.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		1484600.00	0.00		853652.00
0.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	630948.00
0.00		1484600.00	0.00		1484600.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-206 : Characterization of the genetic etiological spectrum and identification of novel genetic etiologies for non-immune fetal hydrops"									
PI : PI : Dr Ashwin B Dalal									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		0.00		Opening Balance	0.00	
0.00		Grant In Aid	300000.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			300000.00		0.00			0.00	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	300000.00	
0.00			300000.00		0.00			300000.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-207 : Genome and transcriptome analysis of chilli anthracnose fungus colletotrichum truncatum									
PI : Dr N Madhusudan Reddy									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		0.00		Opening Balance	0.00	
0.00		Grant In Aid	2456600.00		0.00		Salaries - Manpower	264026.00	
0.00			0.00		0.00		Consumables	68400.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	9584.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			2456600.00		0.00			342010.00	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	2114590.00	
0.00			2456600.00		0.00			2456600.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-208 : National Post Doctoral Fellowship									
PI : PI : Dr Reshma Chowdary Alokam									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		0.00		Opening Balance	0.00	
0.00		Grant In Aid	960000.00		0.00		Salaries - Manpower	586667.00	
0.00			0.00		0.00		Consumables	200000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			960000.00		0.00			786667.00	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	173333.00	
0.00			960000.00		0.00			960000.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
P-209 : Dissecting the contribution and interplay of MSI and CIMP in colorectal cancer in India									
PI : Dr M D Bashyam									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Previous Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		0.00		Opening Balance	0.00	
0.00		Grant In Aid	3054000.00		0.00		Salaries - Manpower	274124.00	
0.00			0.00		0.00		Consumables	398778.00	
0.00			0.00		0.00		Contingencies	5000.00	
0.00			0.00		0.00		Travel	2683.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	387500.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
0.00			3054000.00		0.00			1068085.00	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	1985915.00	
0.00			3054000.00		0.00			3054000.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-211 : " A comprehensive approach towards the chemistry & biology of polyphosphate: the forgotten biopolymer							
PI : PI : Dr Rashna Bhandari							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance		0.00
0.00		Grant In Aid	7602399.66		Salaries - Manpower		378675.00
0.00			0.00		Consumables		110944.00
0.00			0.00		Contingencies		0.00
0.00			0.00		Travel		0.00
0.00			0.00		Overheads		0.00
0.00			0.00		Equipment		0.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
0.00			7602399.66				489619.00
0.00		Excess of Expenditure Over Income	0.00		Closing Balance		7112780.66
0.00			7602399.66				7602399.66

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-212 : Approaching Mycobacterium tuberculosis PPE protein Rv1168c (PPE17) as a potential marker for diagnosis of Tuberculosis (TB) patients in India							
PI : Dr Sangita Mukhopadhyay							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance		0.00
0.00		Grant In Aid	2179000.00		Salaries - Manpower		0.00
0.00			0.00		Consumables		0.00
0.00			0.00		Contingencies		0.00
0.00			0.00		Travel		0.00
0.00			0.00		Overheads		0.00
0.00			0.00		Equipment		0.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
0.00			2179000.00				0.00
0.00		Excess of Expenditure Over Income	0.00		Closing Balance		2179000.00
0.00			2179000.00				2179000.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-213 : Exploring an oncogenic function of p53 mutations identified in Indian squamous cell carcinoma patients							
PI : PI : Dr M D Bashyam							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	0.00	
0.00		Grant In Aid	2724000.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			2724000.00			0.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	2724000.00	
0.00			2724000.00			2724000.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-214 : Studies on Non-Canonical functions of splicing proteins in maintaining genomic stability							
PI : Dr M V Subba Reddy							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	0.00	
0.00		Grant In Aid	854333.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	12000.00	
0.00			0.00		Travel	17893.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
0.00			854333.00			29893.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	824440.00	
0.00			854333.00			854333.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-215 : Understanding Homothorax independent role of Hox cofactor Extracellular in Drosophila neuroblast apoptosis							
PI : PI : Dr Rohit Joshi							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	0.00	
0.00		Grant In Aid	970000.00		Salaries - Manpower	0.00	
0.00					Consumables	0.00	
0.00					Contingencies	0.00	
0.00					Travel	0.00	
0.00					Overheads	0.00	
0.00					Equipment	0.00	
0.00					Books	0.00	
0.00					AMC	0.00	
0.00					Others	0.00	
0.00					Transfer of Funds	0.00	
0.00			970000.00			0.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	970000.00	
0.00			970000.00			970000.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-216 : Investigating the role of mycobacterial protein Rv2966c in modulating the host epigenetic circuitry during infection							
PI : Dr Sanjeev Khosla							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	0.00	
0.00		Grant In Aid	1768000.00		Salaries - Manpower	0.00	
0.00					Consumables	0.00	
0.00					Contingencies	0.00	
0.00					Travel	0.00	
0.00					Overheads	0.00	
0.00					Equipment	0.00	
0.00					Books	0.00	
0.00					AMC	0.00	
0.00					Others	0.00	
0.00					Transfer of Funds	0.00	
0.00			1768000.00			0.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	1768000.00	
0.00			1768000.00			1768000.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-217 : BRICS Research Project - EpiMacroTB, "Epigenetics of macrophages during Mycobacterium tuberculosis infection"							
PI : Dr Sanjeev Khosla							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	0.00	
0.00		Grant In Aid	1141600.00		Salaries - Manpower	0.00	
0.00					Consumables	0.00	
0.00					Contingencies	0.00	
0.00					Travel	0.00	
0.00					Overheads	0.00	
0.00					Equipment	0.00	
0.00					Books	0.00	
0.00					AMC	0.00	
0.00					Others	0.00	
0.00					Transfer of Funds	0.00	
0.00			1141600.00			0.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	1141600.00	
0.00			1141600.00			1141600.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
P-219 : Identification and molecular characterization of the CgHogI Kinase interactome: impact on iron homeostasis and Candida pathogenesis							
PI : Dr Rupinder Kaur							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance	0.00	
0.00		Grant In Aid	1500000.00		Salaries - Manpower	0.00	
0.00					Consumables	0.00	
0.00					Contingencies	0.00	
0.00					Travel	0.00	
0.00					Overheads	0.00	
0.00					Equipment	0.00	
0.00					Books	0.00	
0.00					AMC	0.00	
0.00					Others	0.00	
0.00					Transfer of Funds	0.00	
0.00			1500000.00			0.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	1500000.00	
0.00			1500000.00			1500000.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD COE1/CORE : COE for Genetics and Genomics of silkmooths PI : Dr. J. Nagaraju					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year. Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	12271928.00	Opening Balance	10446277.00
8768000.00	Grant In Aid	0.00	6942349.00	Salaries - Manpower	500408.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
8768000.00		0.00	19214277.00		10946685.00
10446277.00	Excess of Expenditure Over Income	10946685.00	0.00	Closing Balance	0.00
19214277.00		10946685.00	19214277.00		10946685.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD COE1/P-I : Comparative and function genomics of silkmooths. PI : Dr. J. Nagaraju					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year. Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	220587.00	410893.00	Opening Balance	0.00
775000.00	Grant In Aid	0.00	143520.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
775000.00		220587.00	554413.00		0.00
0.00	Excess of Expenditure Over Income	0.00	220587.00	Closing Balance	220587.00
775000.00		220587.00	775000.00		220587.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
COE1/P-II : Development of RNA interference (RNAi) based nuclear polyhedrosis virus (NPV) resistant transgenic silkmoths.					
PI : Dr. J. Nagaraju					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	0.00	593919.00	Opening Balance	144446.00
643000.00	Grant In Aid	0.00	193527.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
643000.00		0.00	787446.00		144446.00
144446.00	Excess of Expenditure Over Income	144446.00	0.00	Closing Balance	0.00
787446.00		144446.00	787446.00		144446.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
COE1/P-III : Identification and Characterization of micro RNAs and their targets in silkmoth genome.					
PI : Dr. J. Nagaraju					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs.	Rs		Rs
0.00	Opening Balance	416412.00	448230.00	Opening Balance	0.00
1090000.00	Grant In Aid	0.00	225358.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
1090000.00		416412.00	673588.00		0.00
0.00	Excess of Expenditure Over Income	0.00	416412.00	Closing Balance	416412.00
1090000.00		416412.00	1090000.00		416412.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD COE2/CORE : DBT Centre of Excellence for Microbial Biology PI : Dr J Gowrishankar, Dr K Anupama, Dr Abhijit A Sardesai, Dr R							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Payments	Current Year Amount	Rs
0.00	Opening Balance	0.00	0.00	23840815.00	Opening Balance	23840815.00	0.00
0.00	Grant In Aid	0.00	0.00	0.00	Salaries - Manpower	0.00	0.00
0.00		0.00	0.00	0.00	Consumables	0.00	0.00
0.00		0.00	0.00	0.00	Contingencies	0.00	0.00
0.00		0.00	0.00	0.00	Travel	0.00	0.00
0.00		0.00	0.00	0.00	Overheads	0.00	0.00
0.00		0.00	0.00	0.00	Equipment	0.00	0.00
0.00		0.00	0.00	0.00	Books	0.00	0.00
0.00		0.00	0.00	0.00	AMC	0.00	0.00
0.00		0.00	0.00	0.00	Others	0.00	0.00
0.00		0.00	0.00	0.00	Transfer of Funds	0.00	0.00
0.00		0.00	0.00	23840815.00		23840815.00	0.00
23840815.00	Excess of Expenditure Over Income	23840815.00	0.00	0.00	Closing Balance	0.00	0.00
23840815.00		23840815.00		23840815.00		23840815.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD COE2/P-1 : Addressing functional properties of E. coli through genome-wide protein-protein linkage analysis PI : Dr. J Gowrishankar							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Payments	Current Year Amount	Rs
0.00	Opening Balance	0.00	0.00	684083.00	Opening Balance	684083.00	0.00
0.00	Grant In Aid	0.00	0.00	0.00	Salaries - Manpower	0.00	0.00
0.00		0.00	0.00	0.00	Consumables	0.00	0.00
0.00		0.00	0.00	0.00	Contingencies	0.00	0.00
0.00		0.00	0.00	0.00	Travel	0.00	0.00
0.00		0.00	0.00	0.00	Overheads	0.00	0.00
0.00		0.00	0.00	0.00	Equipment	0.00	0.00
0.00		0.00	0.00	0.00	Books	0.00	0.00
0.00		0.00	0.00	0.00	AMC	0.00	0.00
0.00		0.00	0.00	0.00	Others	0.00	0.00
0.00		0.00	0.00	0.00	Transfer of Funds	0.00	0.00
0.00		0.00	0.00	684083.00		684083.00	0.00
684083.00	Excess of Expenditure Over Income	684083.00	0.00	0.00	Closing Balance	0.00	0.00
684083.00		684083.00		684083.00		684083.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
COE2/P-2 : Mechanism of transcription termination and antitermination in Escherichia coli					
PI : Dr. Ranjan Sen					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	1441181.00	Opening Balance	1441181.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1441181.00		1441181.00
1441181.00	Excess of Expenditure Over Income	1441181.00	0.00	Closing Balance	0.00
1441181.00		1441181.00	1441181.00		1441181.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
COE2/P-A : Occurrence of R-loops (RNA-DNA hybrids) from nascent untranslated transcripts i E. Coli					
PI : Dr. J Gowrishankar, Dr.K. Anupama					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	1354252.00	Opening Balance	1354252.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1354252.00		1354252.00
1354252.00	Excess of Expenditure Over Income	1354252.00	0.00	Closing Balance	0.00
1354252.00		1354252.00	1354252.00		1354252.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
COE2/P-B : Molecular genetic approaches to dissect the physiology of osmoadaptation in Escherichia coli					
PI : Dr. J. Gowrishankar, Dr. Abhijit A Sardesai					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	1275609.00	Opening Balance	1275609.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	1275609.00		1275609.00
1275609.00	Excess of Expenditure Over Income	1275609.00	0.00	Closing Balance	0.00
1275609.00		1275609.00	1275609.00		1275609.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD					
COE2/P-C : Functional role and mechanisms of the ArgO exporter and the transcriptional regulator ArgP in E. Coli					
PI : Dr. J. Gowrishankar, Dr. Ranjan Sen					
Receipts and Payments Account from 01/04/2017 to 31/03/2018					
Previous Year Amount	Receipts	Current Year Amount	Previous Year Amount	Payments	Current Year Amount
Rs		Rs	Rs		Rs
0.00	Opening Balance	0.00	473354.00	Opening Balance	473354.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	0.00
0.00		0.00	0.00	Consumables	0.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	0.00
0.00		0.00	0.00	Books	0.00
0.00		0.00	0.00	AMC	0.00
0.00		0.00	0.00	Others	0.00
0.00		0.00	0.00	Transfer of Funds	0.00
0.00		0.00	473354.00		473354.00
473354.00	Excess of Expenditure Over Income	473354.00	0.00	Closing Balance	0.00
473354.00		473354.00	473354.00		473354.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
COE2-II/P-1 : In vivo studies on molecular mechanism of Rho-dependent transcription termination									
PI : Dr Ranjan Sen									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
504781.00		Opening Balance	949005.00		1049273.00		Opening Balance		0.00
2100000.00		Grant In Aid	1489000.00		591000.00		Salaries - Manpower	852233.00	
0.00			0.00		0.00		Consumables	15868.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		15503.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
2604781.00			2438005.00		1655776.00			868101.00	
0.00		Excess of Expenditure Over Income	0.00		949005.00		Closing Balance	1569904.00	
2604781.00			2438005.00		2604781.00			2438005.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD									
COE2-II/P-A : Role of R-loops (RNA-DNA hybrids) in generatin of transcription -replication conflicts in E.Coli									
PI : Dr J Gowrishankar									
Receipts and Payments Account from 01/04/2017 to 31/03/2018									
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
1061000.00		Opening Balance	0.00		26068.00		Opening Balance	223603.00	
0.00		Grant In Aid	744000.00		928535.00		Salaries - Manpower	49400.00	
0.00			0.00		330000.00		Consumables	0.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	0.00	
0.00			0.00		0.00		Books	0.00	
0.00			0.00		0.00		AMC	0.00	
0.00			0.00		0.00		Others	0.00	
0.00			0.00		0.00		Transfer of Funds	0.00	
1061000.00			744000.00		1284603.00			273003.00	
223603.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	470997.00	
1284603.00			744000.00		1284603.00			744000.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
COE2-II/P-1 : In vivo studies on molecular mechanism of Rho-dependent transcription termination							
PI : Dr Ranjan Sen							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
504781.00		Opening Balance	949005.00		Opening Balance		0.00
2100000.00		Grant In Aid	1489000.00		Salaries - Manpower		852233.00
0.00			0.00		Consumables		15868.00
0.00			0.00		Contingencies		0.00
0.00			0.00		Travel		0.00
0.00			0.00		Overheads		0.00
0.00			0.00		Equipment		0.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
2604781.00			2438005.00				868101.00
0.00		Excess of Expenditure Over Income	0.00		Closing Balance		1569904.00
2604781.00			2438005.00				2438005.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
COE2-II/P-A : Role of R-loops (RNA-DNA hybrids) in generation of transcription -replication conflicts in E.Coli							
PI : Dr J Gowrishankar							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		Opening Balance		223603.00
1061000.00		Grant In Aid	744000.00		Salaries - Manpower		49400.00
0.00			0.00		Consumables		0.00
0.00			0.00		Contingencies		0.00
0.00			0.00		Travel		0.00
0.00			0.00		Overheads		0.00
0.00			0.00		Equipment		0.00
0.00			0.00		Books		0.00
0.00			0.00		AMC		0.00
0.00			0.00		Others		0.00
0.00			0.00		Transfer of Funds		0.00
1061000.00			744000.00				273003.00
223603.00		Excess of Expenditure Over Income	0.00		Closing Balance		470997.00
1284603.00			744000.00				744000.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
COE2-II/P-B : Role of the ArgP transcriptional regulator and metabolism of basic amino acids Arg and Lys in E.coli							
PI : Dr J Gowrishankar							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Receipts Rs	Current Year Amount	Rs.	Previous Year. Amount	Rs	Current Year Amount	Rs
0.00	Opening Balance	0.00		510077.00		976032.00	
488000.00	Grant In Aid	250000.00		603955.00	Salaries - Manpower	592800.00	
0.00		0.00		350000.00	Consumables	0.00	
0.00		0.00		0.00	Contingencies	0.00	
0.00		0.00		0.00	Travel	0.00	
0.00		0.00		0.00	Overheads	0.00	
0.00		0.00		0.00	Equipment	0.00	
0.00		0.00		0.00	Books	0.00	
0.00		0.00		0.00	AMC	0.00	
0.00		0.00		0.00	Others	0.00	
0.00		0.00		0.00	Transfer of Funds	0.00	
488000.00		250000.00		1464032.00		1568832.00	
976032.00	Excess of Expenditure Over Income	1318832.00		0.00	Closing Balance	0.00	
1464032.00		1568832.00		1464032.00		1568832.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
COE2-II/P-C : Investigating global RNA turnover mechanisms and their interplay with Rho-dependent transcription termination in E. coli							
PI : Dr K Anupaman							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Receipts Rs	Current Year Amount	Rs.	Previous Year. Amount	Rs	Current Year Amount	Rs
577635.00	Opening Balance	1308635.00		0.00		0.00	
1061000.00	Grant In Aid	744000.00		330000.00	Salaries - Manpower	0.00	
0.00		0.00		0.00	Consumables	0.00	
0.00		0.00		0.00	Contingencies	0.00	
0.00		0.00		0.00	Travel	0.00	
0.00		0.00		0.00	Overheads	0.00	
0.00		0.00		0.00	Equipment	0.00	
0.00		0.00		0.00	Books	0.00	
0.00		0.00		0.00	AMC	0.00	
0.00		0.00		0.00	Others	0.00	
0.00		0.00		0.00	Transfer of Funds	0.00	
1638635.00		2052635.00		330000.00		0.00	
0.00	Excess of Expenditure Over Income	0.00		1308635.00	Closing Balance	2052635.00	
1638635.00		2052635.00		1638635.00		2052635.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
COE2-II/P-D : Molecular, genetic and biochemical studies on physiology of K+ION homeostatis and the regulatory mechanisms mediating avoidance of its imbalance in Escherichia coli							
PI : Dr Abhijit A Sardesai							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
300000.00		Opening Balance	439000.00		Opening Balance		0.00
496000.00		Grant In Aid	250000.00		Salaries - Manpower		0.00
0.00			0.00		Consumables	357000.00	0.00
0.00			0.00		Contingencies	0.00	0.00
0.00			0.00		Travel	0.00	0.00
0.00			0.00		Overheads	0.00	0.00
0.00			0.00		Equipment	0.00	0.00
0.00			0.00		Books	0.00	0.00
0.00			0.00		AMC	0.00	0.00
0.00			0.00		Others	0.00	0.00
0.00			0.00		Transfer of Funds	0.00	0.00
796000.00			689000.00			357000.00	0.00
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	439000.00	689000.00
796000.00			689000.00			796000.00	689000.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD							
COE2-II/P-E : Understanding (p) ppGpp-mediated functions in E.Coli by deciphering the physiology of strain lacking (p)ppGpp OR altered in its metabolism							
PI : Dr J Gowrishankar							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
713939.00		Opening Balance	982539.00		Opening Balance		0.00
866000.00		Grant In Aid	744000.00		Salaries - Manpower	326400.00	388826.00
0.00			0.00		Consumables	271000.00	0.00
0.00			0.00		Contingencies	0.00	0.00
0.00			0.00		Travel	0.00	0.00
0.00			0.00		Overheads	0.00	0.00
0.00			0.00		Equipment	0.00	0.00
0.00			0.00		Books	0.00	0.00
0.00			0.00		AMC	0.00	0.00
0.00			0.00		Others	0.00	0.00
0.00			0.00		Transfer of Funds	0.00	0.00
1579939.00			1726539.00			597400.00	388826.00
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	982539.00	1337713.00
1579939.00			1726539.00			1579939.00	1726539.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD COE2-II-Core : DBT Centre of Excellence for Microbiology - Phase II							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
PI : Dr J Gowrishankar							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
1736568.00		Opening Balance	2635661.00		Opening Balance		0.00
3447000.00		Grant In Aid	4929000.00		Salaries - Manpower	2148984.00	
0.00			0.00		Consumables	505165.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	9164.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	307210.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
5183568.00			7564661.00			2970523.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	4594138.00	
5183568.00			7564661.00			7564661.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD COE-I/P-IV : Identification and characterization of immune response genes of silkworms.							
Receipts and Payments Account from 01/04/2017 to 31/03/2018							
PI : Dr. J. Nagaraju							
Previous Year Amount	Rs	Receipts	Current Year Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	303397.00		Opening Balance		0.00
442000.00		Grant In Aid	0.00		Salaries - Manpower	0.00	
0.00			0.00		Consumables	0.00	
0.00			0.00		Contingencies	0.00	
0.00			0.00		Travel	0.00	
0.00			0.00		Overheads	0.00	
0.00			0.00		Equipment	0.00	
0.00			0.00		Books	0.00	
0.00			0.00		AMC	0.00	
0.00			0.00		Others	0.00	
0.00			0.00		Transfer of Funds	0.00	
442000.00			303397.00			138603.00	
0.00		Excess of Expenditure Over Income	0.00		Closing Balance	303397.00	
442000.00			303397.00			303397.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
others : Fellowship / others

PI : OTHERS

Receipts and Payments Account from 01/04/2017 to 31/03/2018

Previous Year		Receipts		Current Year		Payments		Current Year	
Amount	Rs	Amount	Rs.	Amount	Rs	Amount	Rs	Amount	Rs
0.00		Opening Balance		2028298.00		Opening Balance		635021.00	0.00
2028298.00		Grant In Aid		1136685.00		Salaries - Manpower		70700.00	0.00
0.00				0.00		Consumables		0.00	0.00
0.00				0.00		Contingencies		8465.00	0.00
0.00				0.00		Travel		0.00	0.00
0.00				0.00		Overheads		0.00	0.00
0.00				0.00		Equipment		0.00	0.00
0.00				0.00		Books		0.00	0.00
0.00				0.00		AMC		0.00	0.00
0.00				0.00		Others		0.00	0.00
0.00				0.00		Transfer of Funds		0.00	0.00
2028298.00				3164983.00				714186.00	
0.00		Excess of Expenditure Over Income		0.00		Closing Balance		2450797.00	
2028298.00				3164983.00				3164983.00	

फोटो गैलरी
Photo Gallery



Republic Day Flag Hoisting



Yoga Day Celebration at CDFD



Yoga Day Celebration at CDFD



Visit By Dr. Ted Trimble (NCI, USA)



Hindi Day Celebration



Hindi Day Celebration



Independence Day Flag Hoisting at CDFD Uppal Campus



Unicode Workshop at CDFD



India International Science Festival (IISF-2017) at CDFD



Vigilance Awareness Week



CDFD Staff Taking Vigilance Pledge



Dr. Debashis Mitra Taking Over as Director of CDFD



5 km Run on Republic Day



MoU Signing with National Police Academy



Visit by Indian Air Force Officers



CDFD Moves to Permanent Campus in Uppal



CDFD Lab Building in Uppal Campus



Green House in CDFD Uppal Campus



Tree Plantation by Dr. V. S. Chauhan at CDFD New Campus

NOTES
