सी डी एफ डी
CDFD

वार्षिक प्रतिवेदन

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नामपल्ली, हैदराबाद - 500 001

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अधिदेश

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 संगठनों के 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

मुझे डीएनए फिंगरप्रिंटिंग एवं निदान केन्द्र (सीडीएफडी), हैदराबाद की वार्षिक रिपोर्ट प्रस्तुत करते हुए अत्यंत प्रसन्नता है। संस्थान 1996 में स्थापित किया गया था और तब से इसने विविध गतिविधियों में उत्कृष्टता अर्जित की है। संस्थान डीएनए फिंगरप्रिंटिंग, मानव आनुवंशिकी विकारों के लिए नैदानिक परीक्षणों तथा शुद्धता के लिए बासमती चावल के विश्लेषण के क्षेत्रों में सेवाएं प्रदान करता है तथा यह आधुनिक जीव विज्ञान के विभिन्न विषयों में भी बुनियादी अनुसंधान गतिविधियां में संलग्न है। इस वर्ष केन्द्र की कुछ प्रमुख उपलब्धियां और अनुसंधान प्राप्तियां आगे दी गई है, जिनके विवरण अलग अलग प्रयोगशालाओं द्वारा विवरणों में संलग्न किए गए हैं, जो इस रिपोर्ट में संलग्न हैं।

2014-15 की अवधि के दौरान डीएनए फिंगरिप्रिंटिंग सेवा प्रयोगशाला में लगभग 550 मामले प्राप्त। किए गए, जिन्हें न्याय पालिका तथा राज्य और संघीय सरकारों की कानून प्रवर्तन एजेंसियों द्वारा अग्रेषित किया गया था तथा डीएनए परीक्षकों ने पूरे देश की विभिन्न कानूनी अदालतों में अपनी रिपोर्ट प्रदान की है। प्रयोगशाला में फेडरल ब्यूरो ऑफ इंवेस्टीगेशन (एफबीआई), यूएसए से कम्बाइंड डीएनए इंडेक्स सिस्टम (सीओडीआईएस) सॉफ्टवेयर खरीदा गया है, जो आपराधिक न्याय प्रदायगी और दीवानी प्रक्रियाओं के लिए डीएनए डेटा बैंक में भंडारित डीएनए प्रोफाइल के साथ मिलान में सहायता करेगा। जहां तक मानव डीएनए प्रोफाइलिंग विधेयक का संबंध है, केन्द्र जैव प्रौद्योगिकी विभाग, भारत सरकार के साथ संसद द्वारा लागू करने के लिए प्रारूप विधेयक को अंतिम रूप देने का समन्वय करता है।

नैदानिकी प्रभाग द्वारा विभिन्नि आनुवंशिकी लोगों के लिए लगभग 3700 रोगियों को आनुवंशिकी सेवाएं प्रदान की गई। चिकित्सा आनुवंशिकी में एक डीएनबी कार्यक्रम आरंभ किया गया और क्लिनिकल साइटोजेनेटिक्सन तथा क्लिनिकल आण्विक आनुवंशिकी में भी अध्येतावृत्ति कार्यक्रम चलाए गए हैं।

बासमती अपमिश्रण परीक्षण में आने वाली जटिलताओं और चुनौतियों को ध्यान में रखते हुए बासमती डीएनए विश्लेषण के लिए एपिडा-सीडीएफडी केन्द्रो द्वारा किए गए प्रयासों को अपमिश्रण परीक्षण प्रोटोकॉल विस्तारित करने के लिए आगे बढ़ाया गया है। इस दिशा में केन्द्र ने 8 मार्कर के पैनल की विधि का विस्तार एक व्यापक डेटाबेस तैयार करने के लिए सभी अधिसूचित बासमती किस्मों की पहचान के लिए विस्तारित किया है।

कोशिका चक्र विनियमन प्रयोगशाला में एच3के4एमई3 डीमेथिलेस के रूप में आरबीपी2 को चुना है जो ई२एफ4 प्रोटीन के ट्रांसएक्टिवेशन डोमेन के साथ अंत:क्रिया करता है। इस प्रयोगशाला के अनुसंधान परिणाम दर्शाते हैं कि सभी एमएलएल कॉम्प्लेक्स जो हैं एमएलएल, एमएलएल2, एमएलएल3 तथा एसईटी 1ए एस चरण के आगे बढ़ने के दौरान इसके नियमन में एक भूमिका निभाते हैं, केवल एमएलएल और एसईटी 1ए ऐसे हैं जो एम चरण की प्रगति की सुविधा प्रदान करने के लिए जिम्मेदार हैं। आण्विक ओंकोलॉजी प्रयोगशाला में प्रथम गैर डब्ल्यूएनटी (12 जीन) को पहचाना गया है जो मनुष्य में यदा कदा होने वाले मलाशय के कैंसर की जल्दी शुरूआत का संकेत देता है।

कोशिका सिग्नलिंग प्रयोगशाला में प्रदर्शित किया गया है कि आईपी7 ओंकोप्रोटीन सी-एमवायसी पायरोफॉस्फोराइलेट होने के जरिए तथा इसके अर्ध जीवन और यूबीक्विटिलेशन का नियमन करता है। इस प्रयोगशाला के अनुसंधान में दर्शाया गया है कि नर चूहे में आईपी6के1 में अर्ध सूत्री

विभाजन के अवकलन के बाद हुए दोषों के कारण अनुवर्रता होने से गोल स्पर्मेटिड परिपक्व होकर लंबे स्पर्मेटिड बनाते हैं, जिससे एजूस्पर्मिया हो जाता है। क्रोमेटिन जीव विज्ञान तथा एपिजेनेटिक्स प्रयोगशाला द्वारा एसयूपी1 खोजा गया, जो विदलन इस्ट सिरट्यूइन एचएसटी4 का एक नया अत:क्रियात्मक कारक है जो डीएनए पॉलीमरेस अल्फा से संबद्ध रेप्लीकेशन कारक है।

अभिकलनात्मक जीव विज्ञान प्रयोगशाला में सिद्ध किया गया है कि वायरल प्रोटीन से ऐसे मोटिफ बनते हैं जो यूकेरियोटिक लिनियर मोटिफ (ईएलएम) के समान होते हैं और मानव प्रोटीन से जुड़ कर ईएलएम बंधनकारी डोमेन बनाते हैं। अभिकलनात्मक और कार्यात्मक जीनोमिकी प्रयोगशाला में मानव एचवायपीके प्रोटीन को लाक्षणीकृत किया गया है और दर्शाया गया है कि यह हंटिंगटीन जैसे समुच्चय संवेदी प्रोटीनों के साथ किस प्रकार अनुभूति और व्यवधान उत्पन्न करता है।

कैंडिडा ग्लाब्रेटा के साथ कार्य करते हुए कवक रोगाणुजनन प्रयोगशाला में एक आयरन परमिएस सीजीएफटीआर1 नामक एक मल्टी कॉपर ऑक्सीडेस सीजीएफईटी3 और कॉपर ट्रांसपोर्टर सीजीसीसीसी2 एवं एक माइटोक्रॉन्ड्रियल फ्रेक्टेक्सिन सीजीवायएफएच1 अभिज्ञात किया गया है जो उच्च बंधुता अपचायक आयरन परिवहन का प्रधान मूल घटक है और आयरन चयापचय उपकरण के साथ क्रमश: यह सिद्ध करता है कि उच्च बंधुता आयरन अधिग्रहण प्रक्रियाएं जीवों में निर्णायक रोगजनक निर्धारक हैं। पादप सूक्ष्मजीव अंत:क्रिया प्रयोगशाला में कोरम सेंसिंग की भूमिका का अध्ययन जेंथोमोनास ओरिजी पीवी ओरिजी कोला में साइडेरोफोर वाइब्रियोफेरिन के उत्पोदन का नियमन करने में देखी गई है, जो पौधों में वृद्धि और रोगजनकता के लिए आवश्यक है।

स्तानधारी आनुवंशिकी प्रयोगशाला के कार्यों में कार्सिनोजेनेसिस और विकास में डीएनए मिथेलट्रांसफरेस डीएनएमटी3एल और डीएनएमटी2 की भूमिका को समझा गया है। इसने एपिजेनेटिक बदलावों को भी अभिज्ञात किया है जो मेजबान कोशिका में माइकोबैक्टीरियम ट्यूबरकुलोसिस के साथ चुनौती देने पर आते हैं। आण्विक कोशिका जीव विज्ञान प्रयोगशाला द्वारा रिपोर्ट किया गया है कि एम. ट्यूबरकुलोसिस के ईएसएटी-6 प्रोटीन मेजबान बीटा2एम के साथ अंत:क्रिया करते हैं तथा वर्ग 1 माध्मित एंटीजन प्रस्तुततीकरण का संदमन करते हैं। समूह में एम. ट्यूबरकुलोसिस के पीपीई प्रोटीनों द्वारा एंटी तथा प्रोइंफ्लेमेटरी प्रतिक्रियाओं के नियमन में शामिल टीएलआर2 के एक नए आईआरएके3 सिग्नलिंग मार्ग डाउन स्ट्रीम को भी अभिज्ञात किया गया है।

सिल्कमॉथ आनुवंशिकी एवं जीनोमिक्स उत्कृष्टता केन्द्र में किए गए अनुसंधान में दर्शाया गया है कि बॉम्बिक्स लिंग गुणसूत्र जेड में मात्रा का मुआवज़ा दिया जाता है, इसके साथ ही अभिव्यक्ति ऑटोसोम से आधे से भी कम हो जाती है। केन्द्र को बहु स्थान परीक्षणों के आयोजन के लिए औपचारिक अनुमति प्राप्त हो गई है, जिसमें आनुवंशिक रूप से निर्मित सिल्कमॉथ विभेदों की निहित सुविधाएं शामिल हैं।

न्यूरोस्पोरा आनुवंशिकी प्रयोगशाला को अप्रभावी उत्परिवर्तन के लिए साक्ष्य प्राप्त हुआ है जो खास तौर पर वैकल्पिक विखंडन को प्रभावित करता है और इसके साथ समवर्ती 1 सेग्रीगेशन पर कोई प्रभाव दिखाई नहीं देता। अनुलेखन प्रयोगशाला में आरएचओ आश्रित अनुलेखन समापन के आण्विक आधार का मॉड्यूलेशन दर्शाया गया है, जिसके द्वारा दो अनुलेखन कारक एनयूएसए और एनयूएसजी तथा एनयूएसए के बैक्टीरियोफेज एन द्वारा एंटी टर्मिनेटर में रूपांतरण की प्रक्रिया को समझा गया है।

इस वर्ष भी पिछले वर्ष के समान सीडीएफडी के अनेक संकाय सदस्यों तथा अध्येताओं को प्रतिष्ठित पुरस्कार और सम्मान प्राप्त हुए हैं। इन पुरस्कारों में अन्य के अलावा आईसीएमआर बसंती देवी अमिर चंद पुरस्कार, इंसा और रॉयल सोसाइटी, एडिन बर्ग के बीच वैज्ञानिकों के आदान प्रदान के तहत अध्येक्तावृत्ति, छठवें एफईबीएस उन्नत व्याख्यान पाठ्यक्रम में मानव कवक रोगाणुओं पर आरंभिक व्याख्यातता; डीएसटीफएसईआरबी फास्ट ट्रेक युवा वैज्ञानिक पुरस्कार; ए पी अकादमी ऑफ साइंस की ओर से युवा वैज्ञानिक पुरस्कार; डॉ. के वी राव अनुसंधान पुरस्कार; श्यामा प्रसाद मुखर्जी अध्येतावृत्ति; एएसएम यात्रा पुरस्कार; आईसीएमआर यात्रा अनुदान; प्रो. जी पी तलवार ट्रेवल बर्सरी; डीएसटी यात्रा अनुदान आदि। इस अवधि के दौरान

चार अनुसंधान अध्येताओं को पीएचडी की उपाधि प्रदान की गई। अनेक पोस्ट डॉक्टरल अध्येता, परियोजना सहयोगी और ग्रीष्मकालीन प्रशिक्षु सीडीएफडी में कार्य करते हैं और केन्द्र की गतिविधियों में उल्लेखनीय भूमिका निभाते हैं।

केन्द्र के आगामी परिसर की निर्माण गतिविधियां पूरी तेजी पर सरकार की व्यय वित्त समिति द्वारा पूर्व अनुमोदित योजना के अनुसार जारी हैं।

मैं इस अथक सहयोग के प्रति आभार व्यक्त करता हूं जो इसकी गतिविधियों के लिए शासी परिषद, अनुसंधान क्षेत्र पैनल - वैज्ञानिक सलाहकार समिति, शैक्षिक / वित्तीय / भवन समितियों तथा बेशक जैव प्रौद्योगिकी विभाग की ओर से प्रदान किया गया। मैं सभी सदस्यों और अधिकारियों को उनके द्वारा दिए गए समय तथा हमारी गतिविधियों और उपलब्धियों के समर्थन हेतु किए गए प्रयासों के लिए धन्यवाद देता हूं।

मैं सीडीएफडी परिवार के प्रति भी अपना हार्दिक आभार व्युक्त करता हूं जिसने केन्द्र के जारी कार्यक्रमों तथा विकास में एक अहम भूमिका निभाई है।

ज गौरीशंकर

31 मार्च, 2015

Director's Message

I have great pleasure in presenting the Annual Report of the Centre for DNA Fingerprinting and Diagnostics (CDFD), Hyderabad. The institute was established in 1996 and has since excelled in its diverse activities. The institute provides services in the areas of DNA fingerprinting, diagnostic tests for human genetic disorders and analysis of basmati rice for purity, and is also engaged in basic research activities in different disciplines of modern biology. A few of the major achievements and research findings from the Centre this year are given below, the details of which are covered in the descriptions by the individual laboratories that are enclosed in this Report.

During the period 2014-15, the Laboratory of DNA Fingerprinting Services received ~550 cases that were forwarded by the judiciary and law enforcing agencies of State and Federal Governments and the DNA Examiners have defended their reports in various Courts of law throughout the country. The Lab has procured the Combined DNA Index System (CODIS) software from the Federal Bureau of Investigation (FBI), USA, which will aid in matching of DNA profiles stored in a DNA Data Bank for both criminal justice delivery and civil proceedings. As regards the Human DNA Profiling Bill, the Centre is coordinating with the Department of Biotechnology, Government of India, to finalize the draft Bill for enactment by Parliament.

The Diagnostics division provided genetic services to around 3700 patients for various genetic diseases. A DNB program in Medical Genetics has been initiated, as also fellowship programs in Clinical Cytogenetics and Clinical Molecular Genetics.

In view of the complexities and challenges arising in Basmati adulteration testing, efforts are being made by the APEDA-CDFD Centre for Basmati DNA Analysis to further expand adulteration testing protocol. Towards this direction, the Centre extended the method of multiplexed eight markers panel for identification of all notified Basmati varieties to generate a comprehensive database. The Centre also standardized single grain analysis for varietal identification of Basmati rice.

The Laboratory of Cell Cycle Regulation has identified RBP2 as the H3K4me3 demethylase that interacts with the transactivation domain of E2F4 protein. The research results of this laboratory show that while all MLL complexes, namely MLL, MLL2, MLL3 and SET 1A play a role in regulating S phase progression, it is only MLL and SET 1A that are responsible for facilitating M phase progression.The Laboratory of Molecular Oncology has identified the first non-Wnt (12-gene) signature for early onset sporadic rectal cancer in humans.

The Laboratory of Cell Signalling demonstrated that IP_7 pyrophosphorylates the oncoprotein c-Myc and regulates its half-life and ubiquitylation. Research in this Laboratory has shown that male mice lacking IP6K1 display infertility due to defects in post-meiotic differentiation of round spermatids to mature elongated spermatids, leading to azoospermia.The Laboratory of Chromatin Biology and Epigenetics discovered Sup1, which is a DNA polymerase alpha associated replication factor, as a novel interactor of fission yeast Sirtuin Hst4.

The Laboratory of Computational Biology established that viral proteins harbor motifs that mimic eukaryotic linear motifs (ELM) and bind to human proteins harboring ELM-binding domains. The Laboratory of Computational & Functional Genomics has characterized Human HYPK protein and demonstrated how it may sense and interfere with aggregation prone proteins like Huntingtin.

Working with *Candida glabrata,* the Laboratory of Fungal Pathogenesis identified an iron permease CgFtr1, a multicopper oxidase CgFet3 and a copper transporter CgCcc2, and a mitochondrial

frataxin CgYfh1, as principal bonafide constituents of the high-affinity reductive iron transport and the iron metabolic apparatus, respectively, and furthermore established that high-affinity iron acquisition mechanisms are critical virulence determinants in the organism.The Laboratory of Plant-Microbe Interactions has studied the role of quorum sensing in regulating the production of *siderophore vibrioferrin* in *Xanthomonas oryzae* pv. *oryzicola,* which is required for in *planta* growth and virulence.

The work of Laboratory of Mammalian Genetics has dissected out the role of DNA methyltransferases *Dnmt3l* and *Dnmt2* in carcinogenesis and development. It has also identified epigenetic changes that the host cell undergoes when challenged with *Mycobacterium tuberculosis.* The Laboratory of Molecular Cell Biology report that ESAT-6 protein of *M. tuberculosis* interacts with host β2M and suppress class-I mediated antigen presentation. The group has also identified a novel IRAK3 signaling pathway downstream of TLR2 involved in regulation of anti- and pro-inflammatory responses by PPE proteins of *M. tuberculosis.*

Research at the Centre of Excellence in Silkmoth Genetics and Genomics has shown that the Bombyx sex chromosome Z is dosage compensated, with its expression just over half that of the autosomes. Formal permission has been received by the Centre for the conduct of multilocational trials in contained facilities of genetically engineered silkmoth strains.

The Laboratory of Neurospora Genetics found evidence for a recessive mutation that specifically affects alternate segregation, with apparently no effect on adjacent 1 segregation. The Laboratory of Transcription have deciphered the molecular basis of modulation of Rho-dependent transcription termination by the two transcription factors NusA and NusG, as well as the mechanism of conversion of NusA into an antiterminator by the bacteriophage protein N.

This year too as in previous years, several of the CDFD faculty and scholars have been recipients of prestigious awards and honours. The awards include, amongst others, the ICMR Basanti Devi Amir Chand Prize; fellowship under the Exchange of Scientists Programme between INSA and Royal Society, Edinburgh; Plenary Lecturer in the 6th FEBS Advanced Lecture Course on Human Fungal Pathogens; DST-SERB Fast track Young Scientist Award; Young Scientist Award from the AP Akademi of Sciences; Dr KV Rao Research Award; Shyama Prasad Mukherjee Fellowship; ASM Travel Award; ICMR Travel Grant; Prof GP Talwar Travel Bursary; DST travel grant etc. During this period, four research scholars were conferred with PhD degrees. Many postdoctoral fellows, project associates and summer trainees work at CDFD and play significant roles in the Centre's activities.

The Centre's permanent campus construction activities are progressing in full swing as per the plans approved earlier by the Expenditure Finance Committee of the Government.

I take this opportunity to acknowledge the unstinted co-operation which the Centre has received for its activities from the Governing Council, Research Area Panels-Scientific Advisory Committee, Academic/Finance/Building Committees and, of course, the Department of Biotechnology. I wish to thank all the members and officials for their time and effort in supporting our activities and achievements.

I also express my gratitude to the CDFD family who have played a crucial role in the ongoing programs at and development of the Centre.

J Gowrishankar

March 31, 2015

सेवाएँ
Services

LABORATORY OF DNA FINGERPRINTING SERVICES

Faculty	Madhusudan Reddy Nandineni	Staff Scientist
Other members	SPR Prasad	Senior Technical Officer
	Ch V Goud	Technical Officer
	Devinder Singh Negi*	Technical Officer
	Devinder Kumar	Technical Officer
	Sanjukta Mukerjee	Technical Officer
	S Naveenchandra	Technical Officer
	Neelima Thota	Technical Officer
	Pooja Tripathi	Technical Officer
	Joshi Kiranmai	Technical Officer
	Girnar Vijay Amrutarao	Technical Assistant
	Shruti Das Gupta	Technical Assistant
	Chandra Shekhar Singh**	Technical Assistant
Coordinator	DP Kasbekar	Haldane Chair

(*Posted at DNA Profiling Laboratory of CDFD (DPL-CDFD) at the Institute of Life Sciences, Bhubaneswar, Odisha State until 17 Nov. 2014)

(**Posted at DPL-CDFD at the Institute of Life Sciences, Bhubaneswar, Odisha State until 14 Aug. 2014)

Objectives

1. To provide DNA fingerprinting services in cases forwarded by law-enforcing agencies / judiciary of State and Federal Governments, relating to murder, rape, paternity, maternity, child swapping, body 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; and
5. To create DNA marker databases of different populations of India.

Summary of services provided until the beginning of this reporting year (upto March 31, 2014)

A total number of 403 cases were received for DNA fingerprinting examination during the previous reporting period (2013 – 2014). Of these 256 cases were related to identification of deceased, 73 cases were related to paternity / maternity, 46 cases were pertaining to sexual assault (rape), 23 cases were related to murder and 5 cases were pertaining to biological relationship (organ transplantation). Sixteen states, Union Territories of India and one foreign country (Timor Leste) have availed DNA fingerprinting services of CDFD during this period. Andhra Pradesh forwarded the highest number of cases (233) followed by Madhya Pradesh (53), Odisha (45), Chhattisgarh (18), Punjab (14), Delhi (7), Goa (6), Uttar Pradesh (6), Karnataka (5), Maharashtra (5), Bihar (2), Kerala (2), Puducherry (2), Uttarakhand (2), Andaman & Nicobar Islands (1), Jammu & Kashmir (1), and Democratic Republic of Timor-Leste (1).

Details of services provided in the current reporting year (April 1, 2014 – March 31, 2015

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

Biological relationship	014
Identity of deceased	280
Murder	013
Paternity/Maternity	101
Sexual assault (Rape)	151
Total number of cases	**559**

A total number of 559 cases were received for DNA fingerprinting examination during the current reporting period (2014 – 2015). Of these, 280 cases were related to identification of deceased, 151 cases were pertaining to sexual assault (rape), 101 cases were related to paternity / maternity, 14 cases were pertaining to biological relationship (organ transplantation) and 13 cases were related to murder. Eighteen States, Union Territories of India and one foreign country (East Timor) have availed DNA fingerprinting services of CDFD during this period. Madhya Pradesh forwarded the highest number of cases (197) followed by Andhra Pradesh (103), Telangana (79), Chhattisgarh (40), Odisha (29 cases, of which 18 were received at ILS, campus), Uttar Pradesh (29), Punjab (26), Goa (15), Tamilnadu (13), Karnataka (6), Puducherry (5), Kerala (4), Maharashtra (3), Delhi (2), Jammu & Kashmir (1), West Bengal (1) and Democratic Republic of Timor-Leste (1) (Fig. 1).

During this reporting period, an amount of **Rs.1,19,69,837/-** (Rupees one crore, nineteen lakhs, sixty nine thousand, eight hundred and thirty seven only) has been received towards DNA fingerprinting analysis charges, which is inclusive of service charge as levied by Govt. of India.

The cases involving identification of the deceased (50%), sexual assault (27%) and paternity/ maternity (18%) constituted the bulk of the cases received (Fig. 2).

Summary of the State-wise breakup of DNA fingerprinting cases

Name of the State	Biological relationship	Identity of deceased	Maternity / Paternity	Murder	Sexual assault (Rape)	Total No. of Cases
Andaman & Nicobar		1	1			2
Andhra Pradesh		99	4			103
Bihar		1	2			3
Chhattisgarh		19	18		3	40
Delhi		2				2
Goa		11	3		1	15
Jammu & Kashmir		1				1
Karnataka			6			6
Kerala		3	1			4
Madhya Pradesh	1	58	31	8	99	197
Maharashtra			3			3
Odisha		11	11	3	4	29
Puducherry		2	3			5
Punjab		2	1		23	26
Tamilnadu	11	1	1			13
Telangana	2	60	15	1	1	79
Uttar Pradesh		7	1	1	20	29
West Bengal		1				1
Timor Leste		1				1
Total No. of Cases.	**14**	**280**	**101**	**13**	**151**	**559**

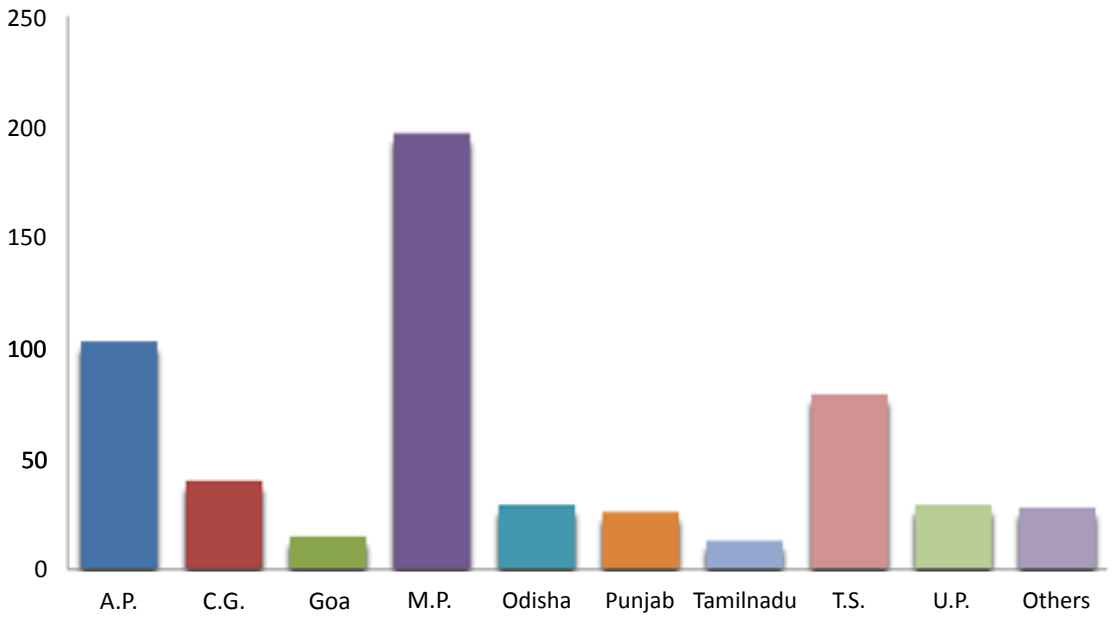

Figure 1. State-wise distribution of cases received during this reporting year.

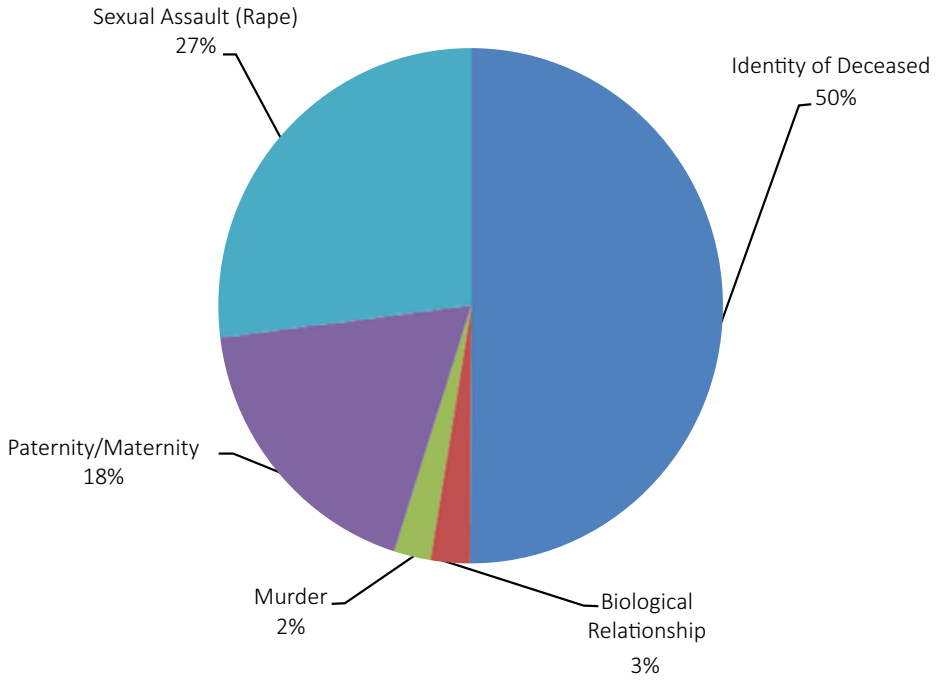

Figure 2. Types of cases received (%).

Prominent cases during April 1, 2014 to March 31, 2015

- Cases from National Investigation Agency (NIA) involving national security and public safety
- Sexual assault and homicide case of a Research Scholar in Agra, forwarded by the Central Bureau of Investigation (CBI), New Delhi
- Sexual assault and homicide case of two cousins in Badaun district of Uttar Pradesh, forwarded by the CBI
- Identification of victims of fire accident in crackers factory in Vishakhapatnam forwarded by the A.P. Forensic Science Laboratory, Hyderabad
- Identification of unknown human skeletal remains forwarded by the National Health Laboratory, Democratic Republic of Timor-Leste

Deposition of evidence in Courts of Law

During this reporting year, the DNA experts defended their reports in 14 cases in various Honorable Courts throughout the country.

Training/Lectures/Workshops on DNA fingerprinting examination

Training

1. Training on DNA fingerprinting techniques to personnel from Rajiv Gandhi Centre for Bio-Technology, Trivandrum, Kerala during 2-13 June 2014.
2. Training in Combined DNA Index System (CODIS) software procured from the Federal Bureau of Investigation (FBI), USA at CDFD for the benefit of DNA Examiners during 7-11 October 2014.

Lectures/Workshops

1. Lecture was delivered at CDFD for the benefit of the Post Graduate students from K. J. Somaiya College of Science and Commerce, Vidyavihar, Mumbai on 09.07.2014.
2. Lecture was delivered for the benefit of the Officers form National Investigating Agency, Hyderabad on 04.08.2014.
3. Lecture was delivered for the benefit of Police Officials at North Eastern Police Academy, Umsaw, Meghalaya on 18.11.2014.
4. Lecture was delivered at CDFD to faculty members from Criminology & Forensic Science School of Social Work, Kankandy, Mangalore on 03.12.2014.
5. Lecture was delivered at CDFD for the benefit of the Air Force Officers from Air Force Intelligence School, Lohegaon, Pune on 02.02.2015.
6. Lecture was delivered at CDFD for the benefit of the Post Graduate students from Modern College of Arts, Science and Commerce, Ganeshkhind, Pune on 03.03.2015.

Publications

1. Ballantyne KN, Ralf A, Aboukhalid R, Achakzai NM, Anjos MJ, Ayub Q, Balazic J, Ballantyne J, Ballard DJ, Berger B, Bobillo C, Bouabdellah M, Burri H, Capal T, Caratti S, Cárdenas J, Cartault F, Carvalho EF, Carvalho M, Cheng B, Coble MD, Comas D, Corach D, D'Amato ME, Davison S, de Knijff P, De Ungria MC, Decorte R, Dobosz T, Dupuy BM, Elmrghni S, Gliwiński M, Gomes SC, Grol L, Haas C, Hanson E, Henke J, Henke L, Herrera-Rodríguez F, Hill CR, Holmlund G, Honda K, Immel UD, Inokuchi S, Jobling MA, Kaddura M, Kim JS, Kim SH, Kim W, King TE, Klausriegler E, Kling D, Kovačević L, Kovatsi L, Krajewski P, Kravchenko S, Larmuseau MH, Lee EY, Lessig R, Livshits LA, Marjanović D, Minarik M, Mizuno N, Moreira H, Morling N, Mukherjee M, Munier P, Nagaraju J, Neuhuber F, Nie S, Nilasitsataporn P, Nishi T, Oh HH, Olofsson J, Onofri V, Palo JU, Pamjav H, Parson W, Petlach M, Phillips C, Ploski R, Prasad SPR, Primorac D, Purnomo GA, Purps J, Rangel-Villalobos H, Rębała K, Rerkamnuaychoke B, Gonzalez DR, Robino C, Roewer L, Rosa A, Sajantila A, Sala A, Salvador JM, Sanz P, Schmitt C, Sharma AK, Silva DA, Shin KJ, Sijen T, Sirker M, Sivakova D, Skaro V, Solano-Matamoros C, Souto L, Stenzl V, Sudoyo H, Syndercombe-Court D, Tagliabracci A, Taylor D, Tillmar A, Tsybovsky IS, Tyler-Smith C, van der Gaag KJ, Vanek D, Völgyi A, Ward D, Willemse P, Yap EP, Yong RY, Pajnic IZ and Kayser M (2014). Toward male individualization with rapidly mutating y-chromosomal short tandem repeats. *Human Mutation* 35: 1021-1032.
2. Parine NR, Lakshmi P, Kumar D, Shaik JP, Alanazi M and Pathan AAK (2015). Development and characterisation of nine polymorphic microsatellite markers for Tephrosia calophylla Bedd. (Fabaceae). Saudi Journal of Biological Sciences 22: 164–167.

DIAGNOSTICS DIVISION

Faculty	Ashwin Dalal	Staff Scientist
Adjunct Faculty	Prajnya Ranganath	Assistant Professor, NIMS
	Shagun Aggarwal	Assistant Professor, NIMS
PhD Students	Anusha Uttarilli	Senior Research Fellow
	Ashish Bahal	Senior Research Fellow
	Anjana Kar	Senior Research Fellow
	Deshpande Dipti Vijayrao	Junior Research Fellow (Since Jul. 2014)
Other Members	Aneek Das Bhowmik	Research Associate
	Maria Celestina Vanaja	Research Associate (Since Jan. 2015)
	Nillawar Anup Narayanrao	SIAMG Fellow (From Sep. 2014 till Feb. 2015)
	Sowmya Gayatri	SIAMG Fellow (Since Sep.2014)
	Matta Divya	Project-Junior Research Fellow
	P Rajitha	Technical Officer
	Angalena R	Senior Technical Officer
	Dutta Usha Rani	Technical Officer
	M Muthulakshmi	Technical Officer
	A Sobhan Babu	Technical Officer
	S Jamal Md Nurul Jain	Technical Officer
	S Vasantha Rani	Technical Officer
	C Krishna Prasad	Technician
	R Sudheer Kumar	Technician
	Rahila Qureshi	Laboratory Technician (From Feb. 2015 to Mar. 2015)

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; and
4. To impart training in genetic evaluation of patients with genetic disorders.

Details of services provided in the current reporting year (April 1, 2014 – March 31, 2015)

Clinical Genetics

A total of 3705 patient samples were analysed for genetic testing, during the year 2014-15. These consisted of patients with chromosomal disorders, monogenic disorders, mental retardation, congenital malformations, inborn errors of metabolism, and other familial disorders. A fellowship program for training in Clinical Cytogenetics and Clinical Molecular Genetics has been initiated in collaboration with Society for Indian Academy of Medical Genetics and one student each joined for the fellowship program.

The Department of Medical Genetics established at Nizam's Institute of Medical Sciences, Hyderabad is functioning successfully. A total of 2571 patients were examined and counseled in the unit during 2014-15. A 3 year training program for Diplomate of National Board (DNB) in Medical Genetics has been initiated with affiliation to National Board of Examinations, New Delhi. The entrance exams were held in December 2014 and two students have joined for DNB in Medical Genetics in April 2015.

Genetic investigations done during 2014-2015

Investigation	Total cases	Positives
Cytogenetics	1388	139 (10.01 %)
Proband	1260	133 (10.5 %)
Prenatal	0128	6 (4.7 %)
Molecular Genetics	1488	518 (35 %)
Proband	1398	499 (35.7 %)
Prenatal	0090	19 (21 %)
Biochemical Genetics	0829	249 (30 %)
Proband	0804	241 (30 %)
Prenatal	0025	8 (32.0 %)

Cytogenetics

Disease	Abnormality	No of cases
Down	47,XY,+21	34
Syndrome	47,XX,+21	15
	46,XY,rob(14;21) +21	5
	46,XX,rob(21;21) +21	1
	46,XX,rob(14;21)+21	1
	46,XX,rob(13;14)+21	1
	47,XX+21/46,XX	1
	47,XY,+21,9qh+	1
Edward	47,XX,+18	1
syndrome	47,SC,+18	2
Patau Syndrome	47,SC,+13	1
Turner syndrome	Monosomy X (45,X)	8
	mos 45,X/ 46,XY	4
	mos 45,X/46,X,i(X)	1
Klinefelter	47,XXY	11
Syndrome	47,SC	1
Triple X Syndrome	47,XXX	1
Sex reversal	Phenotypic female with 46,XY	1
	Phenotypic male with 46,XX	1
Aneuploidy	49,XXXXY	1

Structural chromosomal abnormalities

Inversions	
46,XX,inv(10)	1
46,X,inv(Y)	1
Deletions	
46,XY, 18p-	1
46,XY,del(5)(p)	1
46,XY,del(7)(p32)	1
Duplications	
46,XX,dup(4)	1
46,XY,add(17)(p12)	1
46,SC,add(15)(q23.4)	1
Translocations	
45,XY,rob(13;14)(p11.1;p11.1)	1
45,XX,rob(14;15)(q10;q10)	1

45,XX,t(14;21)(q10;q10)	1
46,XX,t(5;10)(p15.3;q24.3)	1
46,XX,t(1;22)(q25;q11.2)	1
46,XX,der(10),t(5;10)(q23;q26)	1
46,XX,t(10;11)	1
46,XY,t(1;10)	1
46,XY,t(5;11)(p15.3;p11.2)	1
46,XX,t(1;12)(p36.1;q13);inv9	1
46,XY,t(3;16)(q21;p13.3);inv(9)	1
46,XY,t(5;14)(p15.1;q12)	1
46,XY,der(5),t(5;10)mat	1
46,SC,der(5),t(5;10)mat	1
Polymorphic variants	25

Fluorescence in situ Hybridization (FISH)

Disease/translocation	Probe	No of tests
Prader-Willi Syndrome	SNRPN(15q11)/PML(15q24)	1
1p36 deletion syndrome	1p36 probe	3
Di-George Syndrome	TUPLE(22q11.2)/ARSA(22q13)	4
Marker chromosome	WCP-11, WCP-13, 9, 18 SE (X) (Y), Acro-p-arm	12
Spectral karyotyping		2

Quantitative Fluorescent PCR (QF-PCR)

MLPA	Cases	Positives
Prenatal (Aneuploidy)	65	2
Postnatal (Microdeletion syndromes)	70	12

Quantitative Fluorescent PCR (QF-PCR)

Disease/Test	Positives
Urine & Blood Metabolic Screening tests (N=232)	60
Amino acid disorders (N=183)	39
Non Ketotic Hyperglycinemia	13
Hyperornithinemia	5
Tyrosinemia	2
Phenylketonuria	1
Other amino acid disorders	18

Lysosomal storage disorders (N=389)	142
Hurler syndrome(17)	9
Hunter syndrome(21)	15
Sanfilippo B (19)	11
Morquio A disease (31)	28
Arylsulphatase B (13)	6
Sly disease (5)	0
GM1-Gangliosidosis (76)	8

Gaucher disease (23)	6
Krabbe disease (21)	3
Pompe disease (10)	4
Niemann Pick disease (35)	18
Mucolipidosis(12)	7
Metachromatic Leukodystrophy (65)	18
Fabry's disease(3)	0
Mannosidosis (4)	0

Hexosaminidase A/B (34)	
Tay Sachs disease	3
Sandhoff disease	6
Prenatal diagnosis (25)	8
Metachromatic Leukodystrophy	1
Hunter syndrome	1
Morquio A disease	2
GM1- Gangliosidosis	4

Molecular Genetics

Name of disorders	No of cases	Positive	Negative		
DMD/BMD	255	179	76		
DMD Carrier Analysis	34	09	25		
Spinal Muscular Atrophy	131	68	63		
SMA Carrier Analysis	56	27	29		
		Normal	Homozygous	Heterozygous	Compound Heterozygous
β thalassemia and Sickle cell anemia	151	11	88	33	19
Factor V Leiden	253	242	01	10	-
Factor II mutation	156	156	-	-	-
Cystic Fibrosis	113	100	08	05	-
Pancreatitis	15	11	02	02	-
Connexin 26	09	07	-	02	-
Achondroplasia	10	03	-	07	-
Hemophilia	15	10	04	01	-
Triplet Repeat Disorders		**Positive**	**Negative**		
Friedreich Ataxia	44	17	27		
Myotonic Dystrophy	22	17	05		
Huntington Disease	55	36	19		
SCA Panel (1,2,3,6 &7)	83	18	65		
DRPLA	10	02	08		
Fragile X Syndrome	76	04	72		

Prenatal Diagnosis	No of cases	Positive	Negative		
DMD	09	03	06	-	-
Spinal Muscular atrophy	12	02	10	-	-
Cystic Fibrosis	08	01	07	-	-
		Normal	Homozygous	Heterozygous	Compound Heterozygous
β thalassemia	61	10	09	38	04

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 (upto March 31, 2014)

Single gene disorders are rare by themselves but collectively they are an important cause of morbidity and mortality. The identification of genes for single gene disorders has value, not only in prenatal diagnosis and genetic counselling of affected families, but also in basic research towards understanding gene functions and mechanisms of disease. Till date more than 3000 genes causing single gene disorders have been identified using classical linkage analysis methods but still a large number remains to be characterized. The availability of massively parallel sequencing technologies have made it possible to identify gene for a particular disease using just a few affected individuals. 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 plan to employ exome sequencing to identify novel genes in such families.

Details of progress made in the current reporting year (April 1, 2014 – March 31, 2015)

We have performed exome sequencing for two families with rare autosomal recessive disorders. Proband in the first family had complex hand malformations, which have been reported as Camptosynpolydactyly (OMIM: 607539). Patient was born out of consanguineous union. This family also had a pregnancy with fetus showing similar features and hence terminated. Exome sequencing was done using Illumina platform followed by mapping of the reads to reference genome and detection of variants. Filtering for known SNPs, silent, homozygous variants revealed presence of 54 novel likely pathogenic (predicted) variants. Of these, the c.220_221delinsTT mutation leads to p.E74L change in BHLHA9 gene, which is predicted to be pathogenic. Earlier reports have implicated duplication of this gene in patients with split hand foot malformation. Missense mutations in BHLHA9 have been recently reported in patients with MSSD (Mesoaxial synostotic syndactyly with phalangeal reduction). Our patient is showing mutation in the same domain of the BHLHA9 gene as reported earlier in cases with MSSD. The reason for marked difference in phenotype is not clear but it appears that both MSSD and Camptosynpolydactyly are allelic disorders. Functional characterization is being planned to characterize the mutation and its effects on protein function.

Second family had two female siblings affected with microcephaly, macular degeneration and short stature and were born out of consanguineous marriage. The younger sibling was also diagnosed with Wilms tumor in kidney. Exome sequencing showed presence of 34 novel likely pathogenic (predicted) variants. Out of 34 variants a novel variant in BUB1B, c.1670G>T which leads to p.S557I, is predicted to be highly pathogenic. Mutations in BUB1B are known to cause Mosaic variegated aneuploidy syndrome 1 (MVA1, 257300). With the help of exome sequencing we could diagnose this rare disease in the patient and further characterization of this novel variant will throw light on functions of this protein.

Project 2: Clinical, biochemical and molecular analysis of lysosomal storage disorders.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

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 common lysosomal storage disorders.

Details of progress made in the current reporting year (April 1, 2014 – March 31, 2015)

Over last five years we have been able to identify mutations in 250 patients with different lysosomal storage diseases (LSDs) (Table 1). This study has revealed the mutation spectrum in patients with LSDs in the Indian population.

The novel mutations identified in the MPS VI patients were functionally characterized by use of molecular techniques of RNA isolation, Reverse transcriptase PCR & cDNA synthesis, cloning and site directed mutagenesis (SDM). COS-7 cells

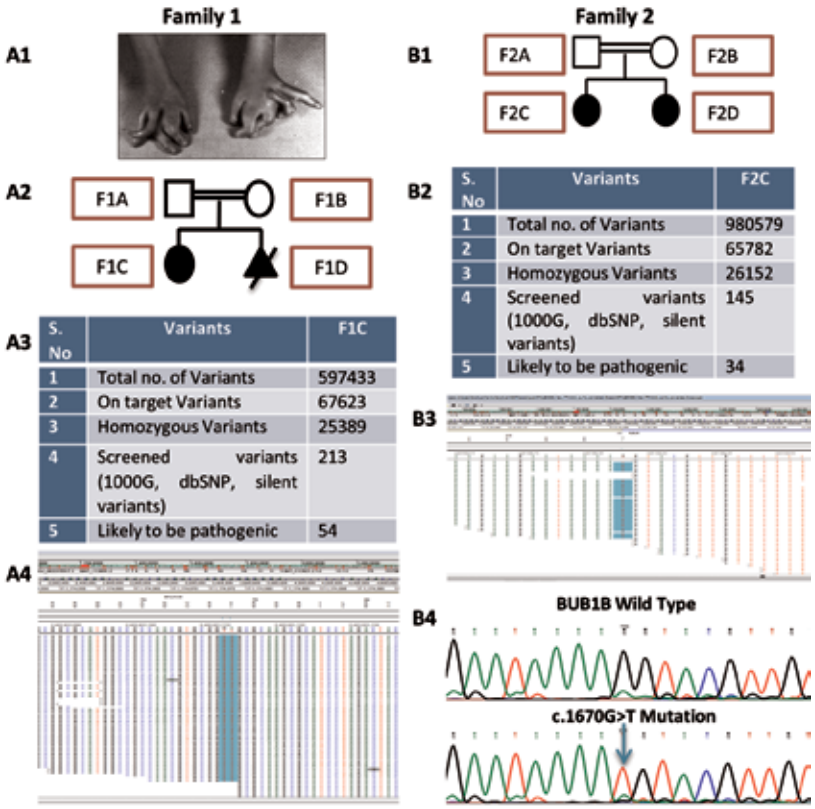

Figure 1. Candidate gene identification in families with rare autosomal recessive disorder.
(A1) Clinical picture in Family 1(Camptosynpolydactyly)
(A2) Pedigree of Family 1
(A3) Variant Statistics information of F1C from family 1
(A4) NEXTgene viewer report of BHLHA9 c.220_221delinsTT
(B1) Pedigree of Family 2 (microcephaly, macular degeneration and short stature)
(B2) Variant statistics information of patient F2C from family 2
(B3) NEXTgene viewer report of BUB1B c.1670G>T
(B4) Electrophoregrams showing normal and mutant sequence for c.1670G>T.

Lysosomal Storage Disorder	Gene	Number of cases	Total mutations	Novel mutations
Niemann-Pick disease types A & B	SMPD1	81	60	26
Metachromatic leukodystrophy	ARSA	79	56	23
Mucopolysaccharidosis I	IDUA	31	22	15
Mucopolysaccharidosis II	IDS	33	20	7
Mucopolysaccharidosis VI	ARSB	38	24	18
Sialidosis	NEU1	5	3	3
Total		**250**	**185**	**92**

Table 1. Data sheet showing mutation analysis for LSDs

maintained in DMEM media were transfected with plasmid DNA of wild type ARSB as well as other mutant cDNA clones constructed by SDM. Cells were harvested after 48 hrs of transfection and cell lysates were used for lysosomal ARSB enzyme assay. The enzyme assay performed for the COS7 cells with over expressed ARSB mutant clones, showed a significant reduction in

the enzyme activities when compared to the wild type ARSB clone. This suggests that the mutants severely affect function of the ARSB protein. For western blot analysis the cell lysate containing protein extract was subjected to SDS–PAGE (10% polyacrylamide) and transferred onto a PVDF Membrane. Western blot experiment was performed using standard protocol. The primary antibody was a polyclonal rabbit anti-ARSB and secondary antibody was a polyclonal goat anti-rabbit HRP labeled peroxidase-conjugated IgG prepared in 1 in 7000 dilution. The blot was developed by incubating the membrane in a luminol solution for 2 min at RT in dark. The chemi-luminescent membrane was exposed to chemifluorescence. Presence of the signal for specific band was checked. Most of the missense mutations, such as D53N, P445L, W450C, L98R, W450L, H393R and D54N showed presence of the full length mutated ARSB protein even though the enzyme activity levels were < 10-13 % of the wild type ARSB protein, whereas two mutants A237D and S320R showed very less amount of total ARSB protein indicating problems with synthesis, maturation or folding of the ARSB protein.

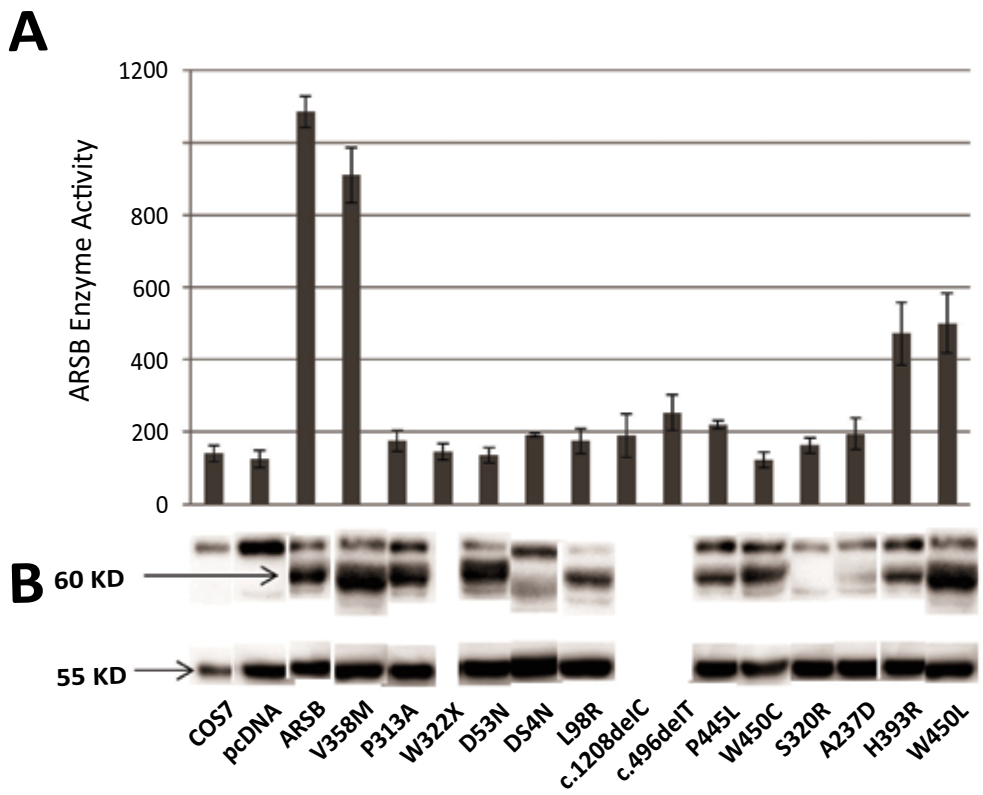

Figure 2. Functional characterization of mutations in ARSB gene

A. Graphical representation of enzyme activity in COS7 cells after transfection with cDNA mutant constructs (Y axisenzyme activity in nmol/hr/mg protein)

B. Western blot showing control lane 1 with untransfected COS7 cells, pcDNA 3.1 lane 2 with vector transfected COS 7 cells, pcDNA 3.1-ARSB lane 3 with over expressed normal COS7 cells and lane 3-14 for various mutants. 60 kD band corresponds to ARSB protein whereas the 55 kD corresponds to the tubulin loading control

Publications

1. Aggarwal S, Coutinho MF, Dalal AB, Mohamed Nurul Jain SJ, Prata MJ and Alves S (2014). Prenatal skeletal dysplasia phenotype in severe MLII alpha/beta with novel GNPTAB mutation. ***Gene*** 542: 266-268.

2. Bashyam MD, Chaudhary AK, Kiran M, Nagarajaram HA, Devi RR, Ranganath P, Dalal A, Bashyam L, Gupta N, Kabra M, Muranjan M, Puri RD, Verma IC, Nampoothiri S and Kadandale JS (2014). Splice, insertion-deletion and nonsense mutations that perturb the phenylalanine hydroxylase transcript

cause phenylketonuria in India. ***Journal of Cellular Biochemistry*** 115: 566-574.

3. Bashyam MD, Chaudhary AK, Kiran M, Reddy V, Nagarajaram HA, Dalal A, Bashyam L, Suri D, Gupta A, Gupta N, Kabra M, Puri RD, Ramadevi R, Kapoor S and Danda S (2014). Molecular analyses of novel ASAH1 mutations causing Farber lipogranulomatosis: analyses of exonic splicing enhancer inactivating mutation. ***Clinical Genetics*** 86: 530-538.
4. Bidchol AM, Dalal A, Shah H, S S, Nampoothiri S, Kabra M, Gupta N, Danda S, Gowrishankar K, Phadke SR, Kapoor S, Kamate M, Verma IC, Puri RD, Sankar VH, Devi AR, Patil SJ, Ranganath P, Jain SJ, Agarwal M, Singh A, Mishra P, Tamhankar PM, Gopinath PM, Nagarajaram HA, Satyamoorthy K and Girisha KM (2014). GALNS mutations in Indian patients with mucopolysaccharidosis IVA. ***American Journal of Medical Genetics*** 164A: 2793-2801.
5. *Chittem L, Bhattacharjee S and Ranganath P (2014). Craniosynostosis in a child with I-cell disease: the need for genetic analysis before contemplating surgery in craniosynostosis. ***Journal of Pediatric Neurosciences*** 9: 33-35.
6. Dalal A (2014). Molecular cytogenetic characterization of chromosomal rearrangem ents - utility in genetic counseling and research. ***Molecular Cytogenetics*** (Suppl 1): I12. doi.10.1186/1755-8166-7-S1-I12.
7. Dutta UR, Ponnala R and Dalal A (2014). A novel de novo balanced reciprocal translocation t(18;22) associated with recurrent miscarriages: a case report. ***Journal of Reproduction & Infertility*** 15: 113-116.
8. Dutta UR, Vempally S, Ranganath P and Dalal A (2014). A novel combined 15q11.2 duplication and a bisatellited supernumerary marker derived from chromosome 22: molecular characterization of the marker. ***Gene*** 539: 162-167.
9. Kantaputra PN, Kayserili H, Guven Y, Kantaputra W, Balci MC, Tanpaiboon P, Tananuvat N, Uttarilli A and Dalal A (2014). Clinical manifestations of 17 patients affected with mucopolysaccharidosis type VI and eight novel ARSB mutations. ***American Journal of Medical Genetics*** 164A: 1443-1453.
10. Kantaputra PN, Kayserili H, Guven Y, Kantaputra W, Balci MC, Tanpaiboon P, Uttarilli A and Dalal A (2014). Oral manifestations of 17 patients affected with mucopolysaccharidosis type VI. ***Journal Inherited Metabolic Diseases*** 37: 263-268.
11. Love JM, Prosser D, Love DR, Chintakindi KP, Dalal AB and Aggarwal S (2014). A novel glycine decarboxylase gene mutation in an Indian family with nonketotic hyperglycinemia. ***Journal of Child Neurology*** 29: 122-127.
12. Nandagopalan RS, Phadke SR, Dalal AB and Ranganath P (2014). Novel mutations in PRG4 gene in two Indian families with camptodactyly-arthropathy-coxa vara-pericarditis (CACP) syndrome. ***Indian Journal of Medical Research*** 140: 221-226.
13. Naushad SM, Krishnaprasad C and Devi ARR (2014). Adaptive developmental plasticity in methylene tetrahydrofolate reductase (MTHFR) C677T polymorphism limits its frequency in South Indians. ***Molecular Biology Reports*** 41: 3045-3050.
14. *Srinivas BH, Puligopu AK, Sukhla D and Ranganath P (2014). Rare association of spondylocostal dysostosis with split cord malformations type II: a case report and a brief review of literature. ***Journal of Pediatric Neurosciences*** 9:142-144.
15. Stephen J, Shukla A, Dalal A, Girisha KM, Shah H, Gupta N, Kabra M, Dabadghao P and Phadke SR (2014). Mutation spectrum of COL1A1 and COL1A2 genes in Indian patients with osteogenesis imperfecta. ***American Journal of Medical Genetics*** 164A: 1482-1489.
16. *Sukalo M, Fiedler A, Guzmán C, Spranger S, Addor MC, McHeik JN, Benavent MO, Cobben JM, Gillis LA, Shealy AG, Deshpande C, Bozorgmehr B, Everman DB, Stattin EL, Liebelt J, Keller KM, Bertola DR, van Karnebeek CD, Bergmann C, Liu Z, Düker G, Rezaei N, Alkuraya FS, Oğur G, Alrajoudi A, Venegas-Vega CA, Verbeek NE, Richmond EJ, Kirbiyik O, Ranganath P, Singh A, Godbole K, Ali FA, Alves C, Mayerle J, Lerch MM, Witt H and Zenker M (2014). Mutations in the Human UBR1 Gene and the Associated Phenotypic Spectrum. ***Human Mutation*** 35: 521-531.

17. Tsurusaki Y, Okamoto N, Ohashi H, Mizuno S, Matsumoto N, Makita Y, Fukuda M, Isidor B, Perrier J, Aggarwal S, Dalal AB, Al-Kindy A, Liebelt J, Mowat D, Nakashima M, Saitsu H, Miyake N and Matsumoto N (2014). Coffin-Siris syndrome is a SWI/SNF complex disorder. ***Clinical Genetics*** 85: 548-554.

18. Aggarwal S, Kar A, Bland P, Kelsell D and Dalal A (2015). Novel ABCA12 mutations in harlequin ichthyosis: A journey from photo diagnosis to prenatal diagnosis. ***Gene*** 556(2):254-256.

19. *Arora R, Aggarwal S and Deme S (2015). Ghosal hematodiaphyseal dysplasia-a concise review including an illustrative patient. ***Skeletal Radiology*** 44(3): 447-450.

20. Dalal AB, Ranganath P, Phadke SR, Kabra M, Danda S, Puri RD, VHS, Gupta N, Patil SJ, Mandal K, Tamhankar P, Aggarwal S and Agarwal M (2015). Prenatal diagnosis in India is not limited to sex selection. ***Genetics in Medicine*** 17: 88.

21. Das Bhowmik A, Rangaswamaiah S, Srinivas G and Dalal AB (2015). Molecular genetic analysis of trinucleotide repeat disorders (TRDs) in Indian population and application of repeat primed PCR. ***European Journal of Medical Genetics*** 58: 160-167.

22. Dutta UR, Hansmann I and Schlote D (2015). Molecular cytogenetic characterization of a familial pericentric inversion 3 associated with short stature. ***European Journal of Medical Genetics*** 58: 154-159.

23. *Gupta D, Gupta V, Singh V, Chawla S, Ranganath P and Phadke SR (2015). Study of polymorphisms in CFH, ARMS2 and HTRA1 genes as potential risk factors for age-related macular degeneration in Indian patients. ***International Journal of Bioassays*** 4: 3747- 3752.

24. Stephen J, Girisha KM, Dalal A, Shukla A, Shah H, Srivastava P, Kornak U and Phadke SR. (2015). Mutations in patients with osteogenesis imperfecta from consanguineous Indian families. ***European Journal of Medical Genetics*** 58: 21-27.

25. *Aggarwal S and Phadke SR. Medical genetics and genomic medicine in India: current status and opportunities ahead. ***Molecular Genetics and Genomic Medicine*** (In press).

26. Anusha U, Ranganath P, Jamal Md NJS, Krishna Prasad C, Anupam S, Verma IC, Phadke SR, Puri RD, Danda S, Muranjan MN, Jevalikar G, Nagarajaram HA and Dalal AB. Novel mutations of the ARSB gene in Indian patients with Mucopolysaccharidosis Type VI. ***Indian Journal of Medical Research*** (In press).

27. Bidchol AM, Dalal A, Trivedi R, Shukla A, Nampoothiri S, Sankar VH, Danda S, Gupta N, Kabra M, Hebbar SA, Bhat RY, Matta D, Ekbote AV, Puri RD, Phadke SR, Gowrishankar K, Aggarwal S, Ranganath P, Sharda S, Kamate M, Datar CA, Bhat K, Kamath N, Shah H, Krishna S, Gopinath PM, Verma IC, Nagarajaram HA, Satyamoorthy K and Girisha KM. Recurrent and novel GLB1 mutations in India. ***Gene*** (In press).

28. Dalal A, Aneek Das Bhowmik, Divya Agrawal and Phadke SR. Exome sequencing and homozygosity mapping help in identification of genetic etiology for spastic ataxia in a consanguineous family. ***Indian Journal of Medical Research*** (In press).

Other publications

1. Dalal A (2014). Phenylketonuria: Past, present and future. ***Genetic Clinics*** 7: 19-24.

2. Dutta UR (2014). Precision in chromosome identification with leads in molecular cytogenetics: an illustrated review. ***Journal of Pediatric Genetics*** Doi: 10.3233/PGE-14083.

3. Ranganath P and Rai GK (2014). Marfan Syndrome: Recent advances in diagnosis and management. ***Genetic Clinics*** 7: 6-10.

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* Partial work done in CDFD

APEDA-CDFD CENTRE FOR BASMATI DNA ANALYSIS

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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. Discovery and mapping of genomic regions governing economically important traits of Basmati rice.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

The work undertaken in earlier years under objective 2 has been summarized in the first part of the corresponding description below.

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

Objective 1: Testing the purity of Basmati samples received from Export Inspection Council (EIC), Ministry of Commerce, Govt. of India, Basmati rice exporters from India and other countries.

During the period under report, a total of 157 Basmati 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)

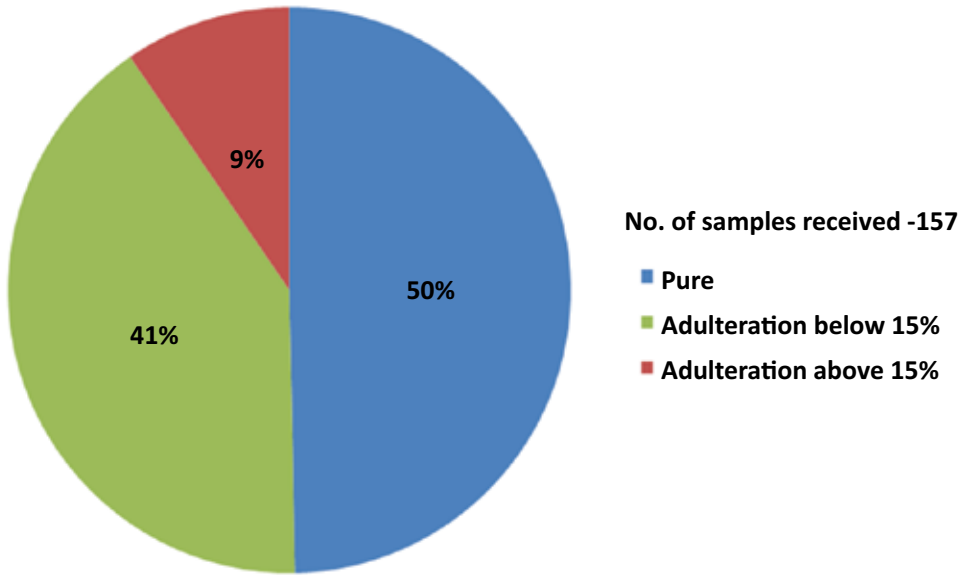

Figure 1. Basmati samples analyzed at APEDA-CDFD Centre in the current reporting year.

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:

i) Updating the database of Basmati varieties

At present our method covers eleven of the twenty varieties of Basmati rice that have been notified by Department of Agriculture and Cooperation (DAC) under Seeds Act, 1966. In view of the new adulterants challenging the existing method, we have extended our method of multiplexed eight markers panel analysis for identification of all the twenty notified varieties to generate a comprehensive database.

ii) Single grain analysis for varietal identification

On the unknown rice samples, where the sample was predominantly one variety, the identification using our standardized method is in good agreement. However, identification of rice varieties in samples of complex mixtures would require the use of a single grain assay. Due to the number of complex mixtures of samples being received, we have standardized single grain analysis to find out Basmati and adulterant varieties in the sample.

iii) Increase the number of SSRs in the panel for better resolution of complex mixtures and varietal identification:

With the constant release of new rice varieties, it becomes imperative to incorporate more number of SSR markers in the present assay. The SSRs selected should be such that they are highly discriminatory between the various rice varieties. In our ongoing research for expansion of the protocol, we have identified additional such SSRs for future use.

Objective 2: Discovery and mapping of genomic regions governing economically important traits of Basmati rice.

A total of 34 Quantitative Trait Loci (QTLs) for 16 economically important traits of Basmati rice were identified employing F_2, F_3 and Recombinant Inbred Line (RIL) mapping populations derived from a cross between Basmati 370 (traditional Basmati) and Jaya (semi dwarf rice). Out of which, 12 QTLs contributing to more than 15% phenotypic variance were identified and considered as major effect QTLs. Four major effect QTLs coincide with the already known genes viz., *sd1, GS3, alk1* and *fgr* governing plant height, grain size, alkali spreading value and aroma, respectively.

During the period under report, Basmati 370 rice DNA sequenced on SOLiD 4 was analyzed using Lifescope v2.5.1 software. Reads in xsq file format were mapped against Nipponbare complete rice genome sequence available at http://rice.plantbiology.msu.edu/. Alignment results were used to detect variations by variant caller algorithm. Based on the Basmati sequence data, candidate genes were identified for a few major QTLs namely *auxin response factor for filled grains, soluble starch synthase 3* for chalkiness and *VQ* domain containing protein for grain breadth and grain weight QTLs. These predictions were made based on non synonymous single nucleotide polymorphisms (nsSNPs) that were identified by comparing Basmati genome sequence with that of Nipponbare.

Publications

1. Archak S and Nagaraju J (2014). Computational analyses of protein coded by rice (*Oryza sativa japonica*) cDNA (GI: 32984786) indicate lectin like Ca2+ binding properties for Eicosapenta Peptide Repeats (EPRs). ***Bioinformation*** 10: 63-67.

2. Vemireddy LR, Satyavathi VV, Siddiq EA and Nagaraju J (2015). Review of methods for the detection and quantification of adulteration of rice: Basmati as a case study. ***Journal of Food Science Technology*** 52: 3187-3202.

Other Publications

1. Satyavathi VV (2014). International exposure to GM research. ***South Asia Biosafety Program Newsletter*** 11: 2.

शोध
Research

LABORATORY OF BACTERIAL GENETICS

Studies on gene regulation, transcription termination, and amino acid and ion-transport in Escherichia coli

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	Aanisa Nazir	Senior Research Fellow
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	Nida Ali	Junior Research Fellow
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	Swati Dubey	Junior Research Fellow (Since Feb. 2015)
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Other Members	VK Mishra	Staff Scientist
	K Anupama	Staff Scientist
	J Krishna Leela	Technical Officer
	TS Shaffiqu	Technical Officer
	Vimala Allada	Project Associate
	P Hima Bindu	Project Associate

The Laboratory of Bacterial Genetics comprises three faculty groups engaged in research 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 in Microbial Biology. The work undertaken in this reporting year is described below under the following objectives:

Objectives

1. To understand the pathology of RNA-DNA hybrids (R-loops) and the mechanisms for their avoidance;
2. Studies on a novel cryptic pathway for potassium translocation in *E. coli;*
3. Studies on basic amino acid export in *E. coli;*
4. To understand genetic interactions between (p)ppGpp and tm-RNA (SsrA)/SmpB;
5. To delineate the role of (p)ppGpp in cell division;
6. To understand consequences of accumulation of (p)ppGpp: relative toxicity of pppGpp versus ppGpp;
7. To use the *ilvGMEDA* operon as a paradigm to study the role of (p)ppGpp/DksA in transcription elongation; and
8. Role of transketolasesin *E. coli* physiology.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

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

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

1. Pathological consequences of R-loops and the mechanisms for their avoidance in *E. coli.*

The R-loop is a molecule in which single-stranded (ss-) RNA invades duplex DNA to base-pair with one of the DNA strands so that

the complementary DNA strand is displaced and rendered single-stranded. This laboratory has suggested earlier that nascent transcripts of protein-coding genes are prone to forming R-loops in the negatively supercoiled DNA region upstream of the moving RNA polymerase; and that such R-loop formation is generally avoided in *E. coli* by two mechanisms, namely, the immediate engagement of nascent mRNA by ribosomes (that is, transcription-translation coupling) and the premature termination of nascent transcripts that are not being simultaneously translated (that is, Rho-dependent transcription termination, which is mediated by the Rho and NusG proteins). In this model, antisense transcripts would also be prone to generating R-loops in the absence of Rho-dependent termination. We have previously obtained several lines of genetic evidence to indicate that the prevalence of R-loops is increased in mutants with inefficient Rho-dependent termination. The R-loops are distributed across the genome including from antisense transcripts, and ectopic expression of an R-loop helicase (UvsW, from phage T4) rescues the lethality associated with deletion of *rho* or *nusG* genes.

R-loops have also been proposed by Kogoma to provoke initiation of DNA replication, which is distinct from that ordinarily initiated at the *oriC* locus of the circular *E. coli* chromosome by action of the essential protein DnaA. Loss of RNase HI, an enzyme that removes R-loops, can restore viability to *dnaA* mutants and this is believed to be because of increased constitutive stable DNA replication (cSDR) from R-loops in these strains. Several putative *"oriK"* loci on the *E. coli* chromosome for R-loop mediated replication initiation have been reported.

We are at present engaged in attempts to understand the phenomenon of DnaA- and *oriC*-independent cSDR in *E. coli.* Based on our earlier finding that R-loops are distributed genome-wide (rather than at discrete loci), we have proposed that cSDR in a clonal population of bacterial cells is characterized by a widespread distribution of origins in the genome, each with a small firing potential. Since the gene organization on the circular chromosome has evolved such as to largely maintain co-directionality of transcription of the highly transcribed genes with replication fork movement, the latter being bi-directional from *oriC* to the antipodal terminus region where the action of a protein Tus creates a "replication fork trap" in an interval bounded by the *Ter* sequences to which Tus binds), cSDR is expected to suffer replication-transcription conflicts as some forks attempt to progress towards *oriC.* Furthermore, the postulated replication fork dynamics in these populations will explain the occurrence in two different mutants exhibiting cSDR (*rnhA* and *recG*) of a distinct peak of gene copy numbers in the chromosomal terminus region (as identified recently by two other groups).

In the present ongoing work, we are examining (i) whether other mutants that are expected to possess increased R-loop prevalence (for example, *rho* and *nusG* mutants, or *topA* mutants defective for topoisomerase I) exhibit cSDR; (ii) whether other mutants that have been reported to show a peak of gene copy numbers in the chromosomal terminus region (for example, mutants defective for three 3′-ss-DNA exonucleases, or for RecD) exhibit cSDR; (iii) the nature of a novel mutation in a laboratory collection strain that suppresses lethality associated with complete loss of DnaA; and (iv) the roles if any of the homologous recombination proteins RecA, RecBCD, and RuvABC in the different examples of cSDR. We are also developing a quantitative model to relate the abundance of R-loops at specific loci to four parameters, namely (i) promoter strength for transcript synthesis, (ii) efficiency of co-transcriptional ribosome engagement on the transcript (relevant for sense but not antisense transcription), (iii) efficiency of Rho-dependent termination of the untranslated transcripts, and (iv) sequence propensity for R-loop formation. Our analysis indicates that transcript abundance may be paradoxically reduced with increased promoter strength if the transcript is R-loop prone. The predictions from this model are being tested with the publicly available datasets on the abundance of sense and antisense transcripts in *E. coli,* and with our own earlier data on the distribution of R-loops across the genome.

Finally, since R-loops are known to exist in eukaryotic cells and to inflict genome damage in G1 phase we have also advanced the proposal that cSDR-like events may also promote aberrant replication initiation in these cells.

2. Studies on a novel cryptic pathway for potassium translocation in *E. coli.*

We have been examining a physiological link between potassium (K^+) metabolism and the paralogous PtsP-PtsO-PtsN phosphorelay system and have previously reported that

consistent with earlier reports a strain lacking PtsN, the terminal phospho acceptor protein, was progressively rendered K^+ sensitive (K^S) as the external K^+ concentration ($[K^+]_e$) was raised above 20 mM in a synthetic glucose minimal medium. The *ptsN* mutant however grew at rates comparable to the parent in a medium of low (1 mM) $[K^+]_e$. A growth inhibitory increase in intracellular K^+ content, resulting from hyperactivated TrkA-G/H mediated K^+ uptake is thought to be causal to this K^S. However our studies suggest that the K^S of the *ptsN* mutant paradoxically results due to a K^+ limitation occurring in media of $[K^+]_e$ > 20mM. We have shown that the moderate K^S displayed by the *ptsN* mutant was exacerbated in a derivative lacking the TrkA and Kup K^+ uptake proteins. Furthermore, overproduction of the K^+ uptake proteins TetA, a truncated KdpA polypeptide (KdpA′), Kup, and a K^+ transporting variant of the ammonia transporter AmtB, $AmtB^{H168D/H318E}$, suppressed the K^S of the *ptsN* mutant. Absence of the predicted inner membrane protein YcgO suppressed the K^S of the *ptsN* mutant and its overproduction while rendering its parent K^S, also displayed an exacerbated K^S phenotype in a strain lacking the Trk and Kup transporters. Similar to that seen for the *ptsN* mutant the K^S of *ycgO* overexpression was suppressed by overproduction of Kup. Lastly we had also found that levels of *ycgO* were comparable in the parent and its *ptsN* derivative. Accordingly the relationship between the *ptsN* mutation and its *ycgO* suppressor mutation on one hand and the K^S caused by YcgO overproduction on the other could be rationalized on the basis that in the parent either the phospho- or the depgospho-form of PtsN may fetter the activity of YcgO. It is thus hypothesized that K^S in the *ptsN* mutant may occur due to K^+ limitation resulting from activation of a pathway of K^+ release mediated by YcgO that is coactivated by $[K^+]_e$ > 20 mM and is normally rendered cryptic by phospho/dephospho-PtsN.

In this year we performed measurements of cellular K^+ content in the parent and the *ptsN* mutant and found that (i) the K^S of the *ptsN* mutant correlated with lowered cellular K^+ levels in the *ptsN* mutant in comparison to its isogenic wild-type parent that was observed during exposure to media of intermediate (40 mM) and high (115 mM) but not in media of low (1 mM) $[K^+]_e$, (ii) as mentioned above, the K^S of the *ptsN* mutant was found to be suppressed by overproduction of Kup, and this correlated with increased cellular K^+ content in the *ptsN* mutant, (iii) the absence of YcgO was associated elevated K^+ content in the *ptsN* mutant and correlated with the suppression of K^S of the *ptsN* mutant and (iv) the K^S phenotype associated with overexpression of *ycgO* also correlated with reduced cellular K^+ content and its suppression by overproduction of Kup was associated with increased cellular K^+ content. These observation lend support to the notion that the growth inhibition of the *ptsN* mutant in media of intermediate and high $[K^+]_e$ is associated with a K^+ limitation.

One aspect of the model for the K^S of the *ptsN* mutant described above, postulates that in the wild type strain either the phospho or the dephospho-form of PtsN may interact with YcgO to fetter its activity. To address this issue, currently we are testing for the probable interaction of the two forms of PtsN with YcgO in vivo. For this purpose we have generated plasmids encoding hexahistidine tagged versions of PtsN and its derivative bearing the H73A amino acid substitution. The H73A substitution renders PtsN in a constitutive dephospho-state. We are currently testing for the copurification of a chromosomally encoded epitope tagged YcgO with the two hexahistidine tagged PtsN variants.

3. Studies on basic amino acid export in *E. coli*

Towards studies on regulation of basic amino acid export in *E. coli* we have previously reported characterization of the ORF *yggA (argO)* that encodes a novel arginine (Arg) exporter ArgO in *E. coli*, whose expression is regulated by the transcription factor ArgP. Towards understanding the mechanism of Arg export mediated by ArgO we have previously conducted mutagenesis and second-site suppressor studies on ArgO and have assessed its topology in the inner membrane. Furthermore we have reported the identification of *ybjE* (*lysO*), a gene whose product mediates export of L-lysine (Lys) and another gene *ydhE* whose product appears to encode a second Arg exporter in *E. coli*.

Previously we had shown that the growth of the *ybjE* mutant was impaired in a medium containing the lysylalanyl dipeptide (Lys-Ala) which correlated with significantly elevated Lys content in the *ybjE* mutant in comparison to its parent. In this year, we demonstrated that overexpression of *ybjE* from a heterologous promoter yielded increased extracellular Lys in

the culture medium, in comparison to its haploid *ybjE*$^{+}$ (vector bearing) counterpart, following growth in a medium containing Lys-Ala. This observation is consistent with the notion that YbjE functions as a Lys exporter in *E. coli.* With regard to the genetic regulation of *ybjE* we had earlier reported the location of the core promoter elements of *ybjE* and had found that expression of a *ybjE-lac* transcriptional fusion was reduced two-fold in Arg supplemented minimal medium in an ArgR dependent manner whereas presence of Lys in the medium did not affect the magnitude of *ybjE-lac* expression. In this year we performed additional studies to delineate the basis of repression of *ybjE* expression by ArgR. We found that ArgR displayed Arg sensitive binding to the *cis* regulatory region of *ybjE* in vitro. Additional studies indicated that the binding site(s) for ArgR in the *cis* regulatory region of *ybjE* represented a weak ArgR binding site(s) since ArgR bound in an Arg sensitive manner with greater avidity to the *argF* DNA template that is known to bear a pair of classical ArgR binding sites (ARG boxes) We generated multiple DNA templates bearing site specific deletions and nucleotide substitutions within the *cis* regulatory region of *ybjE* and tested them for their interaction with purified ArgR. In addition we generated *ybjE-lac* transcriptional fusions bearing the aforementioned modifications in the *ybjE* promoter region to assess the in vivo effect of ArgR on their expression. These studies indicated that ArgR may bind at two sites located between -64 to -47 and -43 to -26 with the second site lying in an overlap with a core promoter element of *ybjE.* Thus ArgR at the *ybjE* promoter appears to exert its repressive effects by interfering with promoter binding of the RNA polymerase holoenzyme. One feature of the ArgR repression of *ybjE* expression was that the magnitude of *ybjE-lac* was not elevated by the absence of ArgR during growth in minimal medium which is in contrast to that seen for genes of Arg regulon that are significantly derepressed in an *argR* mutant. Since ArgR displayed weak binding to the *ybjE* promoter DNA, absence of derepression of *ybjE* expression in an *argR* mutant may be rationalized on the basis that titration of ArgR by other stronger ARG boxes on the chromosome may lead to a pre-existing derepression of *ybjE* expression. We also tested the effects of mutations in genes encoding transcription factors with known roles in Arg/ Lys metabolism, namely ArgP and LysR and we found that both ArgP and LysR exerted no regulatory effects on *ybjE-lac* expression.

In *E. coli*, ArgO, the ortholog of the LysE basic amino acid exporter of *Corynebacterium glutamicum,* has so far been thought to promote export only of Arg and its capacity to mediate Lys export is so far unknown. LysE on the other hand mediates export of both Arg and Lys. In *E. coli argO* expression is subjected to transcriptional regulation by Arg and Lys occurring through the transcriptional regulator ArgP with Arg and Lys mediating respectively induction and repression of *argO.* The expression of *lysE* in *C. glutamicum* is also under the transcriptional control of LysG, which is an ortholog of ArgP. In contrast to that seen for *E. coli* ArgP both Arg and Lys serve to stimulate *lysE* expression via LysG. It seemed probable that ArgO was also capable of mediating Lys export in *E. coli* however its Lys export capacity was normally rendered cryptic due to repression of its expression by Lys. To address this issue we tested whether overexpression of *argO* could mediate export of Lys. We used an *argP* allele bearing a dominant mutation encoding the ArgP^{P274S} substitution whose expression in vivo leads to Lys insensitive *argO* overexpression. Expression of ArgP^{P274S} but not ArgP, in the wild type strain promoted syntrophic cross-feeding of a *lysA* auxotroph and the property of ArgP^{P274S} to cross-feed was absent in a strain lacking ArgO. In addition, heterologous overexpression of *argO* rendered an *argO ybjE* double mutant resistant to the toxic analogue of Lys thialysine. These studies imply that ArgO bears a latent Lys export potential that is rendered cryptic due to Lys mediated repression of its expression by ArgP leading to perhaps a division of labour in the export of Arg an Lys in *E. coli,* a situation that is distinct from that seen in the case of *C. glutamicum,* where one protein LysE exports both Arg and Lys.

Earlier we had reported the isolation of null mutations in *ydhE,* encoding a predicted member of the multidrug and toxic compound extrusion (MATE) family of exporter proteins that rendered an *argO* mutant hypersensitive to the arginylalanine (Arg-Ala) dipeptide. We had initiated this work because we found that the *argO* mutant of *E. coli* was surprisingly not rendered sensitive to the Arg-Ala dipeptide. This observation is in contrast to that seen in *C. glutamicum* wherein a *lysE* mutant is rendered sensitive to both Arg-Ala and Lys-Ala dipeptides. Dipeptides provide a facile means of elevating

the cytoplasmic concentration of an amino acid following their catabolism to their constituent amino acids after their cytoplasmic uptake. The sensitivity of the *lysE* mutant to Arg-Ala and Lys-Ala is compatible with the role of LysE as an exporter of Arg and Lys. The apparent resistance of an *argO* mutant to Arg-Ala is indicative of existence of another mechanism(s) in *E. coli* that mediates resistance to the potential toxic effect of elevated cytoplasmic Arg concentrations attained by uptake of an Arg-containing dipeptide. Currently we are engaged in obtaining estimates of intracellular Arg levels in the parent, and the *argO* and *ydhE* single and double mutants upon their exposure to Arg-Ala.

4. Genetic interactions between (p)ppGpp and tm-RNA (SsrA)/SmpB.

In work described in earlier reports the synthetic lethal phenotype observed during the combined deficiency of (p)ppGpp and tmRNA or SsrA (synthetic lethality) was genetically characterized and the following were inferred,

a) Regulation of transcription by ppGpp alleviates the generation of non-stop mRNA by Rho-dependent transcription termination and prevents the stalling of ribosomes.

b) The ribosomes stalled in a $ppGpp^0$ strain require the SsrA/SmpB system for rescue, the absence of which leads to loss of cell viability.

c) Improved rate of translation in the presence of plasmid pRARE reduced the occurrence of Rho-dependent transcription termination and suppressed the synthetic lethal phenotype.

An important proof needed to support the inferences made above is to demonstrate enhanced Rho-dependent transcriptional polarity in $ppGpp^0$ strain. In results described below we have used northern blotting to study polarity in the $ppGpp^0$ strain. Taking advantage of the stability of *tRNA,* northern hybridisation was done using *lacZ-lacY-tRNA* hybrid mRNA system wherein *lacZ* and lacY' genes are fused to a *tRNA* reporter gene with the transcription under the control of *lac* promoter (Lopez *et al.,* 1994). In this construct, the *lacZ* gene is translated from the *lamB* RBS which is nearly equivalent in efficiency to that of *lacZ* RBS and is followed by a truncated version of *lacY* gene, making this construct tri-cistronic.. The tRNA used here is $tRNA^{Arg5}$, which recognizes the rare arginine codon AGG and ranks amongst the least abundant in *E. coli,* therefore, its genuine expression interferes minimally with that of the reporter. An 'A' to 'U' substitution at the nucleotide 73 of the reporter tRNA decreases its capacity to load arginine without disrupting its structure. In addition, this allows the discrimination between the reporter and the genuine $tRNA^{Arg5}$ by hybridization. As expected, we find an IPTG dependent appearance of signal and the signal is lower in the $ppGpp^0$ strain as compared to that in the WT strain (Fig. 1A). To find out if decreased tRNA expression in the (p) $ppGpp^0$ strain arises from transcriptional polarity we used bicyclomycin an antibiotic that inhibits Rho protein and asked if expression is restored in the (p)$ppGpp^0$. tRNA expression in the $ppGpp^0$ strain was indistinguishable from that seen in the wild type strain which is consistent with the idea of Rho-dependent transcription termination being responsible for the decreased in $tRNA^{Arg5}$ expression in the $ppGpp^0$ strain.

We also find that plasmid pRARE which suppressed the synthetic lethal phenotype rescued transcriptional polarity seen in the $ppGpp^0$ strain (Fig. 1B) consistent with an idea that increased rate of translation in the presence of plasmid pRARE inhibits Rho-mediated termination by facilitating transcription-translation coupling in the $ppGpp^0$ strain.

5. (p)ppGpp and modulation of cell division.

In the previous report we had documented the synthetic lethality of $ppGpp^0$ *lon* mutant. Our studies suggested that SulA-mediated inhibition of FtsZ could be the probable cause of lethality although we did not find evidence for increased *sulA* expression in the (p)$ppGpp^0$ strain.

In work carried out this year, we have gathered more evidence in support of this idea, (i) over-expression of FtsZ suppressed lethality; (ii) microscopy revealed extensive filamentation in $ppGpp^0$ *lon* strains associated with loss of cell viability; (iii) western blotting showed a reduction in FtsZ level in the $ppGpp^0$ strain. These results indicate that (p)ppGpp levels could be important for the protection of cellular division machinery under growth conditions that stress the Lon protease machinery leading to an increase in SulA concentration. We think (p)ppGpp could be important to prevent FtsZ concentration from dropping below the threshold required for SulA inhibition and this could be mediated through the

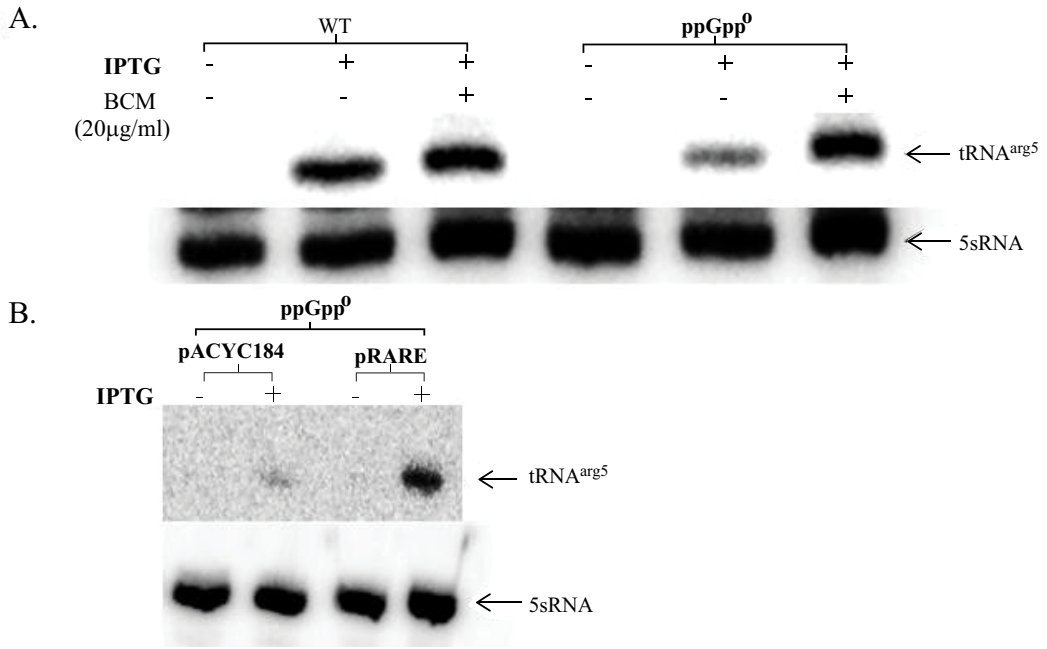

Figure 1. Inhibition of transcriptional polarity by (p)ppGpp and its rescue by bicyclomycin and plasmid pRARE. Expression of $tRNA^{arg5}$and 5sRNA were monitored by northern blotting in the WT and $ppGpp^0$ strainsin the presence or absence of IPTG/bicyclomycin (BCM) (A), in the $ppGpp^0$ strain carrying plasmids pACYC184 or pRARE in the presence or absence of IPTG (B).

positive regulation of FtsZ and /or the Lon protease activity.

6. Accumulation of (p)ppGpp: relative toxicity of pppGpp versus ppGpp.

spoT is an essential gene, and genetic evidence indicate the essential function of SpoT is the degradation of (p)ppGpp. In work described previously we tested this idea by screening for mutations that allow survival of *spoT* deletion. Two mutations, one that truncates the *rel*A-ORF after the 496th codon (Δ*relA*496) and another at the end of the *rlmD* ORF that precedes the *relA* ORF were identified.

In the current year, we characterized the (p)ppGpp accumulation pattern in strains bearing the Δ*relA*496 allele in *spoT*$^+$ and Δ*spoT* background and found higher ppGpp content in the Δ*relA*496 Δ*spoT* strain (compared to Δ*relA*496 *spoT*$^+$) consistent with the resistance displayed by this strain to SMG (serine, methionine and glycine). The results show that synthesis of (p)ppGpp per se is not detrimental to the growth of Δ*spoT* strain. Using a system designed to deplete SpoT protein in the Δ*spoT* strain we followed intracellular (p)ppGpp content in the strain during growth in the absence of starvation. We found gradual accumulation of ppGpp but not pppGpp to accompany the reduction in growth rate. To ask if pppGpp is rapidly converted to ppGpp through GppA (guanosine penta phosphate hydrolase), we deleted *gppA*. Interestingly, the Δ*spoT gppA* mutant was much more sensitive to SpoT depletion (relative to Δ*spoT* strain) as seen from a rapid onset of growth inhibition; accumulation of pppGpp in addition to ppGpp was seen. These results indicate that the accumulation of pppGpp is toxic under these growth conditions and two proteins, namely, SpoT and GppA proteins are redundantly engaged in the removal of this molecule. We also found the viability of the Δ*relA496* Δ*spoT* strain was dependent on GppA, again an indication of toxicity associated with the accumulation of pppGpp.

7. Using the *ilvGMEDA* operon as a paradigm to study the role of (p)ppGpp/DksA in transcription elongation

The addition of amino acids serine, methionine and glycine to minimal glucose media provokes limitation for amino acids isoleucine and valine; strains capable of mounting a stringent response (*relA*$^+$) or having elevated (p)ppGpp or DksA levels adapt by regulating transcription of the *ilvGMEDA* operon to

overcome the limitation (SMG-r); conversely Δ*relA* strains do not grow (SMG-s). The presence of a frame-shift mutation early in the *ilvG* ORF confers transcriptional polarity and is required for the SMG-s phenotype of Δ*relA* strain; mutations that reduce Rho activity confer SMG-r phenotype in Δ*relA* strain. These results raise the possibility that (p)ppGpp/ DksA could modulate expression of the *ilv* operon. Since many reports implicate (p)ppGpp /DksA in the regulation of transcription elongation,we have made transcriptional lac fusions at the *ilvG* and *ilvM* loci and will use it to study the effect of (p)ppGpp/ DksA on transcripts originating at the *ilvG* promoter. We plan to explore the role of (p)ppGpp/DksA in attenuation using the *ilvG-lac* fusion and their effects on elongation/ polarity by comparing the expression of *ilvG-lac* with *ilvM-lac* fusion. Preliminary results obtained using the fusions verify our expectation of polarity relief in *rho* mutant.

8. Transketolase activity regulates glycerol metabolism: Inhibition of glycerol assimilation by Ribose-5-P.

Transketolase activity provides an important link between the metabolic pathways of glycolysis and pentose phosphate shunt. It is widely conserved in life forms and catalyzes inter-conversions between pentose phosphates and glycolytic intermediates. A genetic screen for suppression of the growth defect associated with *tktA tktB* double mutant in LB revealed two mutations, one that rendered the *glpK* expression constitutive and another that inactivated *deoB.* Characterizing these mutations aided in identifying the role of ribose-5-P in the inhibition of glycerol assimilation. Using lacZ fusions, we show that ribose-5-P inhibits the assimilation of glycerol by enhancing GlpR – mediated repression of the *glpFKX* operon. EMSA assays revealed that in the presence of ribose-5-P, dissociation of the DNA-GlpR complex is less sensitive to the inducer glycerol-3-P. In addition to inhibition of glycerol assimilation, ribose-5-P confers glycerol-3-P limitation during growth in casamino acids wherein glycerol-3-P is synthesized *de novo.*

Publications

1. Phulera S, Akif M, Sardesai AA and Mande SC (2014). Redox proteins of Mycobacterium tuberculosis. ***Journal of the Indian Institute of Science*** 94: 127-137.

2. Gowrishankar J (2015). End of the beginning: elongation and termination features of alternative modes of chromosomal replication initiation in bacteria. ***PLoS Genetics*** 11: e1004909.

LABORATORY OF CELL CYCLE REGULATION

Elucidating the role of effector proteins in G1 to S phase progression

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	Swathi Chodisetty	Senior Research Fellow
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	Mallikharjuna Rao	Project-Junior Research Fellow (Since Oct. 2014)
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	SJ Yogaspoorthi	Project Assistant (Till Jul. 2014)

Objectives

1. Identification of new effector proteins involved in regulation of E2F-responsive promoters; and
2. Study of chromatin modifying proteins in cell cycle regulation.

Project 1: Identification of new effector proteins involved in regulation of E2F-responsive promoters.

One of the major roles of E2F proteins is to regulate the transition from G1 to S phase. However, how E2Fs affect passage into S phase is still poorly understood. In this project we aim to identify new effector proteins involved in regulation of E2F-responsive promoters and better understand how these effectors influence transcriptional regulation during G1 to S phase progression.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

We cloned and expressed E2F4 deletions as GST-fusion protein. These can be utilized later to map the domain of E2F4 that associates with E2F4 interacting proteins. We also expressed E2F4 as triple-epitope-tagged fusion protein for tandem affinity purification from HeLa spinner cells.

Details of progress made in the current reporting year (April 1, 2014 –March 31, 2015)

E2F1 has been shown to associate with multiple members of HMT family either directly (Takeda *et. al,* Genes Dev. 2006) or indirectly (Tyagi et al., Mol Cell. 2007) to bring about the H3K4 trimethylation and activation of S phase promoters. One important observation in these studies was that E2F-responsive promoters undergo dynamic H3K4 trimethylation during the cell cycle. Until recently it was believed that H3K4me3 is an irreversible modification. But with discovery of four H3K4me3 demethylase namely RBP2, SMC-X, SMC-Y, and PLU-1 this theory changed. We wish to identify the H3K4 tri demethylase which removes the H3K4me3 residues on the E2F-responsive promoters and characterize its role in E2F-mediated transcription.

To start our study, we took the directed candidate approach. We initiated our studies with Retinoblastoma binding protein 2 (RBP2), as the antibodies to this protein were already available in the laboratory. Further a recent report suggests that RBP2 is a part of Sin3 core repressor complex and causes permanent silencing of a subset of E2F4 targets during muscle differentiation (van Oevelen *et al.,* Mol. Cell. 2008). Also genome-wide location screen of

RBP2 showed that it is associated with a large number of PcG target genes in mouse ES cells. The study also showed that genome wide targets for RBP2 include a subset of genes that have role in cell cycle regulation and cell proliferation (Pasini et al., Genes Dev. 2008).

We used GST-E2F4 to check whether it interacts with RBP2. In order to ensure that the interaction was specific to E2F4 and not GST, we used GST alone for control pull down. The bead-bound GST-E2F4 or GST was incubated with HeLa cell nuclear extract and probed for

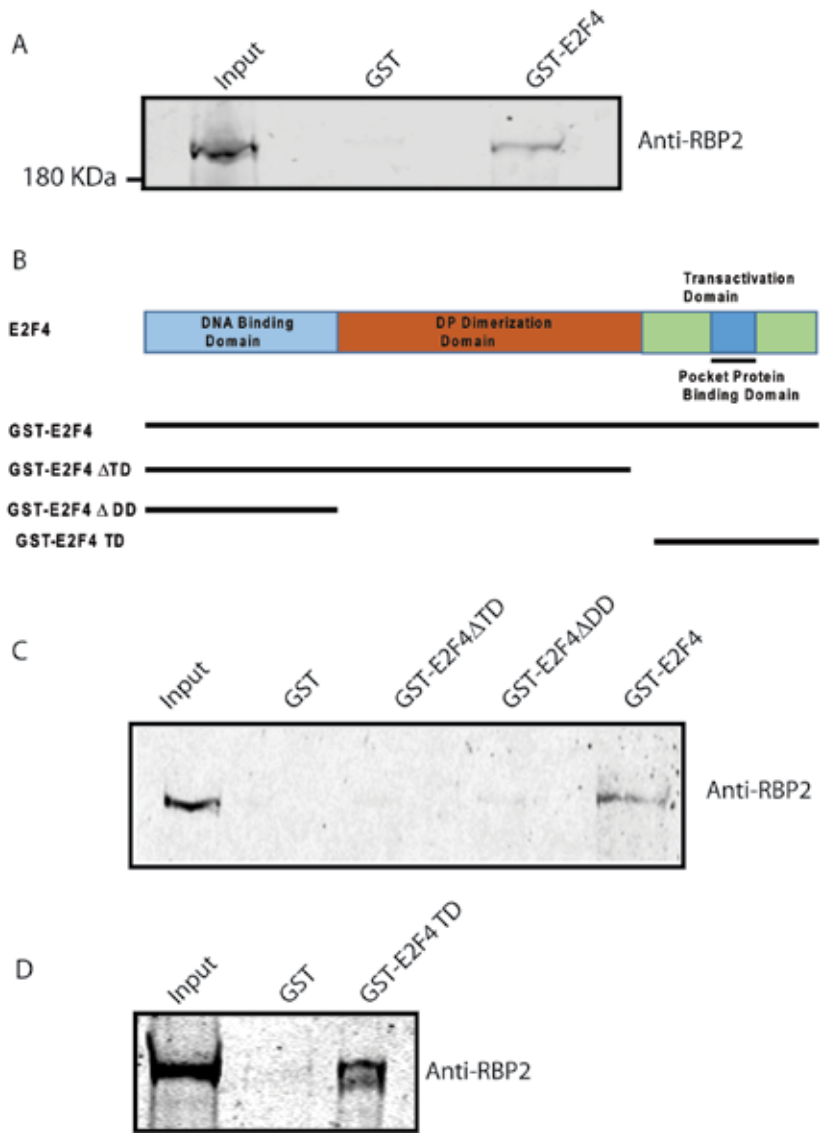

Figure 1. E2F4 interacts with RBP2.

- A Pull down from GST-E2F4 and GST probed with anti RBP2 antibody shows RBP2 interacts with E2F4.
- B Schematic of E2F4 deletions. DD, dimerization domain; TD, transactivation domain.
- C Pull down from GST-E2F4, E2F4 deletions and GST probed with anti RBP2 antibody shows that RBP2 interacts only with full-length E2F4.
- D The interaction of GST-E2F4 TD and GST with RBP2 are shown.

RBP2 after the beads were washed and used for immunoblotting. RBP2 was clearly detected in the GST-E2F4 beads but not in GST alone, indicating that the RBP2 interacted specifically with E2F4 (Fig 1A).

In order to map the region of E2F4 that interacted with RBP2, we made use of our GST-tagged E2F4 deletion (Fig 1B). These GST fusion proteins were incubated with nuclear extract as mentioned before and analyzed for RBP2 binding. Surprisingly, none of the two deletions showed any binding to RBP2 indicating that either the transactivation domain (TD) is important for this interaction or only full-length E2F4 interacts with RBP2 (Fig 1 B and 1C). To test these possibilities, we made a GST fusion of E2F4 TD and checked its interaction with RBP2 (see Fig. 1B and 1D). As shown in Fig 1D, GST-E2F4 TD was able to pull down endogenous RBP2. Interestingly, this region of E2F4 also interacts with pRB homologue, p130 raising the possibility that the E2F4-RBP2 interaction may not be direct.

Project 2: Study of chromatin modifying proteins in cell cycle regulation.

Histone 3 lysine 4 trimethylation is linked to active gene expression, but its precise role in cell cycle regulation is now being uncovered. We have shown that MLL-family of H3K4 histone methyltransferases is linked to cell cycle regulation by interacting with E2F1. In this project, we are looking at other roles of this chromatin-modifying complex that may influence cell cycle regulation.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

To find out the role of MLL complex in cell cycle regulation, in our previous reports we show that MLL has a regulatory role during multiple phases of the cell cycle. RNAi mediated knockdown revealed that MLL regulates S phase progression and, proper segregation and cytokinesis during M phase. Using deletions and mutations, we had narrowed the cell-cycle regulatory role to the C subunit of MLL.

Details of progress made in the current reporting year (April 1, 2014 –March 31, 2015)

SET 1 family members have overlapping as well as unique functions. Our previous results show that loss of both MLL and WRAD results in S and M phase progression defects but, while WRAD may have a role in the M phase functions of MLL, their involvement in S phase functions of MLL is not clear. Further, mutations in WDR5 show that Y191F, which is capable of interacting with MLL, rescues M but not S phase progression defect induced by loss of WDR5. WRAD components complex with other SET1 family members as well and Y191 may have a role in WDR5's interactions with other MLL members (Zhang et al., Nucleic Acids Res. 2012; Dharmarajan *et al.,* J. Biol. Chem. 2012). In order to clarify if other SET members participated or duplicated the functions of MLL in cell cycle progression we undertook further experiments.

We used two different siRNAs to deplete Set1A, from the SET 1A/ SET 1B group; and MLL3 from the MLL3/MLL4 group. Even though, we have studied the effects of MLL from MLL/MLL2, we still choose MLL2 to confirm our findings. We also used the stratergy employed by Wang and colleagues (Wang *et al.,* Mol. Cell. Biol. 2009) and targeted Menin, PTIP or WDR82 mRNA to substantiate our RNAi experiments with MLL/ MLL2, MLL3 and Set 1A complexes respectively.

Loss of Set1A, MLL2 and MLL3 resulted in pronounced and almost similar loss in cell proliferation as observed by failure of BrdU uptake by siRNA treated cells (Fig 2A). In agreement with these results, RNAi of Wdr82, Menin and PTIP also showed reduction in BrdU incorporation (Fig. 2B).

In contrast, when assayed for mitotic defects, only samples treated with Set1A siRNA displayed obvious phenotype, and not MLL2 or MLL3 siRNA-treated samples (Fig 2C). Similarily, knockdown of Set1A protein complex component WDR82 displayed considerable cells with mitotic defects but not Menin or PTIP (Fig 2 D). Together, our results suggest that while all MLL complexes play a role in regulating S phase progression, only MLL and SET 1A are the major protein complexes responsible for facilitating M phase progression.

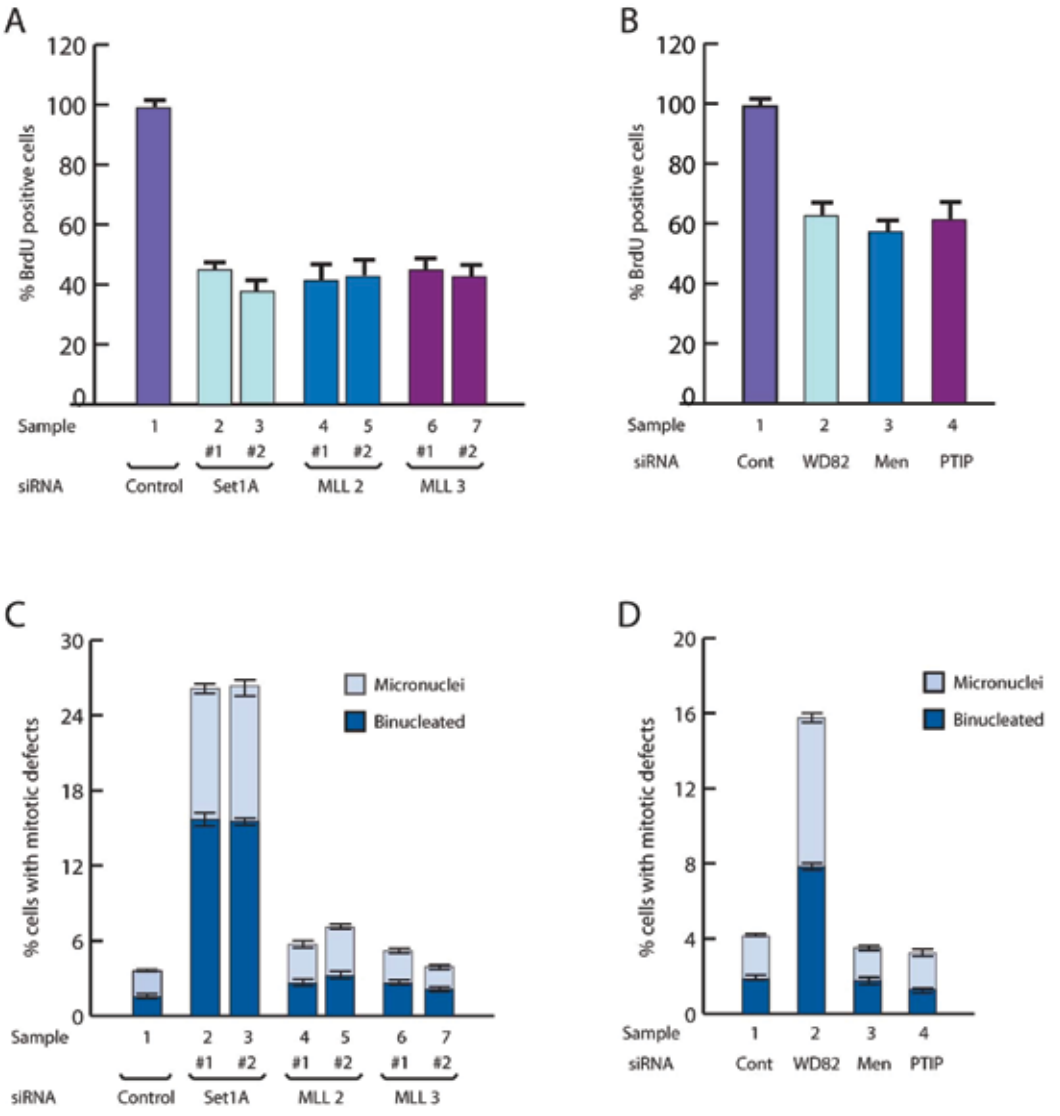

Figure 2. Set1 family regulates cell growth and mitosis.

A-D. Different members of Set1 family were knocked down using siRNA and BrdU incorporation assay **(A-B)** and mitotic defects analyses **(C-D)** were done in U2OS cells as indicated. **(A,C)** #1 and #2 denote two different siRNAs used. Cont, Control; Men, Menin; Wd82, WDR82. Significant p- value (< 0.01) were obtained with Student's t-Test (C-D).

Publications

1. Ali A, Veeranki S N, and Tyagi S (2014). A SET domain-independent role of WRAD complex in cell cycle regulatory function of mixed lineage leukemia. ***Nucleic Acids Research*** 42: 7611-24.

LABORATORY OF CELL DEATH & CELL SURVIVAL

Functional protein networks controlling cell life and death

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	HA Nagarajaram	CDFD, Hyderabad

Objectives

1. To dissect the functional network of phosphatases regulating cell life and death; and
2. To understand the cellular functions of canonical and non-canonical ubiquitination.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

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 initiated our studies on functional phosphatase network with the identification of interacting partners of every phosphatase in the cell. In this work we have already identified and characterized WWP2, PNUTS and TOPK as novel functional partners of PTEN (Maddika *et al.,* Nature Cell Biol. 2011, Kavela *et al.,* Cancer Res. 2013, Shinde *et al.,* Cell Signal 2013). In addition to PTEN interactome, we also started developing networks of other cellular phosphatases. We found a deubiquitinase complex WDR48-USP12 as a regulator of another tumor suppressor phosphatase PHLPP1 (Gangula NR & Maddika S., JBC 2013). Also, 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 (Chaudhary N & Maddika S., Mol Cell Biol 2014).

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

Project 1: Functional studies on phosphatase networks.

Currently, we are focused on actively expanding the network of all the available phosphatases in cell. So far we cloned 145 phosphatases into a triple tagged mammalian expression vector and confirmed their expression in cells. By using tandem affinity purification approach followed by LC-MS/MS analysis we identified the associated protein complexes of 142 phosphatases until now. After filtering out the common contaminants using control GFP purification and internal comparisons, we used Significance Analysis of Interactome (SAINT express) algorithm to score protein-protein interactions. By using a relatively stringent SAINT score cut off of 0.9, we scored about 9900 high confident interactions for all the purified phosphatases. While we aim to build the whole phosphatase network, we

simultaneously started to characterize several of putative functional interactions of these purified phosphatases.

1.1. PTEN modulates EGFR endocytic trafficking by dephosphorylating Rab7

PTEN is a major tumor suppressor that acts to down-regulate cell proliferation, survival and metabolic signaling pathways. Though it is originally identified as a dual specificity protein phosphatase, majority of its tumor suppressor function is attributed to its lipid inositide phosphatase activity. However, several tumor derived PTEN mutants that specifically lack protein phosphatase activity has been identified highlighting the need to identify lipid phosphatase independent cellular functions of PTEN. In this study, we discovered a novel functional role of PTEN in regulating endocytosis. We demonstrated that PTEN attenuates EGFR signaling by promoting late endosome maturation by virtue of its protein phosphatase activity. Loss of PTEN impairs the transition of EGF/EGFR to late endosomes (Fig. 1A) thus leading to their

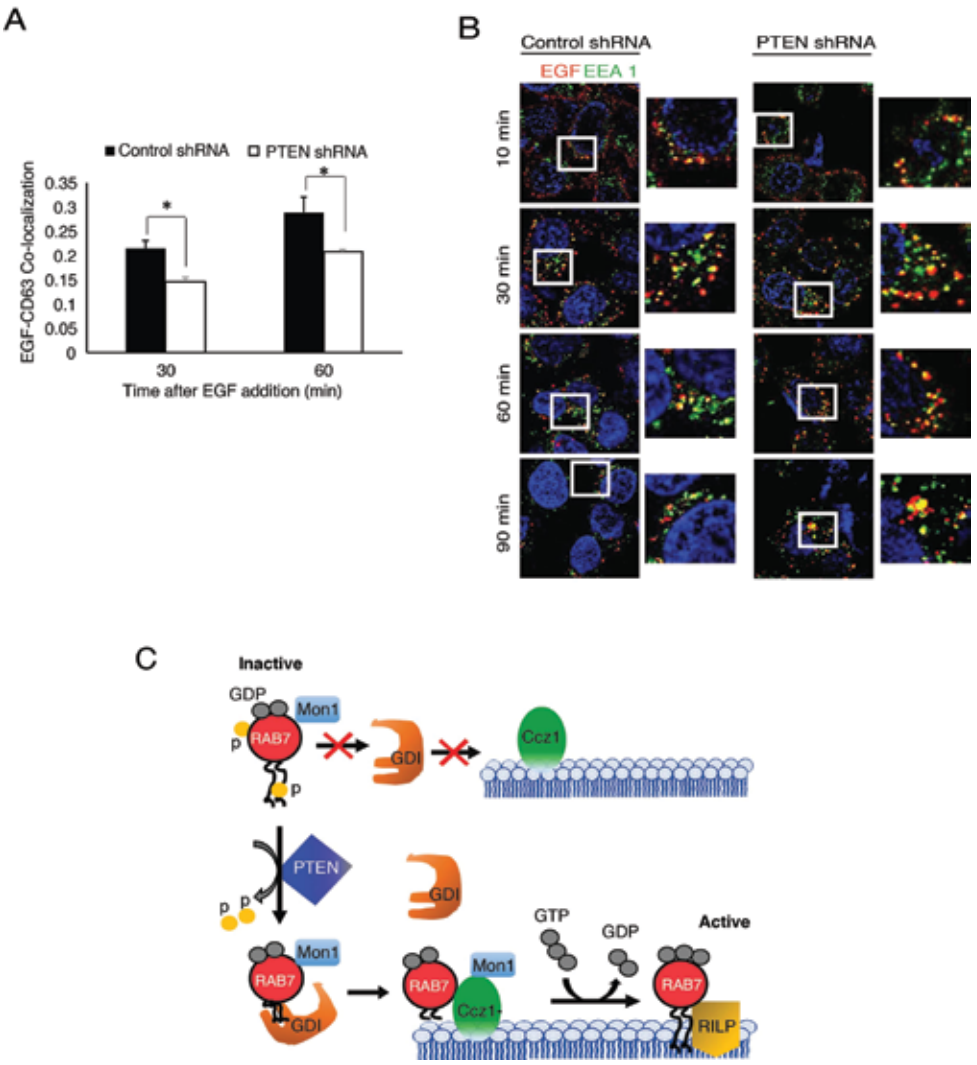

Figure 1. PTEN modulates EGFR endocytic trafficking by dephosphorylating Rab7 (A) The presence of EGF in late endosomes was analysed by co-staining with CD63 antibodies and the extent of their co-localization in control and PTEN shRNA cells was plotted (n=100 cells for each time point), *P<0.05. **(B)** The presence of EGF in early endosomes was analysed by co-staining with EEA1 antibodies. **(C)** Schematic representation of a model to show the role of PTEN in controlling Rab7 activation at late endosomes

accumulation in early endosomes (Fig. 1B). Interestingly, expression of wild type PTEN, but not catalytically inactive C124S and Y138F mutants could rescue the EGFR degradation in PTEN null MDA-MB-468 breast cancer cells, thus underlining the importance of PTEN protein phosphatase activity in this process. Tandem affinity purification followed by mass spectrometry analysis enabled us to identify Rab7 as a PTEN-interacting protein. Rab7, a member of the Ras superfamily of GTPases, is critical for the transit of early endosomes to late endosomes and the transfer of cargo from late endosomes to lysosomes and thus regulates the lysosome-mediated degradation of activated EGF receptors. We found that PTEN dephosphorylates Rab7 on two conserved residues S72 and Y183. In conclusion, we identified that PTEN dephosphorylates Rab7, which is critical for its association with its GDI and subsequent delivery to endosomal membranes for activation by Mon1a-CcZ1 GEF complex, which in turn is required for late endosome maturation (Fig. 1C).

1.2. PPM1G controls α-Catenin at cellular junctions

PPM1G also known as PP2Cγ is a Mg^{2+}/Mn^{2+} dependent nuclear serine/threonine phosphatase that plays an important role in different functions such as nucleosome assembly, cell survival control, mRNA splicing and DNA damage response. During our mass spectrometry analysis, we found α-Catenin as a

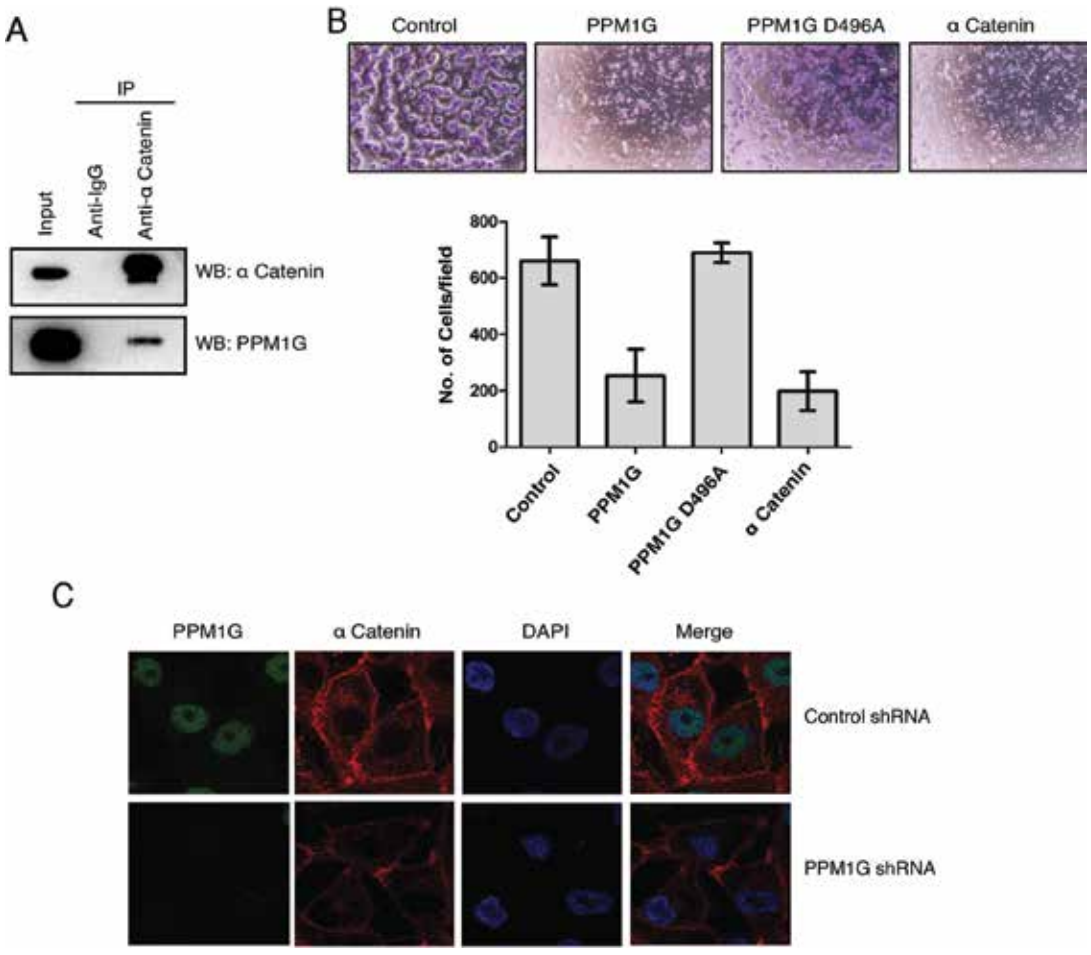

Figure 2. PPM1G controls α-Catenin at cellular junctions (A) Immunoprecipitation of α-Catenin was performed and PPM1G interaction was detected. (B) Effect of PPM1G and α-Catenin on cell migration was assayed by transwell migration assay (C) Localization of α-Catenin in presence and absence of PPM1G was detected by immunofluorescence.

newly associated protein of PPM1G. α-Catenin is a very well studied cell adhesion protein that in concert with β-Catenin and E-Cadherin plays a critical role in linking actin cytoskeletion at the cell junctions. We confirmed the association of PPM1G and α-Catenin in cells (Fig. 2A). In our functional experiments we found that expression of PPM1G, similar to α-Catenin, strongly suppressed the cell migration in invasive breast epithelial cells (MDA-MB-231 cell line) (Fig. 2B). PPM1G is crucial for α-Catenin function at the cell membrane as depletion of PPM1G significantly inhibited the localization of α-Catenin at the cell membrane (Fig. 2C). Our deletion mapping analysis suggested that α-Catenin binds to acidic rich region of PPM1G. The acidic rich region was designated as a substrate-binding domain of PPM1G. Thus, we next tested if α-Catenin is a bonafide substrate of PPM1G. We found that active PPM1G readily dephosphorylates α-Catenin at serine 641 residue. Since α-Catenin plays a critical role in cell junction stability and actin-cytoskeleton dynamics, we are currently hypothesizing that PPM1G interaction with α-Catenin might be important for maintaining proper cell adhesion and preventing cellular migration.

Project 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 project we are interested in studying both the canonical and non-canonical functions of ubiquitination in cells.

2.1. WWP2 ubiquitinates Dvl2 via k63 linkage

WWP2 is an oncogene that we earlier identified as an E3 ligase that degrades its substrates such as PTEN (Maddika *et al.,* Nature Cell Biol. 2011) and p73 (Chaudhary N & Maddika S., Mol Cell Biol 2014) by transferring k48 ubiquitin linkages. In our quest for additional functional cellular substrates of WWP2, we found Dvl2 as its novel interacting protein. Dvl2 is an important player in the transduction of Wnt signaling pathway. We found that WWP2 ubiquitinates Dvl2 but interestingly does not lead to its degradation. By using various ubiquitin K-R mutants, we demonstrated that WWP2 ubiquitinates DVL2 via k63 linkage.

2.2. HACE1 mediated K27 ubiquitin linkage leads to YB-1 protein secretion

While the importance of ubiquitination in controlling the fate and the intracellular functions of various proteins was widely studied, its role in extracellular protein secretion has been unexplored so far. While studying the role of ubiquitination in extracellular protein secretion, we used YB-1 as a model protein and identified the indispensable role of ubiquitination in this process. Importantly, we discovered HACE1 as YB-1 interacting E3 ligase that has the ability to generate functional K27 linked non-canonical ubiquitin linkages on its substrate. K27 ubiquitin linkages on YB-1 are necessary for its interaction with Tumor Susceptibility Gene 101 (TSG101), a component of the Multi Vesicular Body (MVB) pathway, which facilitates its secretion. Intriguingly, the secreted YB-1 unlike intracellular YB-1 displayed a strong EMT suppressor function. In summary, we identified a novel functional role for non-canonical ubiquitin linkages in mediating protein secretion.

Publications

1. Chaudhary N and Maddika S (2014). WWP2-WWP1 ubiquitin ligase complex coordinated by PPM1G maintains the balance between cellular p73 and ΔNp73 levels. ***Molecular and Cellular Biology*** 34: 3754-3764.

2. Jangamreddy JR, Panigrahi S, Lotfi K, Yadav M, Maddika S, Tripathi AK, Sanyal S and Łos MJ (2014). Mapping of apoptin-interaction with BCR-ABL1, and development of apoptin-based targeted therapy. ***Oncotarget*** 5: 7198-7211.

LABORATORY OF CELL SIGNALLING

Investigating the role of inositol pyrophosphates in eukaryotic cell physiology

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	HA Nagarajaram	CDFD, Hyderabad

Objectives

Inositol pyrophosphates are derivatives of inositol that contain pyrophosphate or diphosphate moieties in addition to monophosphates. They include diphosphoinositol pentakisphosphate (PP-IP_5, or IP_7) and bis-diphosphoinositol tetrakisphosphate ($[PP]_2$-IP_4 or IP_8), which participate in diverse biological functions, including DNA recombination, vesicular trafficking, rRNA transcription and osmotic regulation. The beta phosphate group of inositol pyrophosphates can be transferred to pre-phosphorylated serine residues on proteins to form pyrophosphoserine. Pyrophosphorylation occurs on several proteins within the cell, including proteins involved in ribosome biogenesis, vesicular trafficking and glycolysis. 5PP-IP_5 (5-IP_7) is synthesised from inositol hexakisphosphate (IP_6) and ATP by IP_6 kinases. Mammals have three isoforms of IP_6 kinase, IP6K1, IP6K2 and IP6K3, whereas *Saccharomyces cerevisiae* have a single IP_6 kinase, Kcs1.

Our aim is to understand the molecular mechanisms by which various cellular phenomena are regulated by inositol pyrophosphates. 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 inositol pyrophosphate levels are perturbed. In particular, we focus on the following objectives:

1. Understand the molecular details of protein pyrophosphorylation by inositol pyrophosphates;

2. Investigate the cellular functions of mammalian inositol hexakisphosphate kinase 1 (IP6K1); and
3. Study the role of inositol pyrophosphates and IP6 kinases in whole animal physiology.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

We observed that *S. cerevisiae* strains lacking Kcs1 display slow growth, increased sensitivity to translation inhibitors, decreased protein synthesis and reduced ribosome levels compared with wild type yeast. These phenotypes can be reversed upon the expression of enzymatically active Kcs1, but not the inactive form, indicating that these effects can be attributed to the loss of inositol pyrophosphates. The rate of rRNA synthesis, the first step of ribosome biogenesis, is decreased in *kcs1*Δ yeast. We determined that the enzyme responsible for rRNA synthesis, RNA polymerase I (Pol I), is pyrophosphorylated by IP_7 on serine residues falling within mobile regions on the surface of the enzyme. There is no difference in Pol I occupancy on rDNA, but the rate of transcription elongation by Pol I is reduced in *kcs1*Δ yeast. It is possible that IP_7 acts as a metabolic messenger, transducing changes in intracellular ATP to regulate ribosome biogenesis and energy consumption. This work has now been completed and was published in the current reporting year.

To understand the cellular functions of IP_7 in mammals, we use mouse embryonic fibroblasts (MEFs) derived from *Ip6k1* knockout (*Ip6k1*$^{-/-}$) embryos, which have 70% reduced levels of IP_7 compared with wild type (*Ip6k1*$^{+/+}$) MEFs. These cells provide an excellent model to study specific cellular functions of inositol pyrophosphates that may be biochemically linked with protein pyrophosphorylation. In an earlier publication (Jadav *et al.,* J. Biol. Chem. 2013), we described a role for inositol pyrophosphates synthesised by IP6K1 in homologous recombination (HR) mediated repair of DNA double strand breaks in mammalian cells. Subsequent experiments revealed that HR repair is stalled after strand invasion, but prior to the formation of Holliday junctions in cells with reduced IP_7. We are currently attempting to identify the exact molecular targets of IP_7 in the regulation of HR.

To study the role of inositol pyrophosphates in whole animals, we have established a colony of *Ip6k1*$^{+/-}$ heterozygous mice and are breeding them to obtain wild type and knockout litter-mates. Using these mice as a model system, we identified a role for IP6K1 in maintaining *in vivo* haemostasis by influencing platelet polyphosphate levels. Low platelet polyphosphate levels in *Ip6k1*$^{-/-}$ mice lead to lengthened clotting time, altered clot architecture, and protection against pulmonary thromboembolism. This project was completed and published in the previous reporting year (Ghosh *et al.,* Blood, 2013).

We previously reported the preliminary results of our investigation into the cause of male infertility in *Ip6k1* knockout mice. Flow cytometry analysis of germ cells isolated from seminiferous tubules of *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ mice revealed that the loss of IP6K1 does not affect meiosis, and that round spermatids are generated in these mice. However, elongated spermatids that arise from round spermatids by the process of spermiogenesis are reduced in number, have misshapen heads, and absent or bent tails.

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

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

This is a new activity, wherein our goal is to understand how pyrophosphorylation can alter a protein's function. We began by examining the oncoprotein c-Myc (Fig. 1A, B) which contains a PEST domain, a hallmark of short-lived proteins, the deletion of which leads to an increase in the half life of c-Myc. This domain contains acidic serine sequence motifs that may be pyrophosphorylated by IP_7 (Fig. 1B). We overexpressed mouse c-Myc in HEK293T cells, incubated the immunoprecipitated protein with radiolabelled IP_7, and noted that c-Myc is indeed pyrophosphorylated by IP_7 (Fig. 1C). We observed that *Ip6k1*$^{-/-}$ MEFs and mice have higher steady state c-Myc levels compared with their wild type counterparts (Fig. 1D), which we attributed to the greater stability of c-Myc protein in these cells (Fig. 1E, F). c-Myc present in wild type MEFs has a higher level of ubiquitylation compared with c-Myc from *Ip6k1*$^{-/-}$ MEFs (Fig. 1G), suggesting that reduced ubiquitylation triggered degradation is the basis for the longer half life of c-Myc in the absence of IP6K1. We are currently mapping the exact site(s) of pyrophosphorylation on the c-Myc PEST domain to probe the molecular mechanism by which pyrophosphorylation regulates c-Myc ubiquitylation and stability.

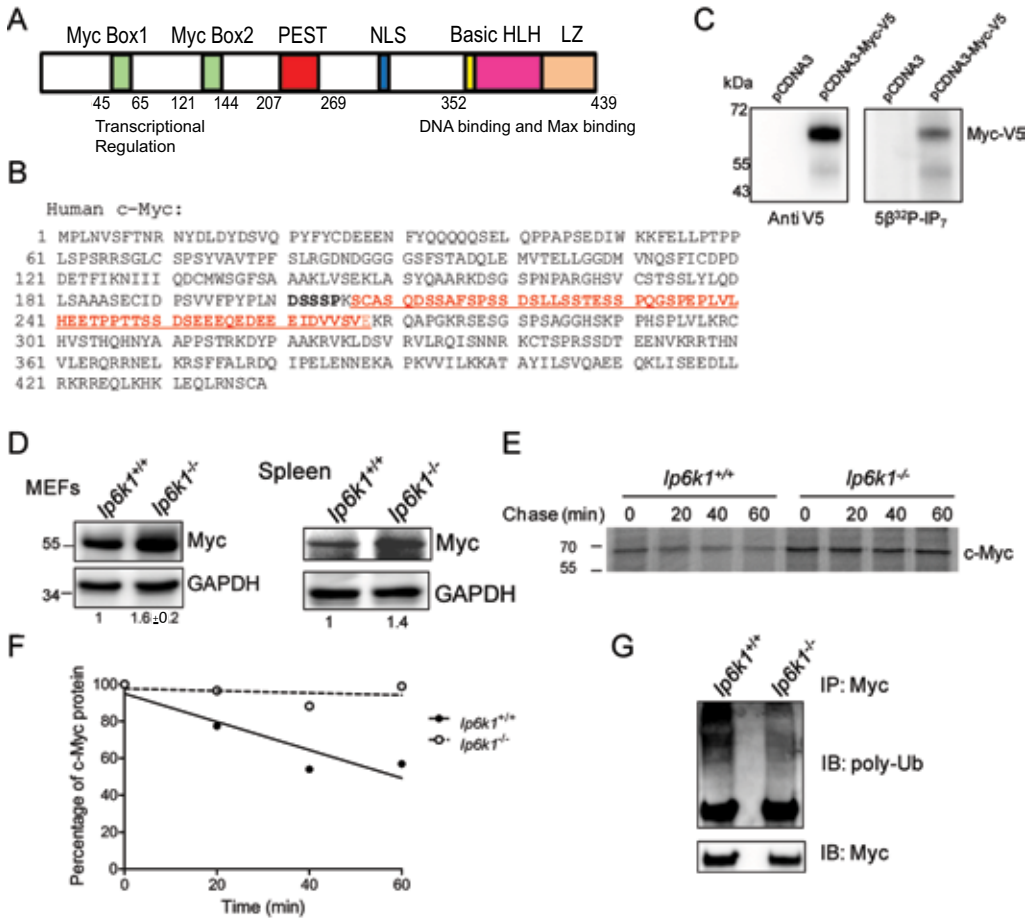

Figure 1. IP6K1 regulates the stability of c-Myc. (A) Schematic representation of the domain architecture of c-Myc. (B) The sequence of human c-Myc highlighting the PEST sequence (underlined) which contains acidic serine sequence motifs that are predicted to be phosphorylated by CK2 and pyrophosphorylated by IP_7. (C) Full length mouse c-Myc was C-terminally tagged with a V5 epitope, expressed in HEK293T cells, immunoprecipitated, and subjected to phosphorylation using radiolabelled IP_7. (D) c-Myc levels in MEFs or spleen from *Ip6k1*$^{+/+}$ or *Ip6k1*$^{-/-}$ mice were detected by western blotting and normalised to the levels of GAPDH in the lysates. The fold change (mean±S.E.M.) in c-Myc levels in the knockout cells/tissue compared with wild type is indicated. (E, F) MEFs of the indicated genotypes were pulse-labelled with an [^{35}S]Cys-Met mixture for 60 min in Cys-Met free DMEM, and chased with excess unlabelled Cys and Met for the indicated time. Endogenous c-Myc was immunoprecipitated, resolved by SDS-PAGE, and transferred to a PVDF membrane. Radiolabelled c-Myc was detected by autoradiography (E), quantified by densitometry analysis, and expressed as a relative percentage of the amount of c-Myc protein at the zero time point (F). Data are representative of two independent experiments. (G) Endogenous c-Myc was immunoprecipitated from *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ MEFs treated with the proteosome inhibitor MG132. Western blotting was conducted with an antibody to detect poly-ubiquitylation of the immunoprecipiated c-Myc, followed by reprobing the blot with a c-Myc antibody

Project 2: Cellular functions of mammalian inositol hexakisphosphate kinase 1 (IP6K1): Role of inositol pyrophosphates in vesicular trafficking.

This is a new activity, which aims to understand the role of inositol pyrophosphates in vesicular trafficking. We monitored the endocytosis and recycling of fluorescently labelled transferrin in *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ MEFs. A short pulse with labelled transferrin followed by a chase with unlabelled transferrin revealed enhanced accumulation and slower recycling of transferrin in *Ip6k1*$^{-/-}$ MEFs (data not shown). After labelling for 1 h, endocytosed transferrin was observed to occupy a perinuclear compartment in *Ip6k1*$^{+/+}$ MEFs whereas *Ip6k1*$^{-/-}$ MEFs exhibited a peripheral staining, suggesting that trafficking to the perinuclear compartment is slower in *Ip6k1*$^{-/-}$ MEFs (Fig. 2A). In addition, the colocalisation of transferrin with the early endosome marker

EEA1 increased in *Ip6k1*$^{-/-}$ MEFs, implying that transferrin is retained in the early endosomes (Fig. 2A, B). Overexpression of a catalytically active form of IP6K1 in the *Ip6k1*$^{-/-}$ MEFs reverted this phenotype, whereas expression of inactive IP6K1 had no effect, indicating the requirement of inositol pyrophosphates for normal transferrin trafficking in a cell. When stained with the cis-Golgi marker GM130, *Ip6k1*$^{-/-}$ MEFs exhibited a fragmented Golgi morphology as opposed to a perinuclear arc-like structure observed in *Ip6k1*$^{+/+}$ MEFs (Fig. 2C, D). Expression of catalytically active but not inactive IP6K1 was able to rescue the Golgi fragmentation phenotype suggesting

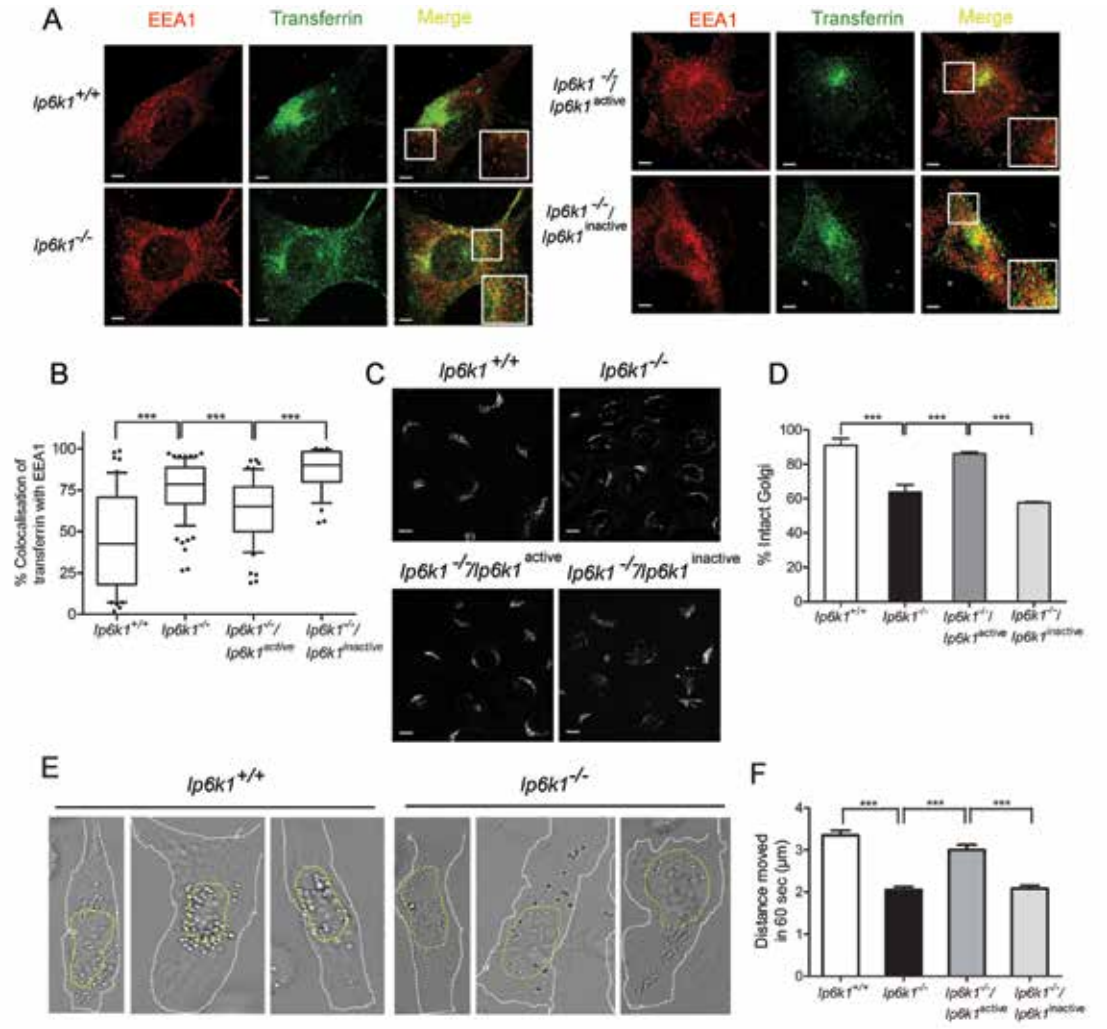

Figure 2. IP6K1 in vesicular trafficking. (A) The indicated cell lines were pulsed labelled with Alexa Fluor 488 labelled transferrin (green) for 1 h and immunostained with an antibody directed against the early endosome marker, EEA1 (red). Scale bars represent 5 μm. (B) Percentage colocalisation of transferrin with EEA1 positive structures in (A). Box and whiskers plot indicate the median, the interquartile range (box) and the 10th and 90th percentile (whiskers). The circles indicate the outliers. n=80 for *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ MEFs and n=40 for *Ip6k1*$^{-/-}$ MEFs overexpressing catalytically active or inactive forms of IP6K1. Data are representative of two independent experiments. (C) MEFs of the indicated genotypes were stained to detect GM130, a cis-Golgi marker. (D) Percentage of cells with intact Golgi morphology in (C) was scored by visual examination in the indicated cell lines. Data (mean±S.E.M. n=150 cells) are representative of three independent experiments. (E) Peritoneal macrophages derived from *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ mice were pulsed with 750 nm latex beads for 1 h and incubated in serum containing medium for an additional hour. DIC images were taken to assess the localisation of beads. (F) Graph showing distance moved by cholera toxin B containing vesicles in the indicated cell lines analysed by Image J. Data (mean±S.E.M. n=100 vesicles) are representative of three independent experiments. P values are from an unpaired two-tailed student's t-test (***, p≤0.001).

that IP_7 is required for Golgi architecture maintenance (Fig. 2C, D).

Golgi fragmentation and decreased transferrin trafficking are characteristic phenotypes of cells with defective function of the motor protein dynein, which enables transport of vesicles from the cell periphery towards the nucleus. To examine dynein-driven vesicle motility, we monitored the trafficking of phagocytosed latex beads in mouse macrophages. 1 h post-internalisation, the beads were observed in the perinuclear region which corresponds to late phagosomes/phagolysosomes in *Ip6k1*$^{+/+}$ macrophages, whereas in the *Ip6k1*$^{-/-}$ macrophages a larger fraction of the beads were still away from the nucleus (Fig. 2E). To monitor vesicle motility in real time, we tracked the movement of cholera toxin B containing vesicles in *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ MEFs. We observe that the rate of vesicle transport is significantly lower in *Ip6k1*$^{-/-}$ compared with *Ip6k1*$^{+/+}$ MEFs, and that this phenomenon is IP_7 dependent (Fig. 2F).

The data presented here reveal a novel role for inositol pyrophosphates in dynein-driven trafficking. To understand the mechanistic link between inositol pyrophosphates and the dynein complex, we will focus on the specific subunit(s) of dynein and its interactors that could be regulated by IP_7-mediated pyrophosphorylation, and test the relevance of this modification in altering dynein function.

Project 3: Physiological role of IP_7 in mice: Regulation of spermatogenesis by IP6K1.

To investigate the cause of infertility in male *Ip6k1*$^{-/-}$ mice, we dissected and compared the testes and epididymides of adult (2 month old) *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ male mice. Testes and epididymides are smaller in *Ip6k1*$^{-/-}$ mice compared to their *Ip6k1*$^{+/+}$ littermates, but do not show any gross morphological defects (Fig. 3A). Immunohistochemical examination of epididymides with MVH (mouse vasa homologue, a marker for round spermatids) revealed mostly MVH-positive round spermatids in the epididymal lumen with only a few degenerating sperm in *Ip6k1*$^{-/-}$ mice compared to *Ip6k1*$^{+/+}$ mice whose epididymides were completely occupied with mature MVH-negative spermatozoa (Fig. 3B). In contrast to the numerous mature spermatozoa found in the epididymides of *Ip6k1*$^{+/+}$ mice, *Ip6k1*$^{-/-}$ epididymides exhibited a dramatic decrease in the sperm count (Fig. 3C). These results clearly demonstrate that male infertility associated with loss of IP6K1 is primarily due to azoospermia, i.e., the absence of mature spermatozoa.

To determine the exact stage at which spermatogenesis is affected in *Ip6k1*$^{-/-}$ mice, we examined the first wave of spermatogenesis by conducting histological examination of postpartum testes of 24, 34 and 60 day old mice (Fig. 3D). At 24 days postpartum (dpp), spermatogonia (Sg), meiotic spermatocytes (pachytene, PS and diplotene, DS) and round spermatids (rS) were observed in the testes of *Ip6k1*$^{+/+}$ mice, but *Ip6k1*$^{-/-}$ testes contained only pachytene and diplotene spermatocytes and no round spermatids, indicating a delay in the completion of meiosis (Fig. 3D, 24dpp). At the end of the first postnatal spermatogenic cycle, which is completed in 34 days, elongated spermatids (eS) were observed in all the seminiferous tubules of *Ip6k1*$^{+/+}$ mice, but *Ip6k1*$^{-/-}$ mice contained only round spermatids as the most advanced postmeiotic germ cells, suggesting a major defect in spermatid differentiation (Fig. 3D, 34dpp). Although 2 month old adult *Ip6k1*$^{+/+}$ testes contained fully mature ready-to-release sperm (Sp), *Ip6k1*$^{-/-}$ testes of the same age exhibited only elongating spermatids and lacked fully condensed sperm (Fig. 3D, 60dpp).

To assess the time course of IP6K1 expression during germ cell development, we examined the expression of IP6K1 by western blotting in *Ip6k1*$^{+/+}$ mice using testes from 14, 18, 28 and 34dpp, and 75 day old adult mice. IP6K1 is detected in 14dpp testes and its expression continues through to 34dpp and adult testes (Fig. 3E). Immunofluorescence staining of 28dpp juvenile testes from *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ mice revealed that *Ip6k1* is expressed in the cytoplasm of round spermatids and pachytene spermatocytes with perinuclear localization (Fig. 3F). IP6K1 shows higher expression in late pachytene and diplotene spermatocytes and round spermatids compared to early pachytene cells (Fig. 3F). In contrast, IP6K1 was absent from spermatogonia, preleptotene and leptotene spermatocytes. These data clearly establish that IP6K1 is expressed only in meiotic and post meiotic germ cells with expression being higher in post meiotic round spermatids. The correlation of spermatogenic defects exhibited by *Ip6k1*$^{-/-}$ testes and the expression pattern of IP6K1 during germ cell development suggests an important role for IP6K1 in mammalian spermatid differentiation. We are currently conducting analyses to determine the exact stage(s) of spermatid differentiation that are dependent on IP6K1.

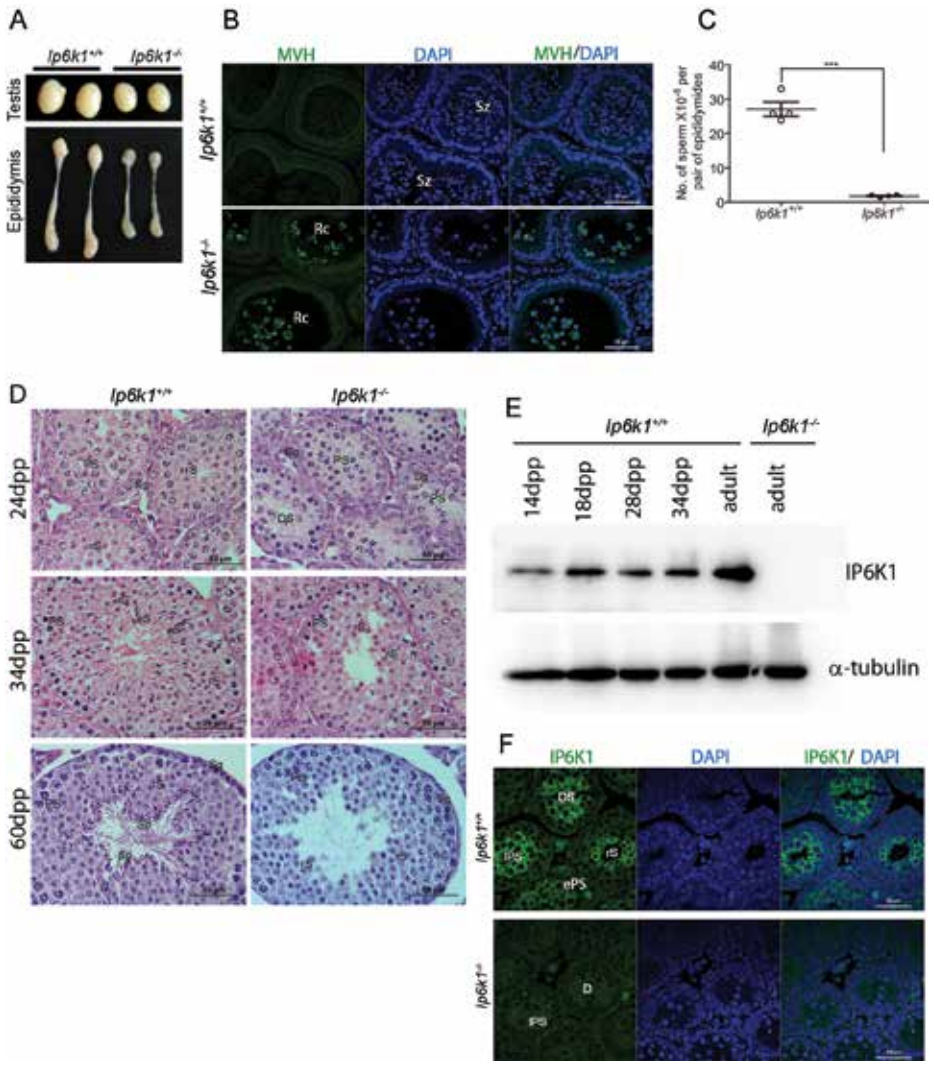

Figure 3. Loss of IP6K1 causes azoospermia in mice. (A) *Ip6k1*$^{-/-}$ mice display smaller testes and epididymides compared to *Ip6k1*$^{+/+}$ mice. (B) Immunofluorescence visualisation of the round spermatid marker mouse homologue of vasa (MVH, green), and nuclei staining with DAPI (blue), in epididymal sections of *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ mice. Cell types marked are mature spermatozoa (Sz) and degenerating round cells (Rc). (C) Cell count of epididymal spermatozoa of *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ mice (age 10 to 12 weeks). Average sperm count per pair of *Ip6k1*$^{+/+}$ epididymides is 27 million, which is reduced to 1 million in *Ip6k1*$^{-/-}$ mice (n = 4). Data points are values for individual mice. The horizontal line is the group mean ± S.E.M. (***P < 0.0001; unpaired Student's t test). (D) Haematoxylin and eosin stained testes cross sections of 24, 34 and 60 days postpartum (dpp) *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ mice. Cell types marked are: spermatogonia (Sg), pachytene spermatocytes (PS), diplotene spermatocytes (DS), round spermatids (rS), elongated spermatids (eS), and fully condensed elongated spermatids ready for release (Sp). (E) IP6K1 expression in juvenile *Ip6k1*$^{+/+}$ and adult *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ testes. Total testis lysates from 14dpp, 18dpp, 28dpp, 34dpp *Ip6k1*$^{+/+}$and adult (75dpp) *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ mice were immunoblotted with an anti-IP6K1 antibody, with alpha tubulin as a loading control. (F) Immunofluorescence localization of IP6K1 in 28dpp testis cross sections of *Ip6k1*$^{+/+}$ and *Ip6k1*$^{-/-}$ mice. Cell types marked are: early pachytene spermatocytes (ePS), late pachytene spermatocytes (lPS), diplotene spermatocytes (DS), and round spermatids (rS). Scale bar = 50μm.

Publications

1. Thota SG, Unnikannan CP, Thampatty SR, Manorama R and Bhandari R (2015). Inositol pyrophosphates regulate RNA polymerase I-mediated rRNA transcription in *Saccharomyces cerevisiae*. ***Biochemical Journal*** 466: 105-114.

LABORATORY OF CHROMATIN BIOLOGY AND EPIGENETICS

Understanding functions of Sirtuin family deacetylases in eukaryotic cell physiology

Faculty	Devyani Haldar	Staff Scientist
PhD Students	Lahari Konada	Senior Research Fellow
	Raghavendra Vadla	Senior Research Fellow
	Amrita Sengupta	Senior Research Fellow
	Shalini Aricthota	Junior Research Fellow
	Mayank Singh Chauhan	Junior Research Fellow (Since Jul. 2014)
Other Members	Nirupama Chatterjee	Technical Officer
	Ananga Ghosh	Project-Junior Research Fellow (Till Jan. 2015)
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	Srinivas Oruganti	DRILS, Hyderabad
	Shekhar Mande	NCCS, Pune

Objectives

1. Understanding the molecular functions of Sirtuin family NAD+ dependent histone/ protein deacetylases;
2. A yeast based screen for discovery of novel Sirtuin inhibitors as anti-cancer agents.

Project 1: Understanding the molecular functions of Sirtuin family NAD+ dependent histone/protein deacetylases.

Reversible acetylation/deacetylation of proteins regulates numerous important cellular processes. The Sirtuin family of protein/histone deacetylases (HDAC) are conserved enzymes that require NAD+ to deacetylate proteins. Sirtuins carry out a broad range of crucial cellular functions ranging from transcriptional silencing to DNA damage response, cell cycle regulation, metabolism and aging etc. We use yeast as well as mammalian cells in culture as models to decipher novel physiological functions of Sirtuins. Yeast genetics is a powerful tool which has been instrumental in discovering many novel genes and characterizing their functions in cellular signalling pathways. Since Sirtuins are conserved from yeast to mammals, we use fission yeast *S.pombe* as model systems to understand and elucidate the molecular functions Sirtuins. 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 of 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 signalling pathways where Hst4 could be functioning.

Project 2: To decipher novel functions of sirtuin family NAD+ dependent histone deacetylase Hst4 of fission yeast.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

We had previously reported that fission yeast *S. Pombe* strains lacking sirtuin hst4+, acetylation of its substrate histone H3 lysine 56 increases and S phase is prolonged.To decipher novel functions of Hst4, a slow growth and DNA damage sensitivity phenotype suppressor screen was carried out. Among the suppressor genes identified by this screen,were a few genes encoding proteins involved in DNA replication. One among these is an accessory factor of DNA polymerase alpha. These genetic interactions indicated that Hst4 may be involved in regulation of DNA replication. To decipher the function of Hst4 in DNA replication, we are studying interaction of Hst4 with suppressor sup1 by determining the mechanisms of suppression.The phenotypes of hst4Δ mutants are mainly attributed to increased H3K56Ac levels.We have observed that the H3K56ac levels remain unchanged on over expression of the suppressor gene indicating that the suppressor does not simply reduce H3K56ac levels by recruiting another deacetylase.We have earlier shown that the phenotypes of the

H3K56R and H3K56Q mutants which mimic constitutive deacetylated and acetylated states respectively are similar to hst4Δ mutants. Sup1 expression could not suppress the phenotypes of these mutants. These results indicate that the H3K56ac is required for phenotype suppression.

The phenotypes of hst4Δ mutants such as slow growth, elongated morphology and sensitivity to DNA damaging agents similar to that of Sup1Δ mutants. To test whether Hst4 and sup1 interact epistatically or exhibit synthetic lethality, the individual hst4Δ mutant and sup1Δ were crossed to generate a double mutant. The double deletion mutants were viable and showed growth rate and MMS sensitivity similar to that of hst4Δ mutants. These results show that sup1 might act in the same pathway downstream of Hst4. Since the suppressor functions in DNA replication, we are planning to investigate potential function of Hst4 in DNA replication.

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

i) S-phase delay of hst4 deletion mutants is partially rescued by over-expression of mcl1:

Hst4 functions in cell cycle progression and DNA damage response by directly deacetylating H3K56.Hst4 deletion mutants show delayed S-phase. The delay in S-phase might be due to elevated and persistent levels of H3K56 acetylation resulting in firing of dormant origins. So, to test whether sup1 is able to recover the S-phase delay of hst4 deletion mutants, strains bearing cdc25-22 mutation which arrests cells at G2 phase of cell cycle were used for synchronization. The wild type cdc25-22 and Cdc25-22:: hst4Δ temperature-sensitive cells transformed with either hst4 or Sup1 were arrested in G2 at 36^0C 4h and then released at 25^0C for 4 hours and samples were collected for every 30 minutes. Cells were monitored for

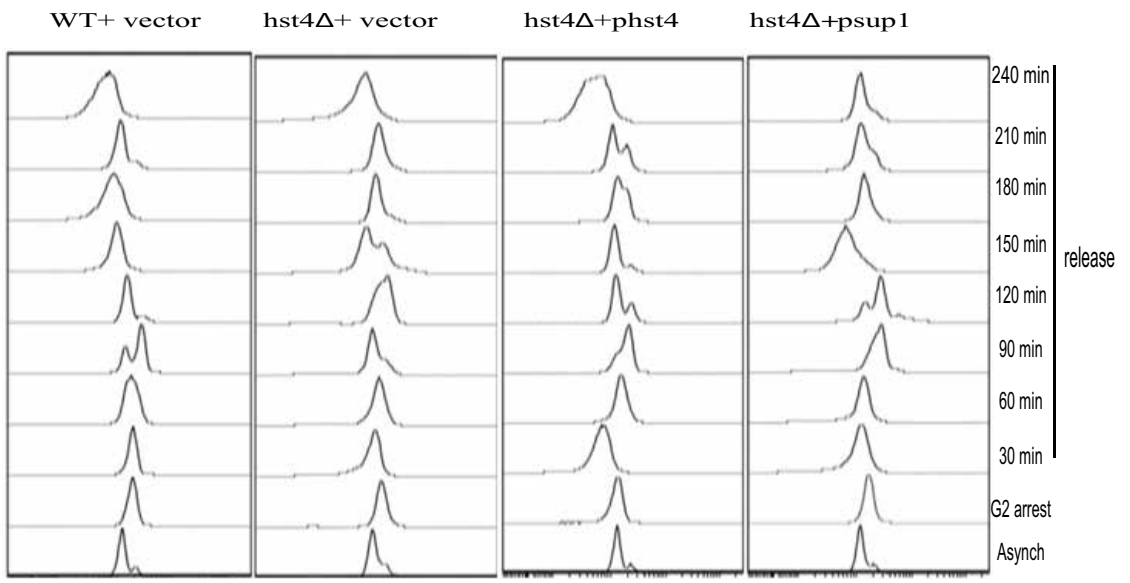

Figure 1. The S-phase delay of hst4Δ mutants recovered by sup1 over expression. Cell-cycle progression analysis of cells synchronized at G2 and released at indicated time points. Wild type and hst4Δ mutant strains *withcdc25-22* mutation and with empty vector and vector expressing hst4 and sup1 genes as indicated were grown at 25°C,then shifted to 36°C for 4 hto synchronize at the G2 phase of cell cycle. To release from G2 they were shifted to 25°C, and cells were collected every 30 min. The flow cytometry profile of asynchronous cells and cells released at indicated times points after G2 arrest is presented above.

progression through the cell cycle using flow cytometry. The FACS data (Fig. 1) indicates that on overexpression of sup1, progression of S-phase is faster than hst4 deletion mutants; however the rate of S-phase progression is slow compared to WT. The results suggest sup1 over expression partially rescues the S-phase delay of hst4 deletion mutants. This partial recovery might be due to hyperacetylated chromatin which may impede replication process.

ii) A yeast based screen for discovery of novel Sirtuin inhibitors asanti-cancer agents:

Epigenetic therapeutics of cancer such as inhibitors of DNAmethyl transferases and histone deacetylases (class I and classII) are already being used in combination with the standard cytotoxics with encouraging results. The Sirtuins (class III NAD-dependent deacetylases) are being considered as important targets for cancer therapeutics as they are up-regulated in many cancers. Inhibition of sirtuins allows re-expression of silenced tumor suppressor genes, leading to reduced growth of cancer cells. However, no sirtuin inhibitors have entered into the clinic yet as an anticancer agent. We would like to identify novel small molecule inhibitors of Sirtuins and characterize their potential as anti-cancer agents using budding yeast, *S. cerevisiae* as model system for compound screening.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

For screening of compounds with Sirtuin inhibitory activity, we have used a yeast *(S. cerevisiae)* strains having the URA3 reporter gene integrated at the silent telomeric locus (Tel::URA3 strain). A reporter silencing assay is based on the ability of yeast Sir2 to keep the URA3 gene silent at telomeric locus and its inhibitor makes it active. The yeast strain which express URA3 will not grow in presence of 5'-fluoroorotic acid (FOA).We have performed the assay and monitored growth of these strains in liquid medium in 96 well plates, without and with FOA. A known Sirtuin inhibitor splitomycin was used as a reference compound. Totally 361 compounds of different chemical classes were explored by following rational drug design and unbiased approaches and subsequently synthesized. These were tested for their activity inhibition using this yeast cell based URA3 reporter silencing assay. Several hit compounds were identified. The identified hit compounds were tested for their ability to inhibit NAD-dependent HDAC activity of recombinant human Sirtuins, hSIRT1 and hSIRT2 *in vitro* using HDAC fluorescent activity assay. One of the potent hit compound, 4bb (ALN-184) was found to inhibit both hSIRT1 and hSIRT2. The effect of treatment of 4bb (ALN-184) on cell proliferation/ viability was determined by MTT assay in several cell lines including HeLa, HEPG2.

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

The effect of treatment of sirtuin inhibitor 4bb (ALN-184) on cell proliferation/viability was determined by MTT assay in more cell lines including HeLa, HEPG2, A549 U2OS, HCT116, hHADF. It was found to be cytotoxic to HEPG2, HeLa and HCT116 cells but not to hHADF cells indicating it is cytotoxic to cancer cells but not dermal fibroblasts. To check whether 4bb treatment causes apoptosis, we have analysed the cell cycle profiles of the treated cells and determined the number of apoptotic sub G1 cells. As indicated in Figure 2A, it did not cause apoptosis in human dermal fibroblasts and killed the HCT116 very efficiently. We next determined whether the apoptosis caused by 4bb was p53 dependent by determining if it activated p53 by increasing its acetylation. It indeed caused acetylation of p53 thereby causing massive cell death in cancer cells. We then checked whether 4bb causes apoptosis by intrinsic pathway by activating caspase 3. The formation of cleaved (active) caspase-3 and PARP cleavage at different time course in the sirtuin inhibitor 4bb treated HCT116 cell line shows that 4bb induced the expression of cleaved caspase-3 at 48 hours, but not at 24 hours after treatment. Cleaved PARP were slightly increased after 24 hours, but expression increases further after 48 hours. Therefore, these data shows sirtuin inhibitor 4bb causes apoptosis by activating caspase 3.

We are currently, checking the molecular mechanism by which 4bb(ALN184) cause apoptosis.Discovery of novel Sirtuins inhibitor would facilitate design and development of novel anti-cancer therapeutics. In addition,deciphering molecular mechanisms involved in eliciting the anti-cancer effect will shed substantial light on the role of Sirtuins in cancer initiation and progression.

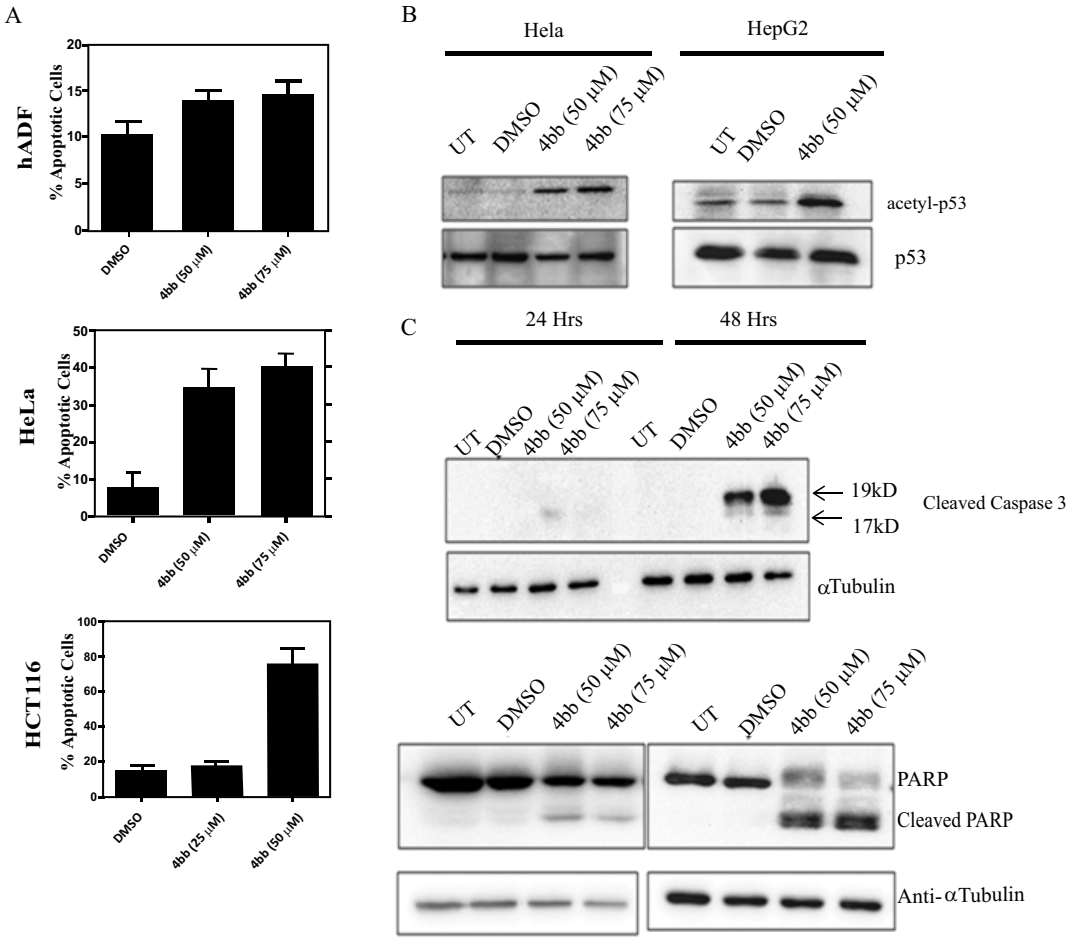

Figure 2. 4bb (ALN-184) is a novel Sirtuin inhibitor that activates p53 and triggers caspase-dependent apoptosis in cancer cells but not in human adult dermal fibroblasts A) Three cell lines (HCT116, HeLa and hHADF) were grown without or with indicated concentrations of 4bb for 48 hours, and flow cytometric analysis of DNA content was carried out by propidium iodide staining and the percentage of sub-G 1cells were calculated and plotted as bar diagram. **B)** HepG2 and HeLa cells were cultured with compound 4bbat indicated concentrations (50 and 75 μM) for 24 and 48 h and level of acetylation of Sirtuin substrate p53 was determined by western blotting, total p53 is shown as a loading control.**C)** HCT116 cells were cultured with compound 4bb indicated concentrations (50 and 75 μM) for 24 and 48 h and the level of caspase3 was determined by western blot, tubulinis shown as a loading control. **D)** HCT116 cells were cultured with compound 4bb indicated concentrations (50 and 75 μM) for 24 and 48 h and the cleavage of PARP at different time points was analyzed by Western blot, tubulin was used as loading control.

Publications

1. Pasha J, Kandagatla B, Sen S, Seerapu GPK, Bujji S, Haldar D, Nanduri S and Oruganti S (2015). Amberlyst-15 catalyzed Povarov reaction of N-arylidene-1H-indazol-6-amines and indoles: a greener approach to the synthesis of exo-1,6,7,7a,12,12a-hexahydroindolo[3,2-c]pyrazolo[3,4-f] quinolines as potential sirtuin inhibitors. ***Tetrahedron Letters*** (In press).

LABORATORY OF COMPUATIONAL BIOLOGY

Computational studies on protein structure, function and interactions

Faculty	HA Nagarajaram	Staff Scientist
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	Manjari K	Senior Research Fellow (Till Sep. 2014)
	HR Rachita	Senior Research Fellow
	Suryanarayana Seera	Senior Research Fellow
	VA Ramesh	Senior Research Fellow
	Rakesh Trivedi	Junior Research Fellow
	Arijita Mitra	Junior Research Fellow (Since Feb. 2015)
Other Members	Rahul S Dhanke	Project-Junior Research Fellow
	Rajkishore Mohapatra	Project-Junior Research Fellow
Collaborators	Srikanth Rapole	NCCS, Pune
	Jochen Schubert	University of Rostock, Germany
	Jose Camara	University of Madeira, Portugal

Objectives

1. Analysis of network properties of human proteins harboring disease causing missense mutations;
2. Structural analysis of instrinsically disordered proteins (IDPs) harboring disease causing mutations;
3. Analysis of Human-Virus PPI (HU-Vir PPI) network; and
4. The New Indigo Project

   a. Multivariate analysis of the volatile compounds (VOCs) detected by the collaborating groups from the breath and urine samples of breast, lung and colon cancer patients and healthy individuals as a means to identify potential cancer biomarkers; and

   b. Development of a database of VOCs detected by collaborators and a webportal hosting the database and other information related to this project

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

1. Our studies on tissue-specific PPI networks revealed that the genes/nodes enriched with splice variants are associated with higher centrality values than the genes with a few/ no splice variants.
2. Our studies on local and global hubs in human tissue-specific PPI networks revealed that:

   a. Local hubs conserve their partners across all the tissues they are expressed whereas global hubs interact with diverse partners in diverse tissues.

   b. The partners of global hubs occupy more diverse sub-cellular localizations than the partners of local hubs.

   c. Both local and global hubs comprise of the hubs that are intramodular (akin to party hubs) and the hubs that are intermodular (akin to date hubs) in nature.
3. We investigated various properties of the human proteins (hVIPs) targeted by viral proteins and found that:

   d. hVIPs are significantly enriched in disordered regions, expressed in more number of tissues and also show high centrality measures (including the new metric introduced by us called pathway centrality) than non-hVIPs.

   e. Localization diversity (LD) of hVIPs is higher as compared with non-hVIPs

suggesting that human partners of viral proteins are wide spread in the cell.

f. hVIPs are evolving at slower rates as compared with non-hVIPs.

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

Project 1: Computational analysis of human and virus protein-protein interaction (Hu-Vir PPI) networks.

1. We identified known domain-motif interactions as well as domain-domain interactions mediating Hu-Vir PPIs. We found that mere 11.6 % of the Hu-Vir PPIs could be identified with known DDIs or DMIs. Viral proteins were found to harbor ELM (eukaryotic linear motif)-like motifs that interact with domains in human proteins.
2. A bipartite network (Fig. 1) of DDIs and DMIs in Hu-Vir PPI revealed that ELMs interact with their unique domain counterparts whereas domains interact with multiple ELMs and domains ($p = 0.01$) (Fig. 2a). There are more DMIs per hu-Vir PPI than DDIs ($p << 10^{-16}$) (Fig. 2b) and also multiple DMIs are used by individual Hu-Vir PPIs when compared with DDIs ($p < 10^{-09}$) (Fig. 2c). It is interesting to note that some of the linear motifs often used by viruses to interact with host proteins are involved in signaling pathways which include kinase functions (Pkinase, SH2, SH3_1 domains), cell cycle regulation (TRAF6, TRAF2, APPCC_Dbox1, WW domains) or protein degradation (MATH domain) or cleavage (Peptidase_S8) pathways. Viruses, therefore, by mimicking human motif interactions interfere in the regulation of the concerned pathways. A DMI, as compared with a DDI, is utilized by more number of viruses ($p < 10^{-09}$) (Fig.2d) and this suggests that a DMI can form a common mode for molecular interactions by multiple viruses whereas DDIs are specific to viruses.

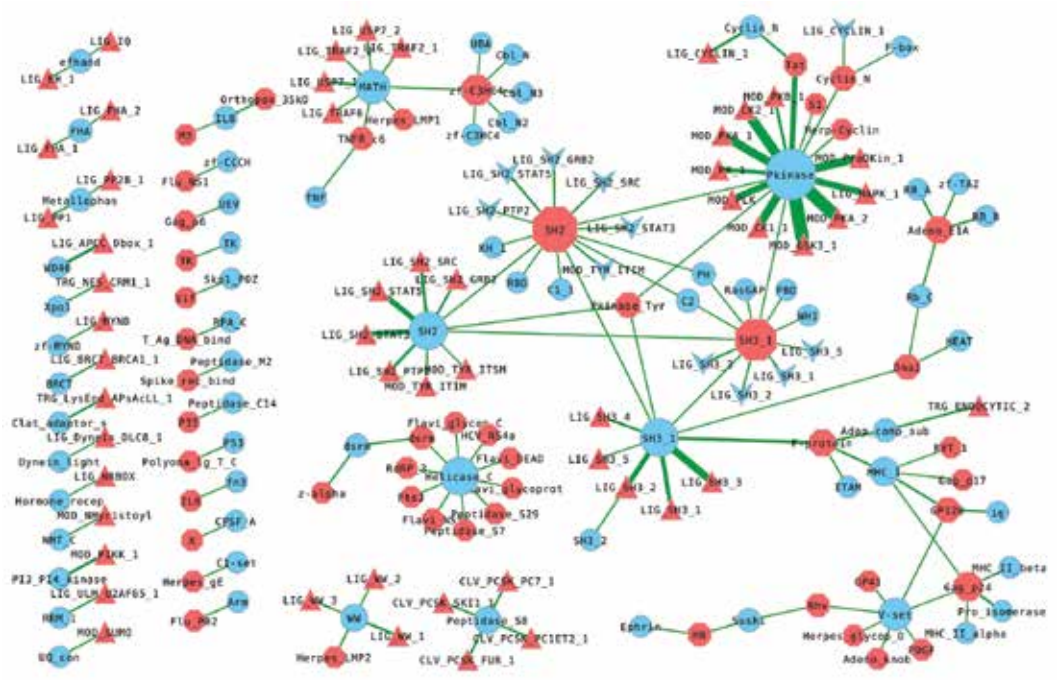

Figure 1. Domain and motif utilization in Hu-Vir PPIs represented by means of Human-virus domain-motif and domain-domain bipartite interaction network. Domains corresponding to human protein are represented by blue circles and motifs by blue vee. Domains corresponding to viral proteins are represented by coral octagon and motifs by coral triangles. Edge thickness corresponds to number of Hu-Vir PPI using particular DMI or DDI and node size corresponds to degree in the current network. Network comprises 54 domain (in hVIP)-motif (in VP) associations from 302 Hu-Vir PPI, 11 motif (hVIP)-domain (VP) associations from 47 Hu-Vir PPI and 99 DDI from 172 Hu-Vir PPIs.

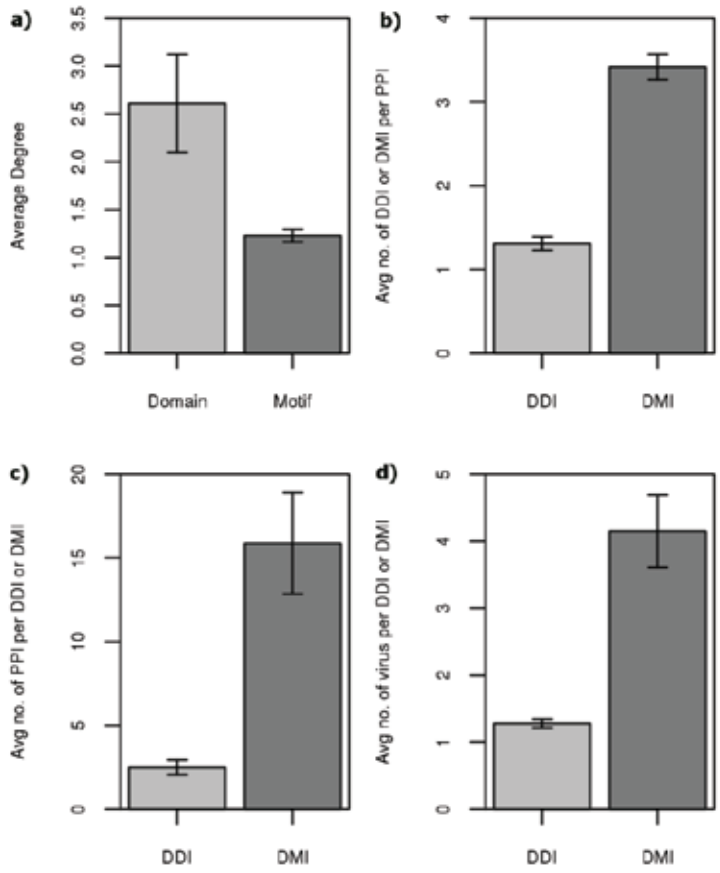

Figure 2. a) Average degree of domains and motifs in bipartite DDI-DMI network. Domains are involved in higher number of interactions than linear motifs ($p = 0.01$). b) Average number of DDI or DMI per Hu-Vir PPI. There are higher number of DMIs per known Hu-Vir PPI than DDIs (p << 10-16). c) Average number of Hu-Vir PPIs using a given DDI /DMI. DMIs are utilized by higher number of Hu-Vir PPIs than DDIs ($p < 10^{-08}$). d) Average number of viruses using a particular DDI/DMI. Multiple viruses same DMI whereas DDIs are virus specific ($p < 10^{-08}$). Error bars represent ± standard error in distribution.

Project 2: Sequence and structural analyses of intrinsically disordered human proteins (IDPs) harboring disease causing missense mutations.

1. In the literature, it is well documented that the pathogenic effects of disease causing mutations at protein structure-function level arise as a consequence of them disrupting the intramolecular interactions that stabilize protein 3D structure. However, not much has been reported on the effects of disease causing mutations on an IDP. Since IDPs are thought to adopt fast interconverting multiple conformations we surmised that the disease causing mutations might destabilize/affect the intramolecular interactions of one or more of these possible conformations thereby affecting the conformational heterogeneity of disordered proteins, which form the very basis of their biological function. In this light, we undertook an in-depth conformational analysis of IDPs harboring disease causing missense mutations.
2. Disordered regions in human proteins were predicted using the program DISPRED3.0 installed on a local server. Any protein with 30% or more of its residues in disordered regions was denoted as IDP. Only those IDPs which form hubs in human PPIN were considered as such proteins are more likely to be associated with large conformational heterogeneity than the other IDPs. The known disease causing missense muations were obtained from HUMSVAR database (www.uniprot.org/docs/humsavar) of which,

416 were found to be mapping on to the disordered regions in IDPs. As a pilot study P53 - one of the IDPs was subjected to long molecular dynamics simulations of more than 200ns and the simulations are currently underway (Fig. 3). We would be analysing the snapshots collected duing simulation to identify various conformational states and analyse how known disease causing mutations affect one or more of the identified conformational states.

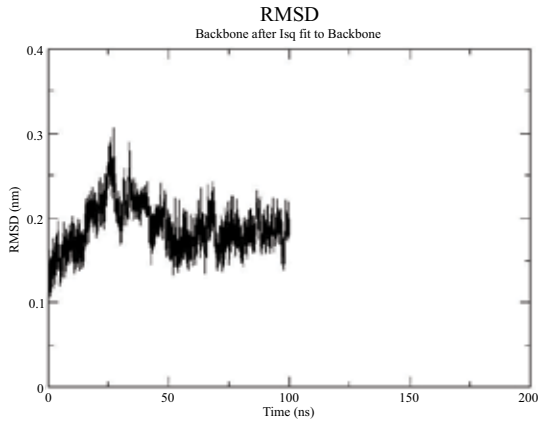

Figure 3. Root Mean Square Deviation (RMSD) trajectory showing the time-evolution of P53 structure. RMSD of an instantaneous P53 structure at a given time is calculated with reference to the starting P53 structure

3. IDPs assume ordered structures when bound to their interacting partners. The kinds of secondary structures they adopt after binding wth their partners depend on their local amino acid sequence and also the intermolecular interactions at the binding interface. We undertook an analysis of known structures of IDPs complexed with their partner proteins. Our preliminary studies show that most of the disordered regions in our sample set adopt helical structures. Further studies are underway to calculate the propensities of residues in disordered regions to assume different secondary structures after forming complexes.

Project 3: Prediction of pathogenic effect of missense mutations: Incorporation of additional features into HANSA

1. We had earlier developed a SVM based method called HANSA (www.cdfd.org.in/HANSA/) that predicts the pathgonic effect (Disease causing or not) of missense mutations in a given protein. This method uses 10 discriminatory features extracted from the human protein and the features include protein position-specific preferences of amino acids, local structural features as well some intrinsic properties of the wild-type and the substituted amino acids. The current version of HANSA had been trained using HUMSVAR dataset of known 12390 disease and 8168 netral missense mutations. Very recently this dataset was revised with more number of missense mutations (22196 disease and 21151 neutral) as compared with the old set. We retrained HANSA using the new dataset and performed 10-fold cross-validation test, which showed that the new HANSA is as good as the old version (AUC = 0.88).
2. We also started investigating the network properties of the human proteins harboring disease causing missense mutations in conjunction with the human proteins harboring begign mutations. Our preliminary analysis reveals that the centrality values of proteins harboring disease mutations are distinct from those of the human proteins harboring neutral mutations (Fig. 4). This observation prompted us to explore further the usefulness of these network parameters of human proteins to train and test our program HANSA as a means to improve its sensitivity and specificity.

Project 4: An attractive and promising strategy for early cancer diagnosis through the assembly of the human cancer volatome (The New Indigo Project).

1. We have developed a webportal such that the front-end directly furnishes introductory details of this project with additional tabs and links for providing information category-wise in detail. The front-end has been designed using web-designing languages PHP, HTML for its general background. Additional effects on the website, including menu-bar, search panel etc Java-scripts, CSS (Cascading Style Sheets) and AJAX (Java Script with XML) have been used. For running of the web-portal in local server (system) Apache Server has been used.
2. The data of VOCs obtained from GC-MS analyses of breath and urine samples is complex because the number of VOCs detected is very large and it usually

outnumbers the number of patients and controls used in such studies. This typically leads to false correlations. We are currently studying some sample datasets sent by our collaborators to test various statistical tools that deal with small sample size with large dimensions. In addition, we are also exploring the methods that estimate missing values, which are often seen in breath and urine VOC data.

Future plans and directions

1. Continuaton of studies on IDPs harboring disease causing mutations.
2. Classification and analysis of disordered regions in proteins.
3. Incorporation of network based features of human proteins in HANSA.
4. Studies on tissue-wise PPI networks integrated with drug-protein interaction data.
5. Standardisation of a protocol for the statistical analysis of the VOCs detected from breath and urine samples collected from cancer patients and healthy individuals toward development of cancer biomarkers. Development of a database for the VOCs detected by the collaborators.

Publications

1. Bashyam MD, Chaudhary AK, Kiran M, Nagarajaram HA, Devi RR, Ranganath P, Dalal A, Bashyam L, Gupta N, Kabra M, Muranjan M, Puri RD, Verma IC, Nampoothiri S and Kadandale JS (2014). Splice, insertion-deletion and nonsense mutations that perturb the phenylalanine hydroxylase transcript cause phenylketonuria in India. ***Journal of Cellular Biochemistry*** 115: 566-574.
2. Bashyam MD, Chaudhary AK, Kiran M, Reddy V, Nagarajaram HA, Dalal A, Bashyam L, Suri D, Gupta A, Gupta N, Kabra M, Puri R, Devi A R R, Kapoor S and Danda S (2014). Molecular analyses of novel ASAH1 mutations causing Farber lipogranulomatosis: analyses of exonic

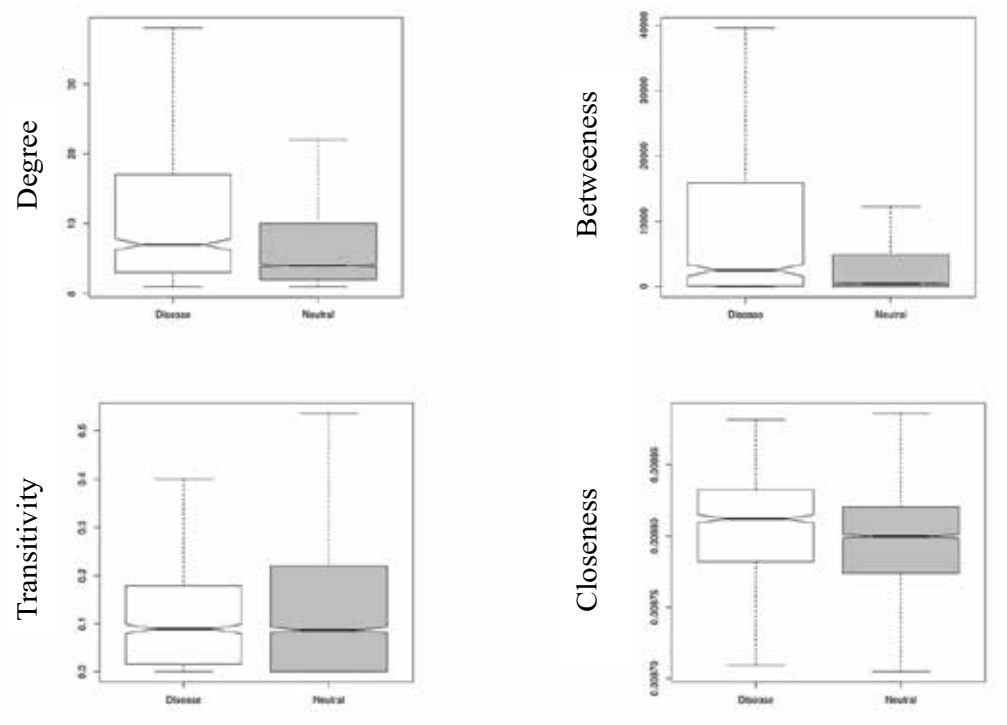

Figure 4. Network Centrality metrics for non-overlapping set of proteins with disease (unshaded box) and neutral mutations (shaded box) from New HUMSAVAR dataset (All p-values are < 4.2e-4).

splicing enhancer inactivating mutation. ***Clinical Genetics*** 86: 530-538.

3. Bidchol A M, Dalal A, Shah H, Suryanarayana S, Nampoothiri S, Kabra M, Gupta N, Danda S, Gowrishankar K, Phadke SR, Kapoor S, Kamate M, Verma IC, Puri RD, Sankar VH, Devi AR, Patil SJ, Ranganath P, Jain SJ, Agarwal M, Singh A, Mishra P, Tamhankar PM, Gopinath PM, Nagarajaram HA, Satyamoorthy K and Girisha KM (2014). GALNS mutations in Indian patients with mucopolysaccharidosis IVA ***American Journal of Medical Genetics*** 164A: 2793–2801.
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5. Patil SJ, Rai GK, Bhat V, Ramesh VA, Nagarajaram HA, Matalia J and Phadke SR (2014). Distal arthrogryposis type 5D with a novel ECEL1 gene mutation ***American Journal of Medical Genetics*** 164A: 2857-2862.
6. Pereira J, Porto-Figueira P, Cavaco C, Taunk K, Rapole S, Dhakne R, Nagarajaram H and Câmara JS (2014). Breath analysis as a potential and non-invasive frontier in disease diagnosis: An overview. ***Metabolites*** 5: 3-55.
7. Rachita HR and Nagarajaram HA (2014). Viral proteins that bridge unconnected proteins and components in the human PPI network ***Molecular BioSystems*** 10: 2448-2458.
8. Sinha A and Nagarajaram HA (2014). Nodes occupying central positions in human tissue specific PPI networks are enriched with many splice variants. ***Proteomics*** 14: 2242-2248.
9. Rachita HR and Nagarajaram H A (2015). Molecular principles of human virus protein-protein interactions ***Bioinformatics*** 31: 1025-1033.
10. Anusha U, Ranganath P, Jamal Md NJS, Krishna Prasad C, Anupam S, Verma IC, Phadke SR, Puri RD, Danda S, Muranjan MN, Jevalikar G, Nagarajaram HA and Dalal AB. Novel mutations of the ARSB gene in Indian patients with Mucopolysaccharidosis Type IV. ***Indian Journal of Medical Research*** (In press).
11. Bidchol AM, Dalal A, Trivedi R, Shukla A, Nampoothiri S, Sankar VH, Danda S, Gupta N, Kabra M, Hebbar SA, Bhat RY, Matta D, Ekbote AV, Puri RD, Phadke SR, Gowrishankar K, Aggarwal S, Ranganath P, Sharda S, Kamate M, Datar CA, Bhat K, Kamath N, Shah H, Krishna S, Gopinath PM, Verma IC, Nagarajaram HA, Satyamoorthy K and Girisha KM. Recurrent and novel GLB1 mutations in India. ***Gene*** (In press).

LABORATORY OF COMPUTATIONAL AND FUNCTIONAL GENOMICS

Computational and functional genomics of biological organisms

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	Ajit Roy	Senior Research Fellow
	Suhail Yousuf	Senior Research Fellow
	Rajendra Kumar Angara	Senior Research Fellow
	Abhishek Kumar	Senior Research Fellow
	Debasish K Ghosh	Junior Research Fellow
	Shailesh Kumar Gupta	Junior Research Fellow (Since Jul. 2014)
Other Members	N Saraswathi	Project-Junior Research Fellow (Till Nov. 2014)
	MJ Vijay Kumar	Project-Junior Research Fellow (Till Oct. 2014)
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Project 1: Studies on the role of Rv2989 (IclR like protein) in the physiology of *M. tuberculosis*

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

In our previous studies, we characterized promoter and binding site of Rv2989 (an IclR like protein) in intergenic region of *leuCD-Rv2989.* In order to understand the physiological functions of Rv2989, we attempted to generate over-expression strain of *M. smegmatis.* However, several attempts to transform *M. smegmatis* with *Hsp60P-Rv2989* failed to produce any viable transformants.

Details of progress made in the current reporting year (April 1, 2014 – March 31, 2015)

Expression from inducible acetamidase promoter *(aceP-Rv2989)* resulted in viable transformants. Induction of protein expression with 0.2% acetamide in growth media leads to *M. smegmatis* growth inhibition (Fig. 1A, 1B). Protein expression levels at different time points of growth stage after induction were monitored through western blot analysis and the growth inhibition coincided with Rv2989 protein expression (Fig. 1C). Further, the *M. smegmatis* aceP-Rv2989 shows motility defect when grown on 0.3% agarose plates containing M63 salts (Fig. 1D). Because the mycobacteria species are known to lack flagella, and motility is because of swarming of growing bacteria, we assume the observed phenotype is because of lack of growth. The *M. smegmatis* aceP-Rv2989 shows rough and dry colony morphology in contrast to control cells, when grown on 7H10 agar plates (Fig. 1E). The rough colony morphology in *M. smegmatis* is a sign of changes in external surface morphology and most probably because of lack of Glycopeptidolipids (GPL) on external membrane. To investigate the morphological changes associated with Rv2989 expression, *M. smegmatis* aceP-Rv2989 strain was visualised under light microscopy at 100X magnification. Most of the cells (~80%) were long approximately 2-5 times the size of control cells (Fig. 1F). The long cells are without any branches or bud like projections. We expect the long cells might be because of defective septum formation resulting in failure of cell division and growth.

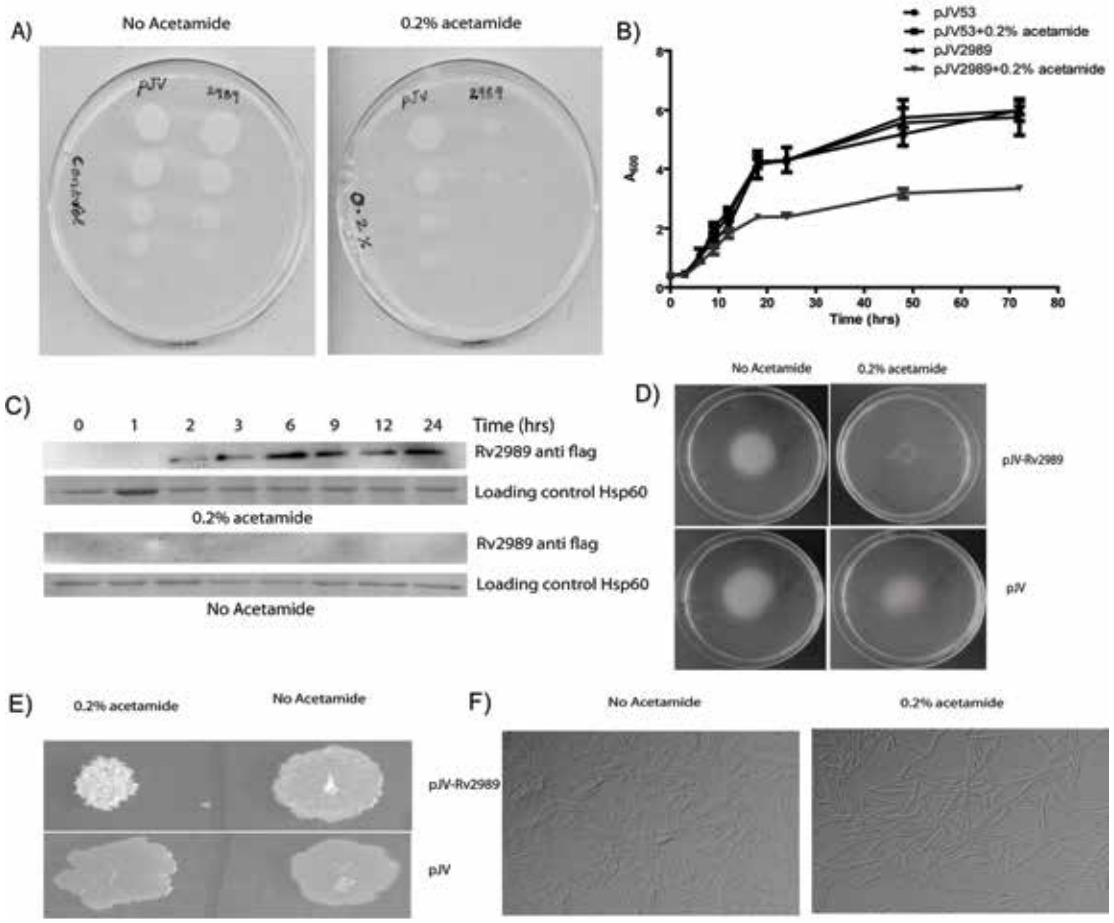


Figure 1. Effect of *M. tuberculosis* Rv2989 over-expression on growth of *M. smegmatis* (A) Effect of *M. tuberculosis* Rv2989 over-expression on growth of *M. smegmatis* on solid media. Ten-fold serial dilutions of cells were spotted on 7H10 agar without or with 0.2% acetamide and incubated for 48-72hrs. (B) Effect of *M. tuberculosis* Rv2989 over-expression on growth of *M. smegmatis* in liquid culture. Growth at different time points was assessed spectrophotometrically. pJV53 is vector control and pJV2989 is 2989 cloned under inducible acetamidase promoter (aceP) The results represent the average and standard deviations from three independent experiments. (C) Analysis of acetamide inducible Rv2989 expression in M. smegmatis. Expression was analysed by western blot analysis of whole cell lysates from *M. smegmatis* at various time points as indicated. Protein was detected using anti flag antibody. (D) *M. smegmatis* aceP-Rv2989 shows reduced sliding motility. Strains were grown on 0.3% agarose plates containing M63 salts. Plates were incubated at 37^{0}C for 3 days. (E) *M. smegmatis* aceP-Rv2989 shows altered colony morphology. The Rv2989 expressing cells show rough colony morphology on 7H10 agar plates. Plates were incubated at 37^{0}C for 3 days. (F) *M. smegmatis* aceP-Rv2989 shows elongated cell morphology. Cells were grown in the absence and presence of 0.2% acetamide for 24 hrs and images captured using bright field microscopy with 100X objective.

Project 2: Characterization and functional studies of FadR like proteins from *M. tuberculosis*.

FadR proteins have been shown to play significant roles in cellular physiology and virulence. *Mycobacterium tuberculosis* genome encodes five proteins belonging to this family and the closest to the well studied *E.coli* FadR is Rv0494.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

Rv0494, one of the FadR like proteins from *M. tuberculosis* showed enhanced expression during nutrient starvation, with expression driven from two independent promoters. The promoter proximal to start codon is sigA dependent and is active during growth under nutrient rich conditions. The second promoter,

sigC dependent, along with sigA promoter was responsible for the increased expression observed during nutrient starvation. Rv0494 was shown to be auto-regulatory with the operator site overlapping with the proximal promoter. Rv0494 was the first FadR family regulator from *M. tuberculosis* shown to be lipid responsive. Long chain fatty acyl CoA molecules with carbon chain length more than 14 carbons were observed to disrupt the protein-DNA interaction. Apart from being auto-regulatory, Rv0494 also repressed the expression of *Rv2326c* operon.

Details of progress made in the current reporting year (April 1, 2014 – March 31, 2015)

Rv0494 is divergently transcribed to a *Rv0493c, a* probable operon. The transcription start site as well the promoter elements of *Rv0493c* operon were determined and were found to be present within the *Rv0494* coding sequence. The operator site of Rv0494 in this organization was acting as road block for *Rv0493c* transcription; however, Rv0494 over-expression in *Mycobacterium smegmatis-pEJ493c* strain had no effect on the promoter activity of pEJ493c. We found that over-expression of Rv0586 significantly repressed the promoter activity of pEJ493c with the site of interaction overlapping with that of Rv0494 (Fig. 2).

Project 3: Identification of novel class of small RNA molecules from *Plasmodium falciparum:* tRNA derived RNA fragments.

tRNA fragments are the novel class of small non-coding RNA molecules derived from tRNA molecules that has been recently discovered and besides microRNA, they have been proposed as the major class of non-coding RNA molecules. Their biogenesis involves RNase Z and Dicer processing machinery and based on their origin and size they have been classified as tRF1, tRF3 and tRF5.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

Previously, we have annotated tRNA modifying enzymes of *P. falciparum* through comparative genomics approach and hypothesized *P. falciparum*

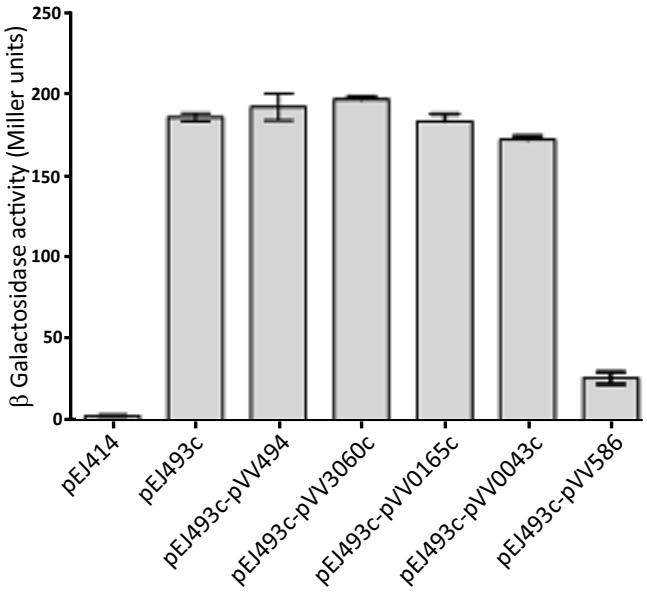


Figure 2. Rv0586 is a transcriptional repressor of Rv0493c operon

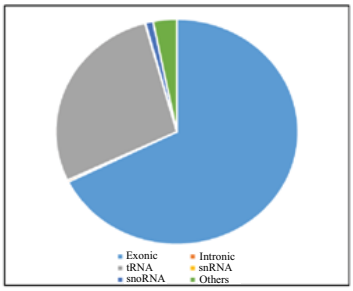
Mycobacterium smegmatis was transformed with pEJ493c to obtain *M. smegmatis: pEJ493c.* The resultant strain was re-transformed with all the FadR paralogs as well as the empty vector (pVV494, pVV586, pVV3060c, pVV0043c, pVV0165c and pVV16). pEJ493c promoter activity was measured in each strain. As can be seen from the bar graph, none of the FadR proteins affected the pEJ493c promoter activity except pVV586 resulting in more than 5-fold decrease in the promoter activity.

apicoplast tRNAguanine transglycosylase as putative target for chemotherapeutic intervention against the parasite. Furthermore, *P. falciparum* adenosine deaminase acting on tRNA (ADAT) was functionally characterized and it was observed to differentially act on different tRNA molecules both ADAT2 and ADAT3 were homogenously expressed from bacterial expression system and it was observed that ADAT3 plays an important role in binding tRNA molecules.

Details of progress made in the current reporting year (April 1, 2014 – March 31, 2015)

The high through-put sequencing of small RNA molecules from asynchronous culture of *P. falciparum* 3D7 strain and their subsequent analysis leads to identification of small RNA molecules that are derived from *P. falciparum* tRNA molecules. The analysis of these populations suggested the presence of tRNA fragments in *P. falciparum* (Fig. 3). Besides the presence of canonical tRFs (tRF5, tRF3 and tRF1), *P. falciparum* consists of two more species of tRNA fragments and based on the site of cleavage, we named them as tRF4, which originate from D loop and extends till the anticodon loop, and tRF2, which consists of sequence between anticodon loop till T loop (Fig. 4A and 4B). Furthermore, tRNA halves of approximately 35 bases in size are abundantly present among the small RNA populations in *P. falciparum* intraerythrocytic stage. Out of total fragments that are derived from the tRNA molecules, 84% belonged to either tRFs or tRNA halves, while remaining 16% mapped to other regions of tRNA molecules.

A

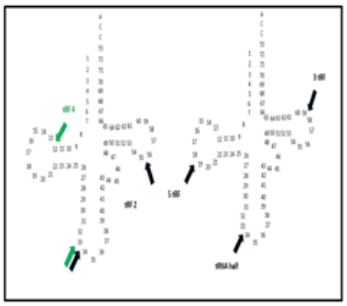


B

Region	Read	Percentage (%)
Exonic	4919059	90
Intronic	21385	0.39
tRNA	202759	3.72
snRNA	5520	0.1
snoRNA	73682	1.35
Others	230074	4.22

Figure 3. Mapping of small RNA molecules (A) Representation of proportion of small RNA molecules identified by high through-put sequencing mapped to different regions of *P. falciparum* transcriptome. **(B)** Tabulated summary of reads that are originated from different region of *P. falciparum* transcriptome.

A



B

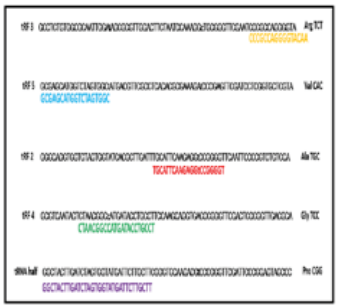


Figure 4. tRNA fragments identified in *P. falciparum (A)* In *P. falciparum,* canonical tRF that originates from 5' and 3' end of tRNA molecules and tRNA halves, that originate from a cleavage event in anticodon loop, were identified. The sequencing data also revealed the abundant presence of two more class of tRFs, tRF4 and tRF2. Arrows denote cleavage sites (black arrow denotes the canonical cleavage site, while green represent new cleavage sites identified in this study). (B) Classification of different tRNA fragments. In *P. falciparum* besides the presence of canonical tRFs, tRF3 (orange), tRF5 (blue) and tRNA half (purple) there exists two more population of tRNA fragments, tRF4 (green) and tRF2 (red) which originates from anticodon loop till T loop and D loop, respectively.

Project 4: Characterization of structural and organizational properties of Huntingtin Interacting Protein K as intracellular aggregation sensor.

Huntingtin Interacting Protein K (HYPK) is ribosome associated protein which modulates intracellular aggregation formation of proteins like poly-glutamine expanded mutant Huntingtin to maintain cellular protesostasis. However, the mechanism of its functional activity towards proteins aggregation recognition and reduction is not understood at molecular level. We addressed the question of how HYPK senses protein aggregation by understanding structural and higher order organizational properties of HYPK.

Details of progress made in the current reporting year (April 1, 2014 – March 31, 2015)

We have characterized HYPK to be capable of existing in a molten globule like state that can drive high oligomerization, finally leading to aggregation like form, both *in vivo* and *in vitro*. Concentration dependent oligomerization of HYPK leads to annular or non-fibrillar amorphous aggregates. Helices in the C-terminal UBA like domain contains patches of hydrophobic regions that can undergo collapse to form small oligomeric seeds. Such seeds initially form the nucleation scaffold upon which addition of other seeds extends the oligomerization in 'Seed nucleation model' to annular complex. A charge rich low complexity region (LCR) spanning between 70-87th residue region participates in electrostatic interactions between seeds to fulfil long range interaction and aggravates formation of non-fibrillar amorphous/ spheroid aggregates. HYPK is structurally bipartite with the N-terminal being unstructured and containing a negative charge rich patch. In cellular condition, this negative charge rich patch loops back to interact with LCR and shield charge(s) of LCR, resulting in prevention of aggregation propagation. Being aggregation prone, HYPK has high affinity towards other aggregation prone proteins like poly-Q expanded mutant Huntingtin, Amyloid beta -42, E46K mutant α-Synuclein but not towards non-aggregating proteins like or ELAV1.

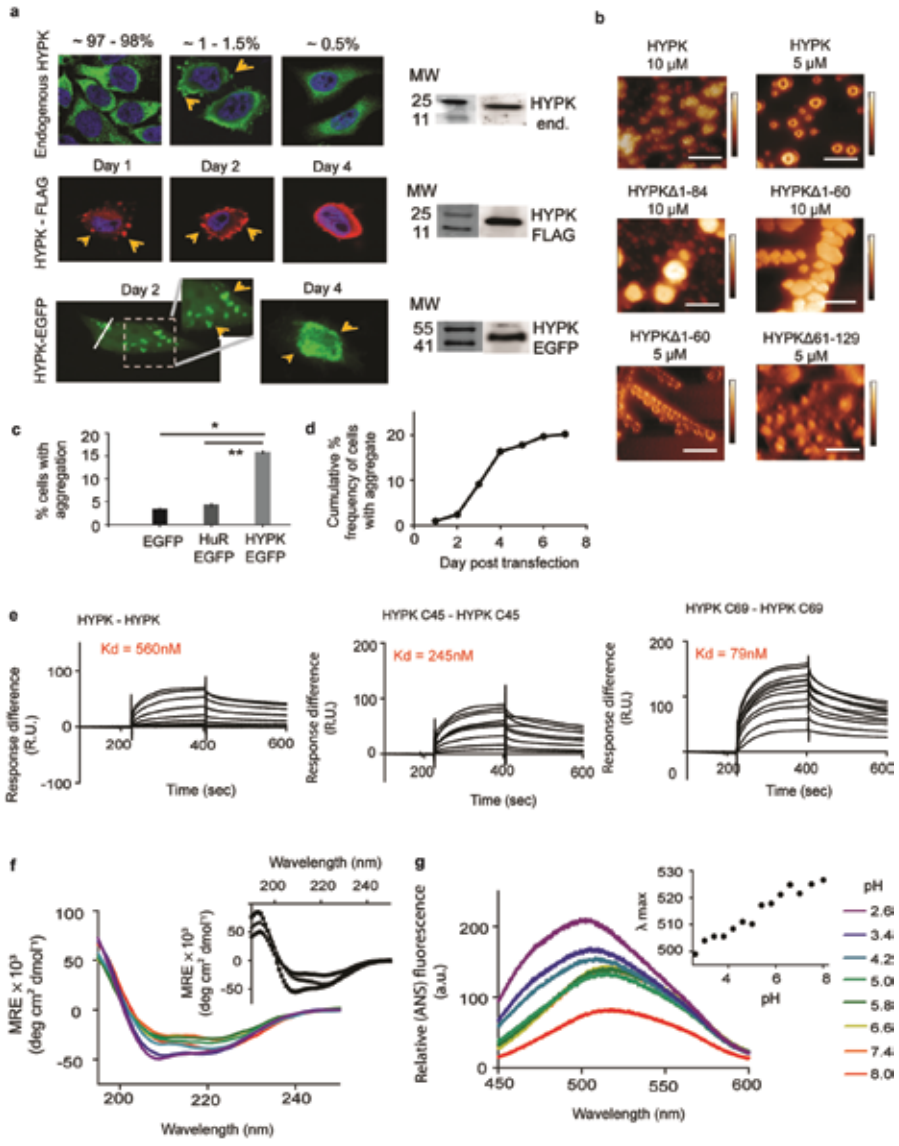


Figure 5. HYPK forms aggregation *in vivo* and *in vitro*. (a) [Upper panel] Endogenous expression of HYPK. Temporal expression of [Middle panel] FLAG tagged HYPK and [Lower panel] EGFP tagged HYPK. [Side panel] Immunoblot of endogenous HYPK, FLAG tagged HYPK and EGFP tagged HYPK. (b) Atomic force microscopic images of aggregates of HYPK, C-terminal 45 residue region, C-terminal 69 residue region and N-terminal 60 residue region at various concentrations. (c) Intracellular aggregate formation of HYPK compared to control EGFP and HuR-EGFP. (d) Temporal induction of intracellular HYPK aggregates. (e) Self association kinetics of HYPK, C-terminal 45 residue region, C-terminal 69 residue region. (f) CD spectrum of HYPK at varying pH. [Inset] CCA deconvoluted three component spectra of pH dependent CD spectral set. (g) HYPK bound ANS fluorescence intensity as function of increasing pH. [Inset] Red shift of emission wavelength maxima with increasing pH indicating burial of hydrophobic surface at higher pH.

Publications

1. Pandey SD, Choudhury M, Yousuf S, Wheeler PR, Gordon SV, Ranjan A and Sritharan M (2014). Iron-regulated protein HupB of Mycobacterium tuberculosis positively regulates siderophore biosynthesis and is essential for growth in macrophages. ***Journal of Bacteriology*** 196: 1853-1865.

2. Yousuf S, Angara R, Vindal V and Ranjan A (2015). Rv0494 is a starvation-inducible, auto-regulatory FadR like regulator from Mycobacterium tuberculosis. ***Microbiology*** 161: 463-476.

LABORATORY OF DROSOPHILA NEURAL DEVELOPMENT

Understanding patterning and development of Central Nervous System using Drosophila melanogaster

Faculty	Rohit Joshi	Staff Scientist & WT-DBT India Alliance Intermediate Fellow
PhD Students	Risha Khandelwal	Senior Research Fellow
	Neha Ghosh	Senior Research Fellow
	Raviranjan Kumar	Senior Research Fellow
	Rashmi Sipani	Senior Research Fellow
	Asif Ahmad Bakshi	Junior Research Fellow (Since Feb. 2015)
Other Members	P Kalyani	Technical Officer
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Objectives

The key objective of the lab is to understand how neural progenitor cells attain their positional identity in developing Central Nervous System (CNS) of an organism and how does this translate into generation of a variety of cell types found in CNS (as represented in the Fig. 1). Hox family of transcription factors are known to play an important role in giving the positional identity to the cells and generation of a variety of cell types along the AP axis of the CNS during development. The molecular basis of this phenomenon is not well investigated. We are interested in understanding the molecular basis of Hox function in patterning CNS using *Drosophila melanogaster* as our model organism, focusing mainly on early embryonic and late 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.

Abdominal region of the *Drosophila larval* CNS has a less number of neurons compared to its thoracic counterpart. Hox gene *Abd-A* in 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. 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. Moreover 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 pNbs and “off” in the neuronal progeny of pNbs.

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

Hox genes express in CNS (in neural progenitor cells) in embryonic stages of development (as represented in Fig. 1) but how does their expression patterns the embryonic nervous system is not well understood. *Deformed* (*Dfd*) is known to express in the cells of subesophageal ganglion of embryonic CNS, this project focuses on understanding auto-regulation of *Dfd* in this region and find out how this helps in giving cells their specific positional identity. This is being done by using a 3.2kb auto-regulatory CNS specific enhancer for *Dfd* which recapitulates the expression of *Dfd* gene in developing embryonic CNS. A smaller region of 630bp of NAE has also been reported to recapitulate the expression of the entire 3.2kb enhancer and this region is also being analysed.

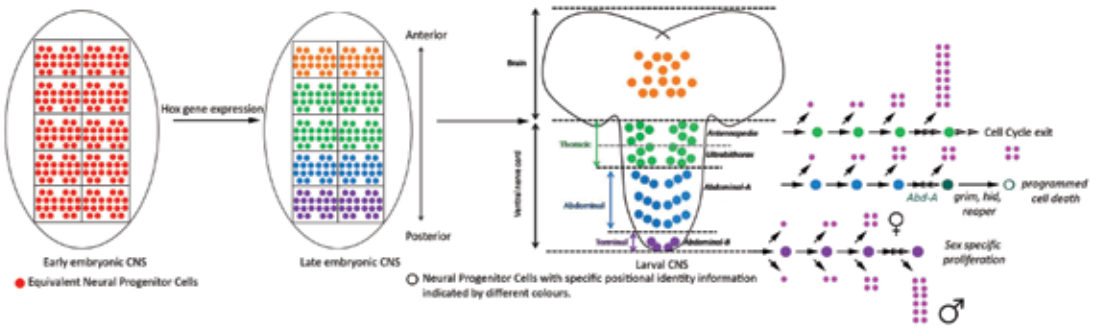

Figure 1. Precursor cells for embryonic Nbs start out as equivalent cells and attain their specific positional identity by Hox gene expression. This gets reflected as specific Nbs identity and thereby determine proliferation and differentiation profile of these cells along the AP axis. In larval stages thoracic, abdominal and terminal post-embryonic Nbs (pNbs) differ in their number and proliferation profile as shown. Thoracic pNbs stop proliferation by cell cycle exit, while abdominal pNbs (in both sexes) and terminal pNbs (tNbs; in females) die as a result of apoptosis, the tNbs in males continue dividing and give rise to more neurons as shown.

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

A set of pNbs in the terminal region of CNS show sex specific proliferation and survival. Although the role of the sex determining hierarchy and Hox gene Abd-B, in growth and differentiation of *Drosophila* genital discs, is well worked out, little is known about how sex determination hierarchy and Abd-B intersects with cell proliferation and survival behavior of terminal Nbs (tNbs) in the larval VNC. Double-sex (Dsx) is the most downstream transcription factor of the sex-specification hierarchy. I intend to test the interaction between Abd-B and Dsx in gender specific proliferation of these cells.

There are 12 pNbs in this region of CNS of which 8 stop dividing in both males and females at mid L3 stage of development. The remaining 4 Nbs which we refer to as tNbs have been known divide differentially in males and females. The hypothesis for this part of work is that Abd-B and Dsx (Double-Sex being the most downstream member of sex specification hierarchy) play a role in sex specific proliferation of these tNbs.

The pNb lineage in terminal region have been characterized and it is known that female specific isoform of Dsx (DsxF) is responsible for the apoptosis of sex-specific tNbs in females. But the molecular mechanism behind the phenomenon of apoptosis of sex-specific tNBs in females, role of Dsx in tNB proliferation and how sex specific tNbs are different from other 8 Nbs is not known

Summary of work done until the beginning of this reporting year (upto 31 March, 2014)

1. Understanding the molecular function of Hox gene *Abd-A* in larval CNS patterning.

It is known that *grim* gene play primary role in this apoptosis and relevant enhancer for the apoptotic genes in Nbs lies in 23kb genomic region referred to as *NBRR-Neuroblast Regulatory Region*. A systematic screening of the 23kb NBRR has been done to identify the enhancer responsible for pNb apoptosis. The 23kb region was divided into 5 overlapping genomic fragments (of 6-10kb) which were screened for their ability to drive pNb specific expression of lacZ reporter in late third instar larval (LL3) brain. These 5 fragments have been amplified by PCR using region specific primers from genomic DNA and all of them have been cloned into *pCasPer-lacZ* shuttle vectors to make transgenic lines. The transgenic line for 3 of the fragments have been analyzed which has helped us to narrow down the search to an overlapping region of two 8kb fragments. A genetic deletion was made by transposon mobilization, this deletion in trans-heterozygotic condition with a bigger deletion blocked pNb apoptosis in abdominal region, suggesting that relevant enhancer is within this smaller deletion generated by us. Simultaneously a 4kb enhancer of *grainyhead* which is responsible for its expression in CNS was sub-fragmented and narrowed down the relevant enhancer for the

expression of grainyhead in CNS to 1kb region. Currently this region is being further analysed to identify transcriptional factors that could be regulating *grainyhead* differentially in Nbs versus neurons.

2. Role of Hox gene *Deformed (Dfd)* in patterning of embryonic subesophageal ganglia

The 608bp *Dfd* autoregulatory element was scanned for Hox-Exd binding sites and was two putative compound binding sites were identified for these two transcription factors. In vitro binding studies were done on these binding sites using EMSA and both of the binding sites showed binding to *Dfd*-Exd hetrodimer. In order to investigate the in vivo relevance of these binding sites, these sites were mutagenized in 608bp DNA element and these various mutagenized forms of the enhancers have been sub-cloned into the *pCasPer-nls-lacZ* shuttle vector and the transgenic lines are being made for the same. These transgenic lines will be tested for their capacity to activate the reporter β-galactosidase to test the relevance of the binding site and direct role for these transcription factors in auto-regulation of *Dfd* gene.

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

We find that Abd-B and Dsx express in tNbs in CNS. Since Grh is already known to play a role in pNb apoptosis. We tested and found that Grh was expressed in tNbs of male larvae at mid L3 stage.

Cyclin E which promotes G1-S transition in dividing cells is being investigated to identify the mechanism behind continued sex specific proliferation of tNbs in male larval CNS. A 1.9kb enhancer element of *cycE* expresses in Nbs, and has binding site for Hox gene *Abd-A* and Abd-B and potential Dsx binding sites. A BrDU, lacZ and Dpn staining of *cycE-1.9kb-lacZ* transgenic flies show that lacZ line marks dividing Nbs in terminal regions of CNS.

Details of progress made in the current reporting year (April 1, 2014 – March 31, 2015)

1. Understanding the molecular basis of Hox gene Abdominal-A (*Abd-A*) in larval CNS patterning.

We had narrowed down the relevant enhancer to 3kb overlapping region of two 8kb fragments after analysis of all 5 *enhancer-lacZ* lines of NBRR. We generated a smaller 2 kb enhancer-lacZ from this overlapping region and found that it is expressed in pNbs of abdominal and terminal region of larval central nervous system.

Genetic isolation of apoptotic enhancer

We had genetically isolated the apoptotic enhancer by mobilizing a transposon inserted in *NBRR* to generate a smaller deletion *(NBRR-22)*. This deletion in transheterozygotic combination with already existing deletion of NBRR gives ectopic pNbs in the abdominal region of CNS at LL3 stage. The PCR mapping indicates that 14.5kb region of the NBRR encompassing the relevant apoptotic enhancer has been deleted in this case.

The expression of 2kb enhancer in abdominal pNb and presence of ectopic pNbs in 14.5 kb deletion suggests that we have narrowed down the relevant apoptotic enhancer from 23kb NBRR to 2kb region of the genome. We are currently working to test Grh and *Abd-A* binding sites in vitro and to test hypothesis of transcriptional activation of Grim by *Abd-A* and Grh.

2. Role of Hox gene *Deformed* in patterning of embryonic subesophageal ganglia.

The costaining of *Dfd* and cell types specific markers like Dpn (a neural progenitor specific marker) Elav (neuron specific marker) and Repo (glial cell specific marker), established that *Dfd* is expressed in neural progenitor cells (neuroblasts-Nbs), neurons and glial cells. Subsequently using the *NAE3.2-lacZ* transgenic line, it was established that expression of *Dfd* is auto regulated in Nbs and neurons since Dpn positive cells in were LacZ positive as well. Subsequently we find that the autoregulation is differentially dependent on Hox cofactor Homothorax in a region specific manner. We find that Homothorax is essential for autoregulation in neural stem cells of maxillary region but not in case in mandibular region of *Dfd* expression. Lastly we find that Homothorax homeodomain is not necessary for *Deformed* neural autoregulation.

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

Grh is known to play a role in pNb apoptosis. We tested and found that Grh was expressed in tNbs of male larvae at mid L3 stage. Subsequently we looked at *grh* mutants and found surviving Nbs in the terminal region of female larval CNS. We

next tested mutant for apoptotic gene *grim* and found surviving ectopic Nbs in the terminal region of female larval CNS. In order to test if the enhancer responsible for the expression of *grim* in dying tNbs lies within NBRR region, we tested the *enhancer-lacZ* lines for their expression in tNbs of male larval central nervous system. We found *enhancer-lacZ* lines expressed in tNbs of male larval CNS. The genetic deletion which deletes NBRR showed ectopic Nbs in terminal region of female larval CNS. These ectopic Nbs in the terminal region of female larval CNS needs to be tested for expression of Dsx gene to conclusively establish that female tNbs are undergoing *grim* mediated apoptosis. We are in process of generating anti-Dsx antibody to carry out these experiment.

LABORATORY OF FUNGAL PATHOGENESIS

Understanding the pathobiology of an opportunistic human fungal pathogen *Candida glabrata*

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	Vivek Kumar Srivastava	Senior Research Fellow
	Vandana	Senior Research Fellow
	Mubashshir Rasheed	Senior Research Fellow
	Priyanka Bhakt	Junior Research Fellow
	Kundan Kumar	Junior Research Fellow (Since Jul. 2014)
Other Members	Suneetha KJ	Technical Officer
	Gaurav Bairwa	Project-Senior Research Fellow (Till Apl.2014)
	Sapan Borah	Project-Senior Research Fellow (Since Jul. 2014)
	Gujjula Rahul	Project-Junior Research Fellow
	Shivarathri Raju	Project Assistant (Till Apl. 2014)
Collaborators	Rajendra Prasad	JNU, New Delhi
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Candida spp. are the leading cause of disseminated fungal infections and rank fourth among the most common nosocomial pathogens. Among *Candida* species, prevalence of *Candida glabrata* is on the rise, and it accounts for up to 30% of total *Candida* blood stream infections. *C. glabrata* is a common resident of the healthy human microflora but is capable of causing life-threatening, systemic infections in the immunocompromised host. It is an asexual haploid budding yeast and exists in the blastoconidial form in both commensal and pathogenic states. In our laboratory, we study multiple virulence aspects of *C. glabrata* with particular emphasis on antifungal drug resistance mechanisms and interaction with host immune cells.

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

Objectives

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

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

Using an *in vitro* system comprised of human monocytic cell line THP-1, we demonstrated that wild-type *C. glabrata* cells are able to impede phagolysosome acidification, counteract/

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. These genes were implicated in diverse biological processes including chromatin and cell wall organization, signal transduction and Golgi vesicle transport. In addition, we showed that CgVps15 and CgVps34 constitute regulatory and catalytic subunit of the class III phosphoinositide 3-kinase (PI3K), and are essential for intracellular survival and virulence in *C. glabrata.*

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

As PI3K in *Saccharomyces cerevisiae* is implicated in regulation of both anterograde and retrograde vesicular trafficking pathways, we examined the role of CgVps15 and CgVps34 in protein trafficking in the current reporting period. For this, we first checked sorting of the vacuolar lumenal hydrolase carboxypeptidase Y (CPY; encoded by the *CgPRC1 gene*) to the vacuole in *Cgvps15*Δ and *Cgvps34*Δ mutants. Any defect in cellular protein sorting pathways is known to result in mislocalization and altered processing of CPY in *S. cerevisiae.* Further, the guanine nucleotide-dependent interaction of Vps15 and Vps34 with Gpa1 (GTP-binding α-subunit of the heterotrimeric G protein) regulates G-protein-coupled receptor (GPCR) pheromone signaling at endosome in *S. cerevisiae.* Hence, we used the mutant lacking CgGpa1 to study the effects of GPCR pheromone signaling on vesicular trafficking in *C. glabrata*. As shown in Figure 1A, compared to *wt* (wild-type) cells, 5-fold higher secretion of CPY was observed in *Cgvps15*Δ and *Cgvps34*Δ mutants. Of note, enhanced secretion of CPY was abrogated in the Cgvps-reconstituted strains (Fig. 1A). The *Cggpa1*Δ mutant did not secrete any appreciable amount of CPY to the extracellular media (Fig. 1A) indicating that PI3K signaling but not GPCR signaling is required for trafficking of CPY enzyme to the vacuole in *C. glabrata*.

To examine if mislocalization of CPY in *Cgvps15*Δ and *Cgvps34*Δ mutants is due to CPY overproduction, we checked CPY levels in mutants via western analysis and observed about two-fold lower amounts of CPY in *Cgvps15*Δ and *Cgvps34*Δ mutants (Fig.1B). However, this CPY form, compared to *wt* cells (~ 59 kDa band), was of slightly higher molecular size (~ 62 kDa band) (Fig. 1B). Further, qPCR analysis revealed ~ 3-fold elevated transcription of the *CgPRC1* gene in *Cgvps15*Δ and *Cgvps34*Δ mutants (Fig. 1C) suggesting that CPY mislocalization could be a combined result of increased production, defective processing and mistrafficking of the CPY enzyme. Notably, no significant change in levels of the cellular CPY was observed in *Cggpa1*Δ cells (Fig. 1B). Together, these data indicate that lack of CgVps15 and CgVps34 results in impaired processing and missorting of CPY in *C. glabrata*.

Next, to examine the trafficking of a GPI (glycosylphosphatidylinositol)-linked cell wall adhesin to the cell surface in *Cgvps15*Δ and *Cgvps34*Δ mutants, we measured expression of the Epa1 (Epithelial adhesin 1) at the cell wall by FACS (Fluorescence-activated cell sorting) analysis using anti-Epa1 antibody. We observed 2- to 6-fold higher Epa1 cell surface expression in *Cgvps15*Δ and *Cgvps34*Δ mutants (Fig. 1D). Notably, Epa1 is trafficked to the cell surface presumably *via* the classical secretory pathway (ER-Golgi-plasma membrane) and required for *in vitro* adherence. To check if Epa1 at the cell wall is functionally active in *Cgvps* mutants, we measured adherence of the *C. glabrata* cells to the Lec2 Chinese hamster ovary epithelial cells. The *epa1*Δ mutant was used as a control for adherence assays. As shown in Figure 1E, compared to ~ 30 % of *wt* cells, ~ 15% of *epa1*Δ cells adhered to Lec2 cells. In contrast, *Cgvps15*Δ and *Cgvps34*Δ mutants exhibited ~2.5-fold higher adherence to Lec2 cells compared to *wt* cells which was restored to the *wt*-adherence level upon ectopic expression of *CgVPS15* and *CgVPS34* gene, respectively (Fig. 1E). As expected, the *Cggpa1*Δ mutant displayed adherence to Lec2 cells similar to that of *wt* cells (Fig. 1E). Notably, GPI-linked aspartyl proteases are known to proteolytically cleave Epa1 from the cell surface and release into the medium. The higher Epa1 levels at the cell surface in *Cgvps15*Δ and *Cgvps34*Δ mutants could be either due to reduced processing or defective retrograde trafficking from the cell wall. Experiments are currently underway to test these hypotheses.

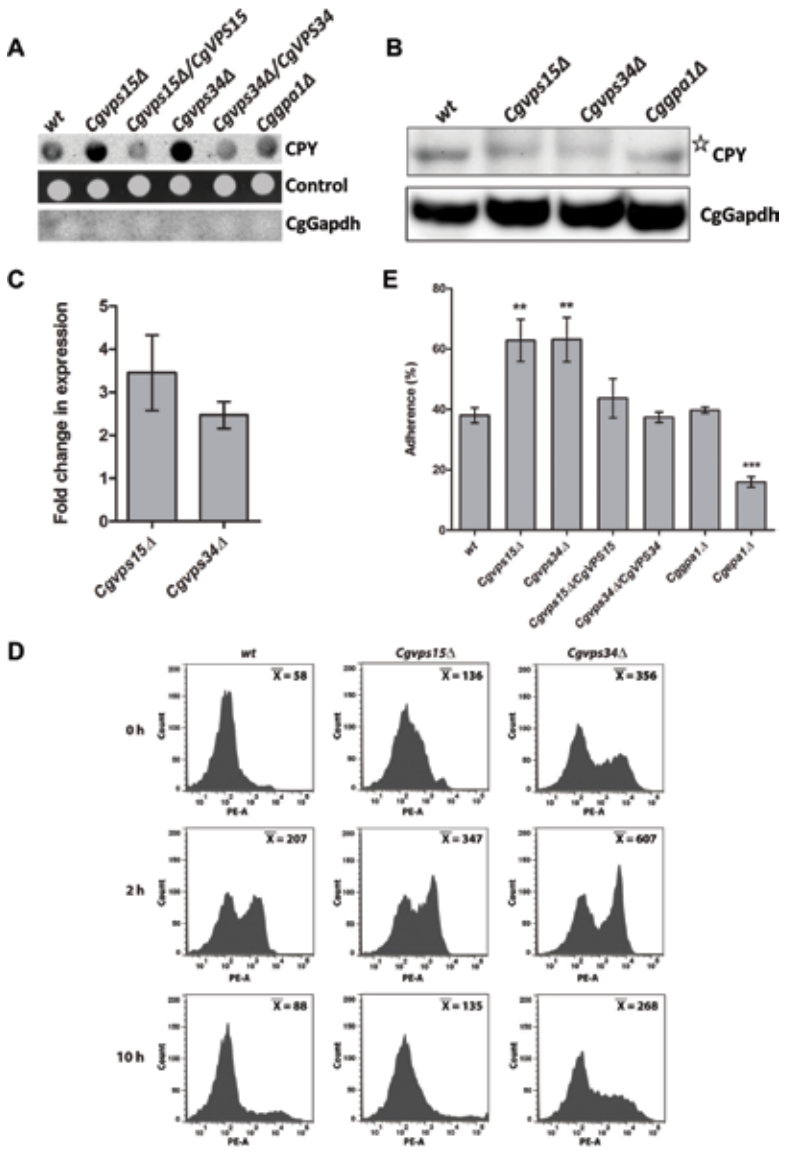

Figure 1. PI3K is required for trafficking of the vacuolar lumenal hydrolase carboxypeptidase Y (CPY) and the major adhesin Epa1 to the vacuole and the cell wall, respectively (A) Representative colony blot analysis illustrating CPY secretion in indicated *C. glabrata* strains. Control lane depicts spotting and growth of equal numbers of yeast cells for each strain. Immunoblot analysis with anti-Gapdh antibody was used as a control for cell lysis. **(B)** Representative immunoblot analysis of CPY levels. *wt* and indicated mutant strains were collected after 4 h growth in the YPD medium (OD_{600}= 0.4-0.6), and proteins were extracted and quantified. 30 μg protein samples were resolved on a 12% SDS-PAGE gel and immunoblotted with anti-CPY and anti-Gapdh antibodies. Asterisk indicates a shift in the molecular weight of CPY in *Cgvps15*Δ and *Cgvps34*Δ mutants. CgGapdh was used as loading control. **(C)** qPCR-based quantification of *CgPRC1* mRNA levels in indicated YPD medium-grown, log-phase *C. glabrata* strains. Data (mean ± SEM of three independent experiments) were normalized to an internal *CgGAPDH* mRNA control and represent fold change in expression in *Cgvps15*Δ and *Cgvps34*Δ mutants compared to *wt* cells. **(D)** FACS analysis of Epa1 surface expression in indicated *C. glabrata* strains. *C. glabrata* cells were grown in YPD medium for 10 h. At indicated time points, cells were collected and labelled with an anti-Epa1 antibody. A FITC-conjugated secondary antibody was used to examine cell surface expression of Epa1 and the mean of fluorescence intensity is indicated. **(E)** Adherence analysis of indicated S^{35}-labelled *C. glabrata* cells to Lec2 ovary epithelial cells. Data represent mean ± SEM of three to four independent experiments. Statistically significant differences in the percent adherence between *wt* and mutants are marked (*, p≤0.05, **, p≤0.01; ***, p≤0.001, two-tailed Student's unpaired t-test).

Project 2: Mechanisms of iron acquisition and iron homeostasis in *C. glabrata*.

Objectives

1. Identification of major iron acquisition and iron homeostasis mechanisms;
2. Identification of the *C. glabrata* genes specifically induced in response to iron availability; and
3. Role of the identified genes in iron homeostasis

This is a new activity.

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

The ability to acquire iron form host tissues is a major virulence factor of pathogenic organisms, and a significant correlation between host iron content and pathogenicity of an organism has been reported.This project is aimed at elucidation of the strategies that *C. glabrata* employs to acquire, transport, utilize and store iron in accordance with the iron availability. Using a reverse genetics approach, we have identified and attempted to disrupt *C. glabrata* orthologs of the genes that are implicated in iron uptake and homeostasis in other fungal species. These genes encode components of the high-affinity iron uptake (CgFtr1, CgFet3, CgCcc2 and CgFre6), low-affinity iron transport (CgFet4), siderophore uptake (CgSit1), iron storage and utilization (CgYfh1, CgFth1 and CgFet5), host-specific iron utilization (CgHmx1, CgCcw14 and CgMam3) and transcriptional regulatory (CgAft1 and CgAft2) systems in *C. glabrata*. Of this set of 14 genes, we were unable to delete the *CgAFT1* gene, whose ortholog in *S. cerevisiae* codes for a master regulator of the high-affinity iron uptake system, suggesting that CgAft1 may be essential for cell viability in *C. glabrata*. Phenotypic profiling of the created 13 deletion strains revealed that three mutants, *Cgftr1*Δ (disrupted for an iron permease), *Cgfet3*Δ (disrupted for a multicopper oxidase) and *Cgccc2*Δ (disrupted for a copper transporter), displayed sensitivity to iron starvation caused by cell impermeable, extracellular Fe^{2+} specific chelators, bathophenanthroline disulfonate (BPS) and ferrozine. Their attenuated growth was largely due to iron-limitation as it was rescued by supplementing the medium either with ferric chloride or hemoglobin.

Measurement of the intracellular iron levels in log-phase *C. glabrata wt* and mutant cells revealed iron content of *Cgftr1*Δ, *Cgfet3*Δ and *Cgccc2*Δ mutants to be ~ 20-50% lower than that of the *wt* cells (Fig. 2A) in accordance with their increased sensitivity to iron-limitation. However, although growth of the *Cgfet4*Δ (disrupted for low-affinity plasma membrane Fe(II) transporter) mutant was not attenuated in the iron-poor medium, it still accumulated 40% less iron levels than the *wt* strain (Fig. 2A) which may be reflective of an overall perturbed ion homeostasis. As reported for *S. cerevisiae*, disruption of the mitochondrial matrix iron chaperone, CgYfh1, led to 3.5-fold higher levels of intracellular iron in *C. glabrata* (Fig. 2A) indicating the conserved Fe-S cluster assembly machinery between these two yeasts. Surprisingly, we also observed ~ 40-50% increase in the intracellular iron content in *Cgccw14*Δ (lacking the cysteine-rich CFEM (common in fungal extracellular membranes) domain-containing cell wall structural protein) and *Cgmam3*Δ (lacking the putative hemolysin) mutants, implying a perturbed iron homeostasis.

To address the molecular mechanisms underlying high intracellular iron content in *Cgccw14*Δ, *Cgmam3*Δ and *Cgyfh1*Δ mutants, we examined expression of the genes encoding constituents of the high-affinity iron uptake system by quantitative real-time PCR (qPCR) analysis. Compared to *wt* cells, transcript levels of *CgAFT1, CgAFT2, CgFTR1, CgFET3* and *CgFET4* genes were found to be 2-, 3-, 5-, 3- and 5-fold higher, respectively, in the *Cgyfh1*Δ mutant (Fig. 2B). Further, contrary to the *Cgyfh1*Δ mutant, the *Cgmam3*Δ mutant did not display an activated high-affinity iron uptake system (Fig. 2B). Notably, transcript levels of *CgFTR1, CgFET3* and *CgFET4* were found to be about 1.5-fold higher in the *Cgccw14*Δmutant (Fig. 2B). Collectively, these data indicate that the reductive iron transport system may not be the sole regulator of iron homeostasis in *C. glabrata*, and the elevated iron content of *Cgmam3*Δ and *Cgccw14*Δ mutants could be due to yet to be identified mechanisms.

Next, to investigate which components of iron acquisition and homeostatic machinery are required for virulence of *C. glabrata*, we examined fungal burden in Balb/c mice infected intravenously either with the wild-type or the mutant strains. As shown in Figure

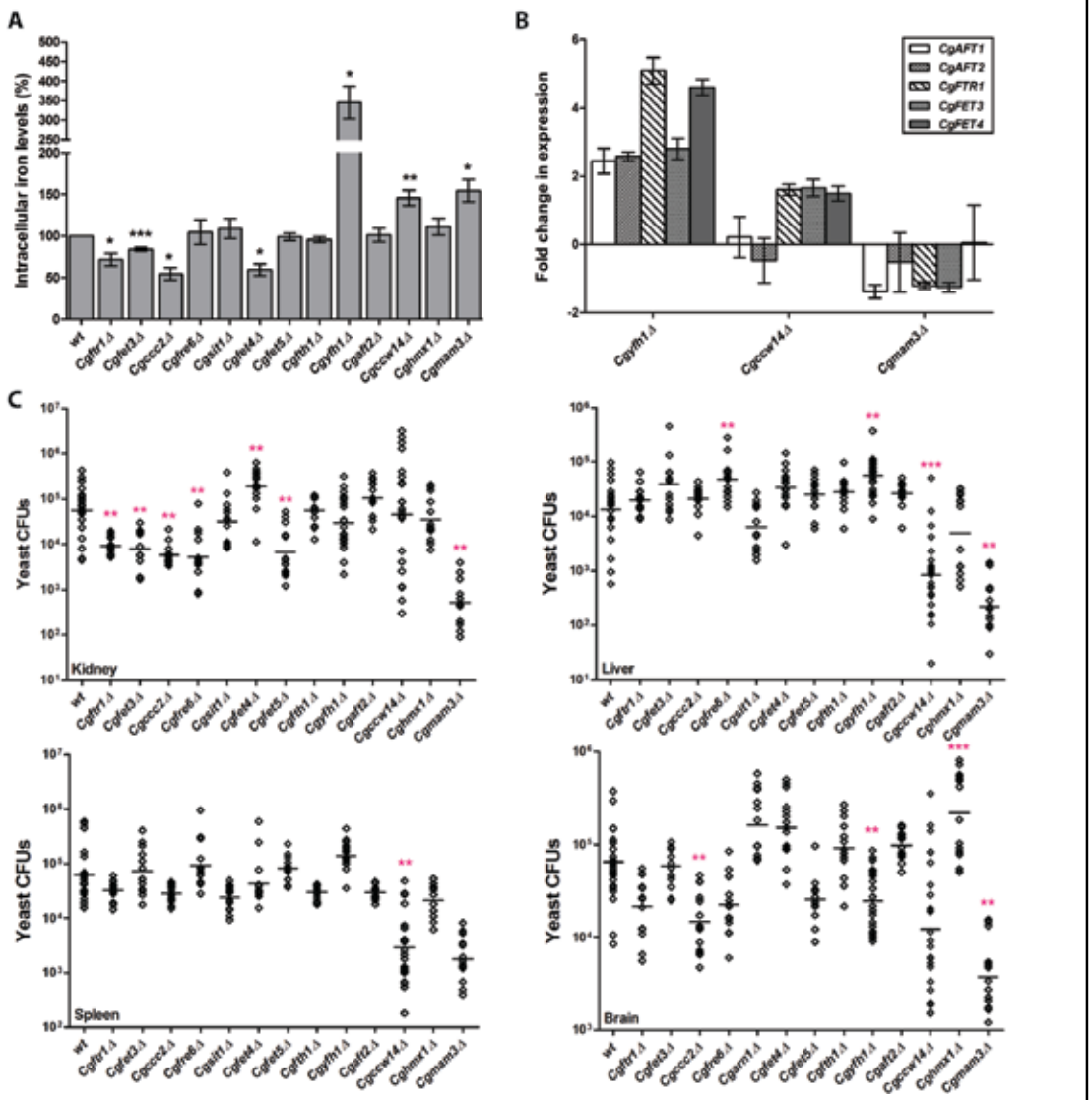

Figure 2.Genes involved in iron homeostasis are required for virulence of *C. glabrata*. **(A)** Intracellular iron content of the indicated, YPD medium-grown, log-phase *C. glabrata* cells was quantified by the BPS-Fe complex absorbance. Data are expressed as the percentage (mean ± SEM, n = 3-6) of the iron levels in the mutants relative to the wt sample (taken as 100%). Statistical analysis was performed using a paired, two-tailed, Student's t test (*, p≤0.05; **, p≤0.01; ***, p≤0.001).**(B)** qPCR analysis of *CgAFT1, CgAFT2, CgFTR1, CgFET3,* and *CgFET4* transcript levels in YPD medium-grown, log-phase wt and mutant strains. Data (means of 3-5 independent experiments ± SEM) were normalized to an internal *CgACT1* mRNA control, and represent fold change in expression in mutant cultures compared to *wt* cells.**(C)** 6-8 week-old, female BALB/c mice were infected intravenously with $4X10^7$ *C. glabrata* cells and sacrificed 7 days post infection. Diamonds represent the CFUs recovered from target organs, kidneys, liver, spleen and brain, for individual mice. Bars represent the geometric mean (n=12-24) of CFUs per organ. Statistically significant differences in the CFUs between wt and mutant strains are indicated (**, p≤0.01 and **, p≤0.001; two-tailed Student's unpaired t-test).

2C, we recovered about 6-10-fold lower yeast CFUs from the kidneys (primary target organ of infection) of mice infected with *Cgftr1*Δ, *Cgfet3*Δ, *Cgccc2*Δ, *Cgfre6*Δ and *Cgfet5*Δ mutants compared to CFUs retrieved from the kidneys of *wt*-infected mice. Notably, no statistically significant differences in the fungal burden were seen between the kidneys of *wt*- and *Cgsit1*Δ, *Cgfth1*Δ, *Cgyfh1*Δ, *Cgaft2*Δ, *Cgccw14*Δ and *Cghmx1*Δ-infected mice (Fig. 2C). Unexpectedly, fungal load in kidneys of the *Cgfet4*Δ-infected mice were 3-fold higher than the mice infected with the *wt* strain (Fig. 2C). In contrast, mice infected with the *Cgmam3*Δ

mutant displayed 20-110-fold reduced fungal burden in kidneys, liver and brain compared to *wt*-infected mice (Fig. 2C). Statistically similar yeast CFUs were obtained from the liver of *wt* and *Cgftr1*Δ, *Cgfet3*Δ, *Cgccc2*Δ, *Cgfet4*Δ, *Cgfet5*Δ, *Cgfth1*Δ, *Cgaft2*Δ, *Cgsit1*Δ and *Cghmx1*Δ-infected mice (Fig. 2C). Interestingly, the *Cgccw14*Δ mutant showed 16-fold reduced survival in the liver. In contrast, CFUs obtained from the liver of *Cgfre6*Δ- and *Cgyfh1*Δ-infected mice were 3- and 4-fold higher, respectively, than those recovered from the liver of *wt*-infected mice (Fig. 2C). In spleen, we observed 21-fold reduced yeast CFUs for the Cgccw14Δ mutant (Fig. 2C). Lastly, we recovered about $6X10^4$ yeast cells from the brain of mice infected with *wt C. glabrata* cells, while the *Cgccc2*Δ- and *Cgyfh1*Δ-infected mice exhibited 4- and 3-fold lower fungal load, respectively, in the brain (Fig. 2C). Intriguingly, the *Cghmx1*Δ mutant displayed 3-fold higher survival in the brain (Fig. 2C). It is worth noting that differential survival of iron homeostasis-defective mutants in target organs, kidneys, liver, spleen and brain, may be reflective of iron abundance in these organs. Moreover, diminished survival of the *Cgccw14*Δ mutant in the liver and the spleen of Balb/C mice underscore the role of CgCcw14 in survival in vivo. Unexpectedly, *Cgsit1*Δ and *Cgaft2*Δ mutants, disrupted for siderophore-mediated high-affinity iron transporter and transcriptional factor, respectively, exhibited virulence similar to that of the *wt* cells (Fig. 2C) indicating that *C. glabrata* cells probably do not acquire iron through the siderophore-uptake pathway in the mammalian host, and that either *CgAFT1* or another yet to be characterized transcriptional factor, is the master regulator of iron homeostasis genes under both *in vitro* and *in vivo* conditions. Although diminished survival in three organs was observed only for the *Cgmam3*Δ mutant, attenuated kidney fungal burden in mice infected with mutants disrupted for the high-affinity iron uptake system implicate *CgFTR1*, *CgFET3, CgCCC2, CgFRE6* and *CgFET5* genes in virulence of *C. glabrata* in the murine model of disseminated candidiasis. Future investigations will be directed to delineate the role of hemolysin-like protein CgMam3 and the cell wall structural protein CgCcw14 in iron homeostasis.

Publications

1. Bairwa G, Rasheed M, Taigwal R, Sahoo R and Kaur R (2014). GPI (glycosylphosphatidylinositol)-linked aspartyl proteases regulate vacuole homeostasis in Candida glabrata. ***Biochemical Journal 4*** 58:323-334.

2. Borah S, Shivarathri R, Srivastava VK, Ferrari S, Sanglard D and Kaur R (2014). Pivotal role for a tail subunit of the RNA polymerase II mediator complex CgMed2 in azole tolerance and adherence in *Candida glabrata.* ***Antimicrobial Agents and Chemotherapy*** 58: 5976-5986.

3. Shah AH, Singh A, Dhamgaye S, Chauhan N, Vandeputte P, Suneetha KJ, Kaur R, Mukherjee PK, Chandra J, Ghannoum MA, Sanglard D, Goswami SK and Prasad R (2014). Novel role of a family of major facilitator transporters in biofilm development and virulence of *Candida albicans*. ***Biochemical Journal*** 460:223-235.

4. Srivastava VK, Suneetha KJ and Kaur R (2014). A systematic analysis reveals an essential role for high-affinity iron uptake system, haemolysin and CFEM domain-containing protein in iron homeostasis and virulence in *Candida glabrata*. ***Biochemical Journal*** 463:103-114.

5. Rai MN, Sharma V, Balusu S and Kaur R (2015). An essential role for phosphatidylinositol 3-kinase in the inhibition of phagosomal maturation, intracellular survival and virulence in *Candida glabrata*. ***Cellular Microbiology*** 17:269-287.

6. Srivastava VK, Suneetha KJ and Kaur R. The mitogen-activated protein kinase CgHog1 is required for iron homeostasis, adherence and virulence in *Candida glabrata.* ***FEBS Journal*** (In press).

LABORATORY OF GENOMICS AND PROFILING APPLICATIONS

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	Soumya Rao	Senior Research Fellow
	Mugdha Singh	Senior Research Fellow
Other Members	Anilkumar Challagandla	Project Assistant (Since Oct. 2014)
	Vineesha Oddi	Project-Junior Research Fellow

Objectives

1. Human genetic diversity studies among various population groups in India; and
2. Plant-fungal interaction studies in the chilli-*Colletotrichum pathosystem*

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

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

In an attempt to build a single nucleotide polymorphism (SNP)-based panel for human identification (HID) in Indian populations, a panel of 270 identity-testing SNPs were shortlisted for genotyping individuals from different population groups. Based on studies with externally visible characteristics (EVCs) in worldwide populations, a set of 43 SNPs associated with skin pigmentation, 6 SNPs for body mass index (BMI) and 17 SNPs for body height were also shortlisted to investigate the genotype-phenotype correlation in the Indian populations. In addition to interrogating the markers on the autosomes, the uni-parental markers (mitochondrial DNA and non-recombining region of Y-chromosome, NRY), which carry the information relevant to maternal and paternal lineages, respectively were also studied to understand better the genetic diversity in these populations. We had also previously reported about the attempts to study the human salivary microbiome employing next generation sequencing (NGS) approach by investigating the informative 16S rRNA region in the microbes.

Details of progress made in the current reporting year (April 1, 2014 – March 31, 2015)

a) SNPs for HID purposes

A detailed description of the 384-plex SNP genotyping panel comprising of SNPs for identity-testing, skin pigmentation, BMI and body height was given previously. In this reporting period, ~ 370 unrelated individuals from different geographical regions of India were genotyped for the target loci using GoldenGate® Genotyping assay (Illumina, Inc, USA) according to manufacturer's instructions. Genotyping of additional samples is under progress. The resultant data is being analyzed to design a panel of 60-80 SNPs which can be employed for HID in Indian populations. The genotyping data from these individuals will also be used for the genotype-phenotype correlation studies of the various EVCs described above to assess the phenotype-informative markers in these populations.

b) Studying genetic variations in Indian populations employing uniparental markers.

As part of the population diversity studies based on uniparental markers, the genetic variations among and between Indian populations were studied by genotyping short tandem repeats (STRs) on Y-chromosome using PowerPlex® Y23 chemistry (Promega Corporation). For comparative studies, autosomal STRs from the same set of samples were also genotyped using PowerPlex® Fusion chemistry (Promega Corporation). Six newly incorporated markers viz., DYS481, DYS570, DYS576, DYS549, DYS643 and DYS533 in PowerPlex® Y23, which are scantly studied in the Indian populations, were genotyped along with other markers. The studies on these markers are expected to provide information about the allelic distribution in these populations, which could be useful in calculation of allelic frequencies for forensic HID applications. DYS570 and DYS576, the two

rapidly mutating (RM) Y-STRs markers, which have the potential to differentiate individuals even within the same paternal lineage owing to their high mutation rate (> 1 x 10^{-2}/nucleotide/generation) as compared to other Y-STRs (1 x 10^{-3}/nucleotide/generation) were also investigated. In the preliminary study, 120 male individuals from four different populations viz., Himachal Pradesh (HP), Jammu and Kashmir (JAK), Maharashtra (MH) and Rajasthan (RAJ) were genotyped for the various loci using the above chemistries and the data analysis was performed using GenALEx 6.501 and Arlequin v3.5.1.2 to infer statistical parameters such as gene diversity indices, molecular variance within and between the populations. Gene diversity from the samples studied so far for autosomal loci indicated that RAJ and MH populations have the highest and the lowest polymorphic loci, respectively. The analysis of molecular variance revealed that the variation within the studied populations was greater than the variations among populations. The significance of these findings is being investigated further.

The RM Y-STR loci, DYS570 and DYS576 were found to be the most polymorphic markers in the studied populations which is in concordance with the other reported studies. In future, in order to get a clearer and more comprehensive picture, additional samples from various populations would be genotyped for the autosomal and Y-chromosomal STR markers and compared with the rest of the world populations. For the mitochondrial (mt) DNA analysis, our preliminary studies have shown that the control (hypervariable) region alone is less informative as compared to the complete mt genome sequence; and therefore for our further studies it was decided to adopt a high throughput NGS strategy for analysis of the complete mt genome.

c) Studies on human salivary microbiome in Indian populations.

The NGS data of salivary microbiome consisting of variable regions (V1 and V2) of the 16S rRNA region from ~90 individuals from eight different geographical locations in India, viz., South India (Tamil Nadu, Andhra Pradesh and Telangana comprising 12, 11 and 12 samples, respectively), North India (Jammu & Kashmir and Uttarakhand comprising 12 and 10 samples, respectively) and Eastern India (Jharkhand, West Bengal and Assam comprising 11, 14 and 10 samples, respectively), obtained by employing Illumina MiSeq platform (Illumina, Inc, USA) was analyzed. A total of 2,766,655 reads were obtained after preliminary processing of the raw data. The length of the reads varied from 251 to 489 bases (Median length = 356). A stringent filtering criterion was used to process the initial data to discard the uninformative reads as well as the sequences containing two or more barcode sequences, or no barcodes, or no primer sequences or primer sequences in the middle of the reads. Further, the reads containing ambiguous bases (N), or a homopolymer stretch of more than 8 bases or reads either extremely small (<330 bases) or large (> 430 bases) were also discarded with the help of mothur software. Finally, a total of 2,558,248 reads obtained after filtration were used to compare the 16S rRNA sequences from different individuals.

The filtered sequence reads were imported to USEARCH and were dereplicated and sorted by size after discarding the singletons. The reads were then clustered at 97% identity to identify the species-level Operational Taxonomic Units (OTUs). In order to discard the chimeras generated during the amplification steps of the library preparation, the processed reads were compared to the GOLD database incorporated in USEARCH and the chimeras so detected were discarded. The filtered OTUs were serially numbered and stored in a new database. A total of 785 high quality unique OTUs were obtained in the current study. To identify the bacterial genera present in each sample, the filtered reads obtained were aligned by mothur, followed by BLAST at 80% identity cutoff against the 16S Ribosomal Database Project. For the unifrac analysis, the OTUs were aligned in mothur using the 16S rRNA Silva database as template and the aligned sequences were utilized to construct a phylogenetic tree using the generalized time-reversible (GTR) model available in Fasttree which was subsequently used in FastUnifrac.

The preliminary results showed plenty of sharing of the OTUs among the individuals irrespective of their geographical location. This observation was also corroborated with AMOVA studies based on the abundance of bacterial genera which showed that the distribution of variance between the studied populations was much smaller as compared to the diversity within populations across the major geographical locations. It is known from earlier studies that the salivary microbiome could be affected by

climatic conditions, food habits, health status and ethnicity among others and therefore it is possible that the low sample size per population (10-12) was not sufficient to bring out the variations, in the tested samples. It is proposed to study microbial diversity in additional samples from these and other geographical locations in India by NGS strategy to better understand the salivary microbiome and to investigate whether there is a core microbiome for Indian populations

Project 2: Plant-Fungal Interaction studies in the Chilli - *Colleotrichum* Pathosystem.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

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 six *Colletotrichum* species, the chilli - *C. truncatum* pathosystem offers an excellent model system for studies on the infection process and molecular interactions between the host and 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 random insertional mutagenesis.

We have earlier reported the *de novo* whole genome sequencing of *C. truncatum* employing the Illumina HiSeq platform (2x100 bp reads) and that the sequence assembly consisted of 81 scaffolds with a total length of 55.3 Mb, equivalent to 460X coverage. For preliminary annotation of the assembly, scaffolds were aligned to the predicted gene set of well annotated *C. higginsianum* genome using BLASTX with threshold expect value of $1e^{-3}$ identifying 6,511 unique genes. In the other experiment, in order to identify pathogenicity genes in *C. truncatum* through forward genetics approach, random insertional mutagenesis of *C. truncatum* conidia by *Agrobacterium tumefaciens* mediated transformation (ATMT) was taken up using *A. tumefaciens* strain C58C1 harboring binary vector pBIN-GFP-hph and resultant fungal transformants were selected on potato dextrose agar (PDA) containing hygromycin. The mitotically stable transformants were screened for partial or complete loss of pathogenicity on chilli.

Details of progress made in the current reporting year (April 1, 2014- March 31, 2015)

a) Whole genome *de novo* sequence analysis

The whole genome sequence of *C. truncatum* was analysed by a computational method, Core Eukaryotic Genes Mapping Approach pipeline (CEGMA v. 2.5), to derive an initial set of reliable annotation of core genes with accurately identified exon-intron structures that helps in automated annotation of the genes in a draft assembly. The CEGMA core genes dataset, built using the NCBI euKaryotic clusters of Orthologous Groups (KOGs) database, consists of 458 core proteins that are present in a wide range of taxa. The CEGMA pipeline could also be used for assessment of completeness of the assembly based on coverage of orthologs of 248 core eukaryotic genes (CEGs). The genome assembly was found to be 94.76% (235/248 CEGs covered) complete in CEGMA analysis. The missing KOGs (16/458) in CEGMA output could be identified through tBLASTn of *C. gloeosporioides, C. graminicola* and *C. higginsianum* gene models to *C. truncatum* genome with high alignment score ($<1e^{-10}$, 78-100% of query coverage). The sequences of these reliable 458 core eukaryotic genes would be used in training *ab-initio* gene prediction programs for complete genome annotation in future.

A phylogenetic analysis was carried out for the genus *Colletotrichum* based on multilocus alignment of the five genes [internal transcribed spacer of rRNA (ITS), chitin synthase-1 (CHS-1), histone3 (HIS3), actin (ACT) and tubulin (TUB2)] in *C. truncatum* and 27 other *Colletotrichum* species using MEGA 6 by neighbor joining (NJ) method with 1000 bootstrap replicates. *Monilochaetes infuscans* was taken as an outgroup for the analysis (Fig. 1). *C. truncatum* (MTCC no. 3414) that infects chilli clustered together with the other isolate of *C. truncatum* that infects *Phaseolus lunatus* and was found to be closely related to gloeosporiodes clade. The position of *C. truncatum* in the cladogram would help in carrying out comparative genomics studies with the other sequenced species of *Colletotrichum.* Further analysis of the genomic data for gene annotation by *ab initio* gene prediction methods and functional characterization of pathogenicity genes would be carried out in future.

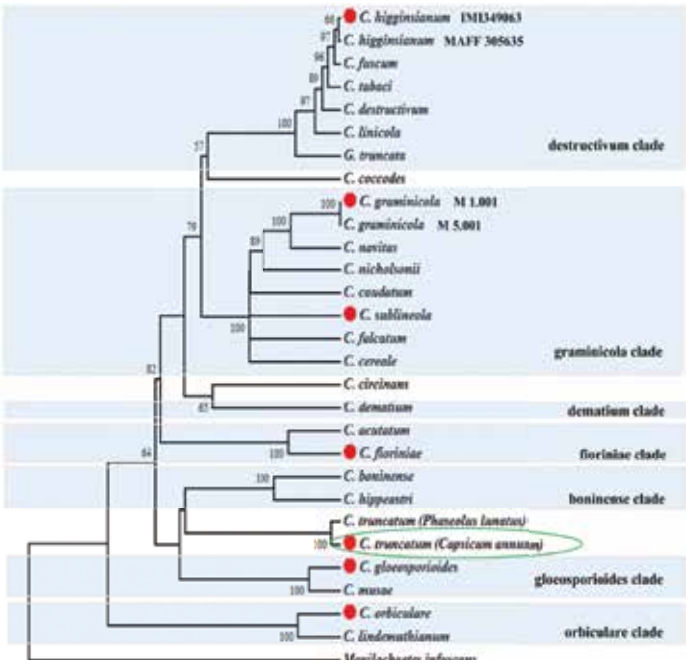

Figure 1. Phylogenetic tree of 27 Colletotrichum species obtained with Neighbour Joining method; the sequenced species are marked with red circles and the species of C. truncatum that was sequenced de novo in the present study is highlighted inside the green circle. The seven clades have been shown in gray scale.

b) Pathogenicity assay of fungal transformants

Around 1000 *C. truncatum* transformants generated through ATMT were screened for the complete or partial loss of pathogenicity on chilli. The conidia were collected from the transformant colonies growing in 24 well plates by flooding the wells with Milli-Q water and the conidial suspensions were used to inoculate *C. annuum* fruits at mature green stage for pathogenicity assay through wound and drop method. The fruits inoculated with Milli-Q water and wild type conidia were used as negative and positive controls, respectively.

There were several transformants that produced much smaller and inconspicuous lesions as compared to the wild type fungus. To confirm the loss of pathogenicity, secondary and tertiary screenings were performed in which most of the transformants reverted back to pathogenic phenotype. Two of the transformants were found to retain the non-pathogenic phenotype in secondary and tertiary screens so far, whose molecular characterization would be carried out in future. Further, additional mutants with loss of pathogenicity would be characterized to understand host-pathogen interactions at the molecular level.

Publications

1. Sharma V and Nandineni MR (2014). Assessment of genetic diversity among Indian potato (*Solanum tuberosum* L.) collection using microsatellite and retrotransposon based marker systems. ***Molecular Phylogenetics and Evolution*** 73: 10-17.

LABORATORY OF IMMUNOLOGY

Understanding the role of Profilin to suppress tumorigenesis

Faculty	Sunil K Manna	Staff Scientist
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	Neeharika Verma	Senior Research Fellow
	Pankaj Gupta	Senior Research Fellow
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	Nune Raviprakash	Project-Senior Research Fellow
	Binay Kumar Sahoo	Project-Junior Research Fellow
Collaborators	Biswadev Bishayi	Calcutta University, Kolkata
	Tushar Basu Baul	NEHU, Shilong

Objectives

1. Understanding and regulation of inflammatory and tumorigenic responses;
2. Understanding and regulation of advanced glycation endproducts (AGE)-mediated lipogenesis; and
3. Understanding the molecular mechanism of autophagy.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

Advanced glycation end products (AGE) accumulate in diabetic patients and aged persons due to high amounts of 3- or 4-carbon derivatives of glucose. AGE increased lipid accumulation not only in liver cells, but also in other cell types as shown by Oil Red O staining. AGE-mediated upregulation of several transcription factors, like NF-κB, AP-1, NRF, SREBP, etc. Antioxidant like NAC or known activator troglitazone, an anti-diabetic agent, except mangiferin, a c-glycosyl xanthone glucoside and a known polyphenol, were unable to protect AGE-induced activation of SREBP and subsequent lipid accumulation. Mangiferin not only inhibits AGE-mediated ROI generation that requires NF-κB activation, but also inhibits ERK and IKK activity, thereby suppression of SREBP activity and lipogenesis. Mangiferin has shown a double-edged sword effect to suppress AGE-mediated ailments by reducing ROI-mediated responses as antioxidant and inhibiting SREBP activation thereby lipogenesis, suggesting its potential efficacy against diabetes and obesity-related diseases.

We have investigated the molecular mechanism for the antioxidant property of mangiferin. Mangiferin blocks TNF-induced NF-κB and AP-1 activation in a dose dependent manner. Mangiferin, like known anti-oxidants inhibits TNF-induced reactive oxygen intermediates (ROI) generation, but was most potent in inhibiting NF-κB and AP-1 activation induced by TNF as well as other inflammatory agents like PMA, endotoxin, oleamide and H_2O_2. Mangiferin was found to increase the catalase activity in vitro and thereby reduced lipid peroxidation more potently than known inhibitor of catalase, aminotriazole (ATZ). Mangiferin and ATZ interact with the catalytic site of catalase, but in separate amino acid residues and the predicted amino acids were detected. The affinity of catalase is more with mangiferin than ATZ as detected from the free energy binding data. Hence mangiferin with its ability to inhibit NF-κB and to increase the catalase activity may prove to be a potent drug for anti-inflammatory and anti-oxidant therapy.

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

1) Profilin-PTEN interaction suppresses NF-kappa B activation via inhibition of IKK phosphorylation.

The molecular mechanism of Profilin for its tumor suppressor activity is still unknown. NF-κB is known to activate many target genes involved in cell proliferation. In this study, we provide evidences that support the involvement of Profilin in regulation of NF-κB, which might repress the tumorigenic response. Transient transfection

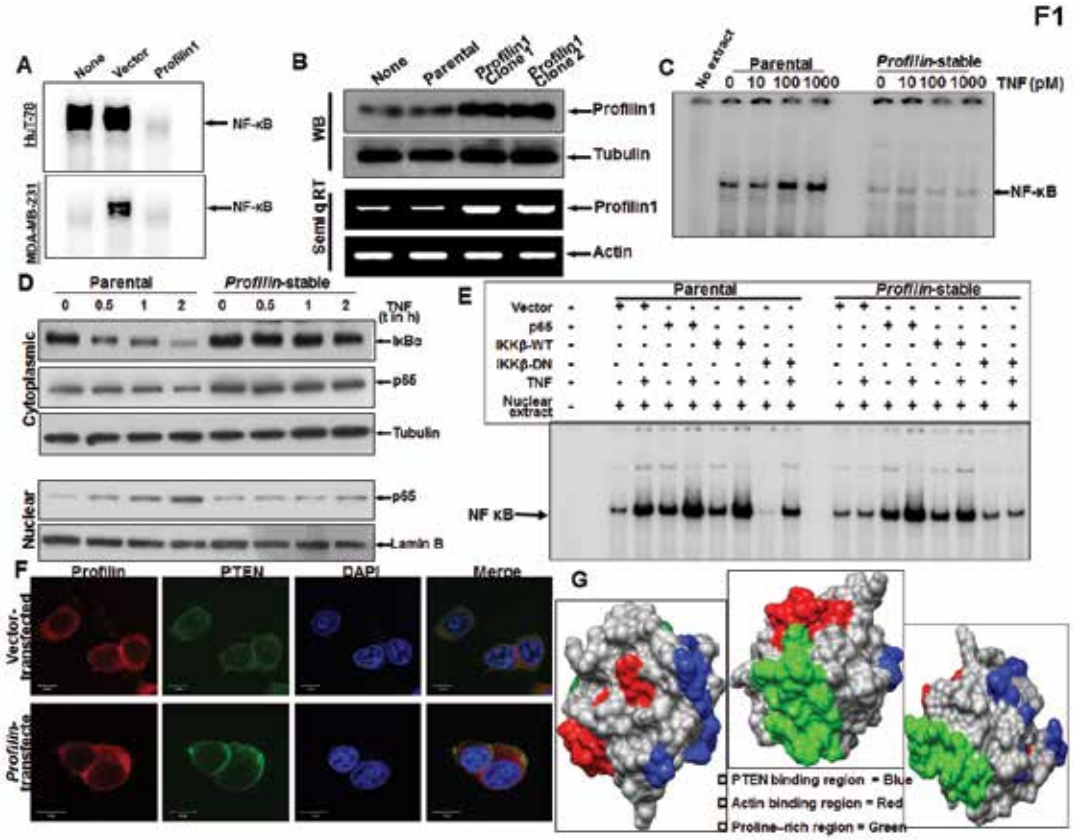

Figure 1. Effect of *Profilin* on NF-κB activation. HuT-78 and MB-231 cells were transfected with vector or *Profilin1* for 6 h followed by culture for 24 h. The nuclear extracts (NE) prepared and NF-κB DNA binding was assayed by gel shift assay (A). Stable *Profilin* overexpressing and vector control are generated in MB-231 cell lines. Western blot and RT-PCR further confirm *Profilin* overexpression in these cells (B). Parental and Profilin-stable MB-231 cells were treated with varying concentrations of TNF for 1 h. NE were prepared and assayed for NF-κB DNA binding activity (C). Parental and *Profilin*-stable MB-231 cells were stimulated with 1 nM TNFα for different time. NE and CE (cytoplasmic extracts) were prepared and IkBa was measuser from CE and p65 from CE and NE by Western blot (D). Parental and *Profilin*-stable cells were transiently transfected with *IKK*β*, p65,* or *IKK*β*-DN* constructs with reporter construct NF-κB-SEAP and GFP. After 12 h, GFP positive cells were counted. Cells were then treated with TNF for 30 min. NE were prepared and assayed for NF-κB DNA binding by gel shift assay (E). MB-231 cells were transiently transfected with *Profilin* and immunofluorescence staining using antibody of *Profilin* (green) and PTEN (red) was done. The right panels are the overlay of Profilin, PTEN and nuclear 4′phenylindole (DAPI; blue) staining of the same field (A). Scale bar, 10 μm (F). Different regions of *Profilin* interacting with PTEN (blue), Actin (red) and proline rich region (green) is shown (G).

of profilin shows the decreased amount of NF-κB DNA binding either in high basal or induced activity of NF-κB (Fig.1A). This prompts us to profilin-stable cells and breast tumor cells (A-231) cells were used to make this stable cell generation as shown by Western blot and RT-PCR (Fig.1B). Profilin overexpressing cells show low basal activity of IKK, high amount of cytoplasmic IκBα and p65, and low nuclear NF-κB DNA binding activity (Fig.1C & 1D). To determine the mechanism of Profilin-mediated suppression of NF-κB, parental and Profilin-stable cells were transfected with IKKβ full-length (*IKK*β), IKKβ kinase dead domain (*IKK*β-DN), or *p65* and then stimulated with TNF. The *IKK*β or *p65* transfected cells showed increase in the NF-κB DNA binding activity and TNF increased this activation marginally in parental cells. In Profilin-stable cells, Profilin did not suppress NF-κB activation when transfected with p65 or IKKβ, with or without TNF stimulation (Fig.1E). This suggests that Profilin might be acting at the upstream level of p65. Co-localization (Fig.1F) and *in silico* studies (Fig.1G) suggest that Profilin interacts

with a protein phosphatase, PTEN and protects it from degradation. In turn, PTEN physically interacts and maintains low phosphorylated state of IKK complex and thereby suppresses NF-κB signaling. Thus, Profilin overexpressing cells show decrease in NF-κB activation mediated by most of the inducers and potentiates cell death by repressing NF-κB-dependent genes involve in cell cycle progression.

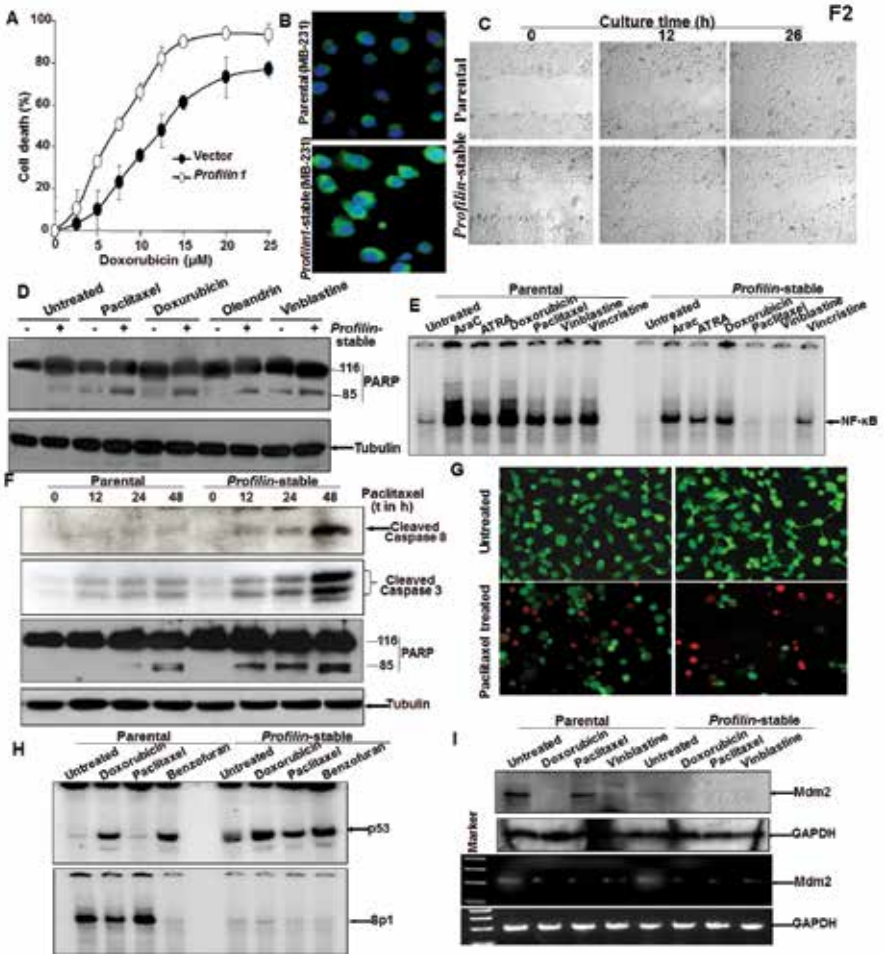

Figure 2. Effect of *Profilin* on chemotherapeutic agents mediated cell death. MDA-MB-231 cells were transiently transfected with *Profilin 1* for 24 h, followed by treatment with different concentrations of doxorubicin for 48 h. The cell viability was assayed by MTT dye and the absorbance was taken at 570 nm and indicated as percentage of cell death (A). MDA-MB-231 cells were prepared for *Profilin 1* stable cells (*Profilin*-stable). Parental and *Profilin*-stable cells were subjected to immunocytochemistry to detect the amount of *Profilin* using anti-Profilin antibody followed by FITC-conjugated secondary antibody (B). Parental and *Profilin*-stable cells were made scratch at 90% confluency with sterile needle and images were captured at different time intervals in phase contrast microscope (C). Parental and *Profilin*-stable MB-231 cells were treated with paclitaxel, vinblastine, doxorubicin and oleandrin for 48 h. The amount of PARP was determined from WCE of these cells with similar treatments and detected by Western blot (D). Parental and *Profilin*-stable cells were treated with ATRA, AraC, doxorubicin, vinblastine, paclitaxel, or vincristine for 6 h. Cells were washed with PBS and pelleted. Nuclear extracts (NE) were prepared and used to measure NF-κB DNA binding by gel retardation (E). Parental and *Profilin*-stable cells were treated with 200 nM Paclitaxel in a time dependent manner. Cells were washed and extracted for WCE. 100 μg WCE protein was then probed for PARP, cleaved caspase 8 and 3 by Western blot (F). Cell death was detected by Live & Dead Cytotoxicity assay kit from similar treatment and detected by Calcein (for live cells, stained as green) and Ethidium homodimer (for dead cells, stained as red) stained cells (G). The amount of p53 and Sp1 DNA binding was determined from 8 μg of NE extracted from parental and *Profilin*-stable cells treated with doxorubicin, paclitaxel and benzofuran for 6 h by gel shift assay (H). The amount of Mdm2 was determined by Western blot and RT-PCR from same treatment (I).

Mutation in Profilin is known to cause Familial amyotrophic lateral sclerosis (ALS), a neurodegenerative disorder resulting from motor neuron death. This suggests there might be a possibility that alterations in other functions of Profilin may contribute to the tumorigenesis. As Profilin is downregulated in human breast tumors and correlates with low PTEN expression, we propose that mutation or loss of Profilin function may drive mammary cells for tumor progression. Modulating the expression level of Profilin may be useful in mitigating the tumorigenic growth and can be targeted along with other agents that are used against different pathways for effective combination therapy.

2) Profilin potentiates chemotherapeutic agents mediated cell death via suppression of NF-kappaB and upregulation of p53.

Profilin acts as tumor suppressor. The mode of action to exert this effect is somehow unknown. Several chemotherapeutic agents used till date either have unfavorable side effects or developing resistance. In this study, we have investigated the mechanism by which Profilin negatively regulates cell survival. As, NF-κB and p53 are the key players in apoptosis, we are detecting any role of Profilin in their regulation. Role of Profilin in chemotherapeutic agents mediated cells death was determined by several apoptotic assays, such as MTT cell viability assay (Fig.2A), cleavage of PARP (Fig.2D) and caspases (Fig.2F) are determined by Western blot; morphology is visualized by phase contrast microscope; nuclear fragmentation and dead cells are determined by flow cytometer and fluorescence microscope (Fig.2G). Transcription factors, like NF-κB, p53 and Sp1 are determined by gel shift assay and their dependent genes are by RT-PCR and reporter gene luciferase assay. Profilin potentiates several chemotherapeutic-agents mediated cell death. Profilin overexpression suppressed migration and invasiveness of breast cancer cells (Fig.2C). Paclitaxel and vinblastine-mediated NF-κB (Fig.2E) and NF-κB-dependent genes activation was completely inhibited in Profilin overexpressing cells, as determined by the amount of profilin in these cells (Fig.2B). The increased p53 DNA binding activity was potentiated in Profilin overexpressing cells (Fig.2H). The Sp1 DNA binding followed by Mdm2 expression was completely abrogated in Profilin overexpressing cells (Fig.2H & 2I). Thus, Profilin suppress NF-κB activation and increase p53 activity by suppressing Sp1 and thereby, Mdm2 expression. Profilin synergizes with chemotherapeutic drugs to induce tumor cell death by attenuating NF-κB and upregulating p53. Thus, modulation of Profilin may be useful for effective combination therapy.

Publications

1. Basu Baul TS, Kundu S, Linden A, Raviprakash N, Manna SK and Guedes da Silva MF (2014). Synthesis and characterization of some water soluble Zn(ii) complexes with (E)-N-(pyridin-2-ylmethylene)arylamines that regulate tumour cell death by interacting with DNA. ***Dalton Transactions***. 43: 1191-1202.
2. Mahali SK, Verma N and Manna SK (2014). Advanced glycation end products induce lipogenesis: regulation by natural xanthone through Inhibition of ERK and NF-κB. ***Journal of Cellular Physiology*** 229: 1972-1980.
3. Raviprakash N and Manna SK (2014). Short-term exposure to oleandrin enhances responses to IL-8 by increasing cell surface IL-8 receptors. ***British Journal of Pharmacology*** 171: 3339-3351.
4. Ghosh C, Prakash NR, Manna SK and Bishayi B (2015). Presence of toll like receptor-2 in spleen, lymph node and thymus of Swiss albino mice and its modulation by Staphylococcus aureus and bacterial lipopolysaccharide. Indian Journal of Experimental Biology 53: 82-92.

LABORATORY OF MAMMALIAN GENETICS

Epigenetic mechanisms underlying developmental pathways

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	Thushara Thamban	Senior Research Fellow
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	M Srilalitha	Technical Officer
	Vaishnavo Pai	Project Associate (Till Mar. 2015)
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	Shekhar Mande	NCCS, Pune
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	Sharmistha Banerjee	UoH, Hyderabad

Project 1: *DNMT3L*: Role in Development

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

Previous work from our laboratory has examined the reason for loss of DNA methylation at DNMT3L promoter in cancer samples. Based on reporter gene assays we showed presence of a PRE with this promoter region. In addition, we showed DNMT3L to be involved in nuclear reprogramming.

Details of progress made in the current reporting year (April 1, 2014- March 31, 2015)

We uncovered the role of DNMT3L in nuclear reprogramming when HeLa cells overexpressing DNMT3L were found to have undergone nuclear reprogramming gradually and showed morphological changes only in the 20th generation post transfection of *DNMT3L* construct (Gokul et. al. 2009; Epigenetics 4: 322-329).

When transgenic Drosophila that ectopically expressed *DNMT3L* (carrying *Tubulin-Gal4* driver) were maintained for more than 5th generations, some of the flies showed melanotic tumors. None of these larvae with melanotic tumors survived beyond the larval stages. All adult flies had melanotic tumors and were normal and fertile. In all the subsequent generations (maintained till G20), 5-8% of the 3rd instar larvae consistently showed melanotic tumors and did not survive whereas the rest of the progeny were normal and fertile. Real-Time RT-PCR analysis showed that the expression of DNMT3L remained constant in all the generations from G1 to G5 suggesting that the appearance of the larvae with tumors in 5th generation progeny was not due to an abrupt change in its expression. This was true for all DNMT3L transgenic Drosophila lines as also with the use of other *Gal4*-drivers (*Actin* and *Daughterless*).

Since the melanotic tumors were present in the hemolymph the influence of DNMT3L expression on the number and types of hemocytes in the hemolymph was examined by comparing the number of hemocytes in control *UAS-DNMT3L*

flies and 5th generation *Tubulin-DNMT3L* flies with or without melanotic tumors by staining with Phalloidin. The number of proliferating hemocytes were significantly more in *Tubulin-DNMT3L* flies (both with and without melanotic tumors; Fig. 1A, B). The types of hemocytes present in the hemolymph was also markedly different with the *Tubulin-DNMT3L* flies showing a large numbers of lamellocytes (Fig. 1C).

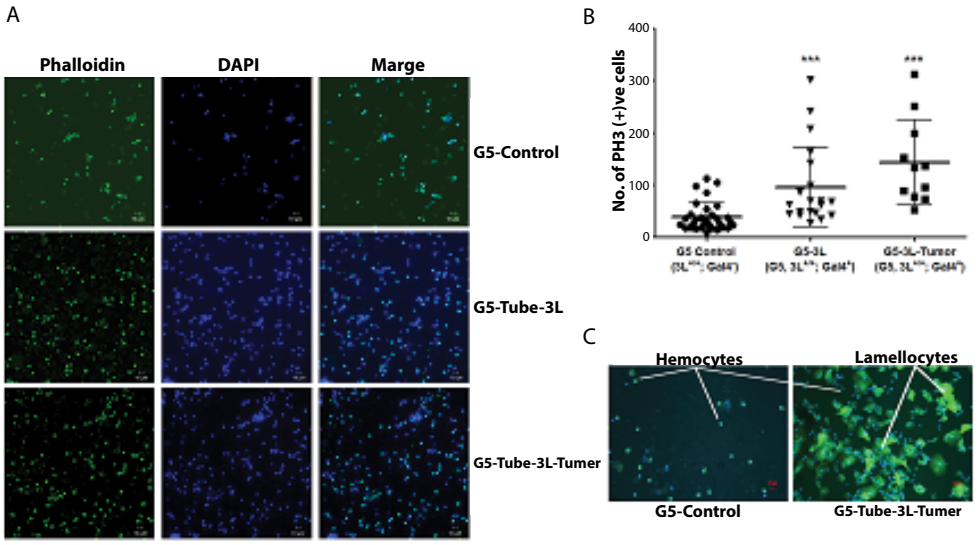

Figure 1. Ectopic DNMT3L expression affects the number and types of hemocytes. (A) Florescent microscopic images of Phalloidin stained hemocytes from the control and the DNMT3L expressing transgenic flies in the fifth generation. A significant increase in the number of hemocytes in the DNMT3L expressing transgenic flies expressing Dnmt3L was observed and is graphically represented in (B). G5-Control- 5th generation transgenic *UAS-DNMT3L* flies (without any *Gal4* driver). G5-*Tub-3L*: 5th generation transgenic flies expressing DNMT3L with *Tubulin-Gal4* driver. G5-*Tub-3L*-tumor: 5th generation transgenic flies expressing DNMT3L with melanotic tumors. The error bars represent Standard Deviation (S.D.). * indicate significant difference (Student's t test, *** - $p < 0.001$). (C) Phosphohistone 3 (PH3) staining of the hemolymph prepared from 5th generation control and DNMT3L expressing larvae (with melanotic tumors).

Project 2: Host epigenetic response to infection

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

We have previously identified a putative DNA methyltransferases (DNMTs), Mtbmeth1, which had the ability to be secreted out of a mycobacterial cell and localize to the THP1 nucleus in a transient transfection assay. Mtbmeth1 protein was also found to be phosphorylated.

Details of progress made in the current reporting year (April 1, 2014- March 31, 2015)

To determine the specificity of DNA binding activity of Mtbmeth1 protein (Rv2966c), EMSA analysis was done with 100-300 bp sonicated THP1 DNA fragments. Rv2966c-bound DNA fragments in the EMSA gels were excised, cloned and sequenced. A few identified DNA sequences from them were PCR amplified and used in EMSA analysis. Rv2966c showed binding to all of them and as expected, phosphorylated Rv2966c bound DNA much more strongly than the unphosphorylated form (Fig. 2A). But when pBluescriptSK MCS was used as a DNA substrate, negligible binding was observed (Fig. 2A), suggesting that Rv2966c was binding to specific DNA sequences.

DNA methylation analysis was done by performing Bisulfite sequencing on in vitro Rv2966c methylated DNA fragments (from the *H2AFY2* gene) to examine whether Rv2966c was capable of methylating these specific DNA sequences. As compared to control (incubation

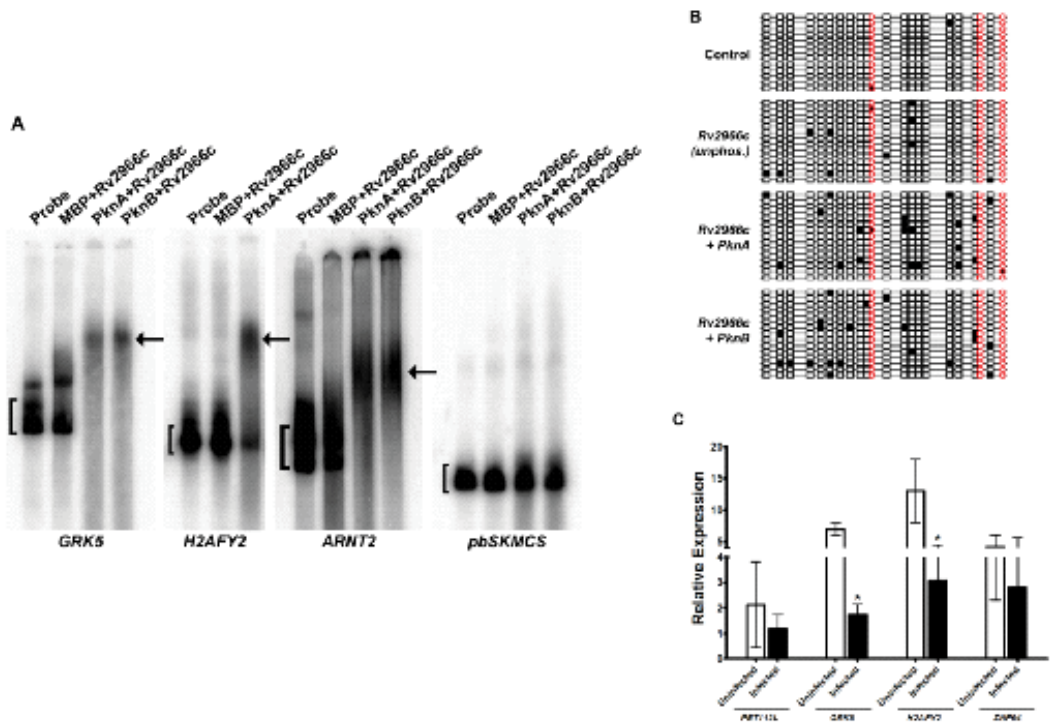

Figure 2. Rv2966c binds and methylates non-CpG cytosines within specific DNA sequences from the THP1 genome. A.) Rv2966c binds specific DNA sequences. α^{32}P-labelled DNA probes were prepared for three fragment as indicated and control pBluescript vector. EMSA was done with both unphosphorylated and PknA/PknB phosphorylated proteins in the presence of poly (dI-dC). pbSKMCS is the probe from the pBluescript vector. Brackets indicate the band corresponding to the probe; Arrow indicates the DNA-protein complex band. B.) Bisulfite analysis of *in vitro* Rv2966c methylated *H2AFY2* gene fragment. 13 or more clones were screened for each sample. Control indicates incubation with BSA. Red circles represent CpG dinucleotides. Non-CpG cytosines in CpA, CpC and CpT dinucelotides are represented by rectangles. Filled symbols indicates methylation of cytosine in the dinucleotide. C.) Real-time RT-PCR was performed for the indicated genes (mentioned below the X-axis) on RNA isolated from uninfected and *M. tuberculosis* H37Rv infected (48 hrs post infection) THP1 macrophages. To take into account the effect of culturing on expression the levels were normalised against the expression of these gene at the time of infection (0 hrs). Open bars indicate expression in uninfected cells while filled bars are for infected cells. The experiment was done at least thrice in duplicates. The error bars represent Standard Deviation (S.D.). * indicate significant difference (Student's t test, * - $p < 0.05$).

with BSA), incubation with both phosphorylated (PknA/PknB) and unphosphorylated Rv2966c protein showed significant methylation. Surprisingly, instead of CpG methylation (denoted by red circles), significant amount of non-CpG methylation (denoted by boxes), specifically in CpA and CpT dinucleotides was observed (Fig. 2B).

To examine the functional significance of the observed non-CpG methylation during infection, expression of some of these genes was examined upon *M. tuberculosis* H37Rv infection of THP1 cells by Qualitative Real-time RT-PCR. As can be seen in figure 2C, significant decrease in expression was seen for the *H2AFY2*and *GRK5* genes but not for *PET112L* and *ZNF64.*

Work is also ongoing in the laboratory on two mycobacterial proteins that can work as DNA demethylase and histone methyltransferase in the host cell.

Publications

1. Basu A, Dasari V, Mishra RK and Khosla S (2014). The CpG island encompassing the promoter and first exon of human DNMT3L gene Is a PcG/TrX response element (PRE). ***PLoS One*** 9:e93561.
2. Sharma G, Upadhyay S, Srilalitha M, Nandicoori VK and Khosla S. The interaction of mycobacterial protein Rv2966c with host chromatin is mediated through non-CpG methylation and histone H3/H4 binding. ***Nucleic Acids Research*** (In press).

LABORATORY OF MOLECULAR CELL BIOLOGY

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

Faculty	Sangita Mukhopadhyay	Staff Scientist
PhD Students	Nazia Parveen	Senior Research Fellow (Till Sep. 2014)
	Atul Udgata	Senior Research Fellow
	Gourango Pradhan	Senior Research Fellow
	Parul Singh	Senior Research Fellow
	Vishwanath Jha	Senior Research Fellow
	D Komal Chandreshkumar	Junior Research Fellow
	Shruti Srivastava	Junior Research Fellow
	KM Rohini	Junior Research Fellow (Since Aug. 2014)
Other Members	R Nagender Rao	Scientist
	Niteen Pathak	Senior Technical Officer
	Philip Raj Abraham	ICMR Research Associate
	Asma Ahmed	DBT Research Associate (Till Jun. 2014)
	Nidhi Shrivastava	DBT-Research Associate (Since Jan. 2015)
	Khalid Hussain Bhat	Research Associate
	Rahila Qureshi	Project-Junior Research Fellow
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	Sudip Ghosh	NIN, Hyderabad
	SE Hasnain	IIT, Delhi
	Jala Ramachandra Reddy	IICT, Hyderabad
	RBN Prasad	IICT, Hyderabad

Objectives

Signal transduction pathways in macrophages regulating its innate-effector functions and mechanisms by which *Mycobacterium tuberculosis* interferes with macrophage-signaling cascades to modulate host's protective responses.

Project 1: Examining virulence mechanism of ESAT-6 protein.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

Mycobacterium tuberculosis is a highly successful pathogen that has evolved several mechanisms to manipulate the host immune regulatory network. Despite a host of studies highlighting modulation of immune responses by ESAT-6, there have not been many that identified host proteins interacting with ESAT-6. Therefore, we hypothesized that the crucial role played by ESAT-6 in the virulence of mycobacteria could be due to its interaction with some host cellular factors. Thus, a yeast two-hybrid screen was set up to identify host protein(s) that interacts with ESAT-6.

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

a. ESAT-6 interacts with human β2M:

For yeast two-hybrid (Y2H) screening, ESAT-6 cloned in the bait vector pGBKT7 was used to screen a human leukocyte cDNA library cloned into the prey vector pACT2. The Mat-α strain AH109 harboring the bait vector pGBKT7-ESAT-6 was mated with Mat-α strain Y187 transformed

with prey library plasmid and the mating mixture was plated on QDO plates (SD/–Ade/–His/–Leu/–Trp) for high stringency of selection. The prey plasmids, rescued from the colonies that appeared on selection plates, were sequenced using 3' AD Sequencing Primer and were identified by querying these sequences against the NCBI GenBank database using the MegaBlast program. One of these cDNA sequences in the prey plasmid was found to have very high similarity with human beta-2-microglobulin (β2M) (Fig. 1A). The physical interaction between ESAT-6 and β2M was confirmed by a co-purification assay where ESAT-6 was cloned into the first multiple cloning site (MCS) with an N-terminal His-tag and β2M cDNA from the prey plasmid was cloned into the second MCS without any tag in a dual expression vector pETDuet-1. When the over-expressed His-tagged ESAT-6 protein was purified from the transformed *Escherichia* coli using TALON affinity binding resin, β2M was found to be co-purified along with His-tagged ESAT-6, indicating a positive interaction between ESAT-6 and β2M. The GST pull-down and co-immunoprecipitation assays indicated that ESAT-6 could interact with the β2M naturally expressed in macrophages (Fig. 1A). Physical interaction of ESAT-6 and β2M was further confirmed by using surface plasmon resonance, the Kd value of the ESAT-6:β2M complex as determined from the surface plasmon resonance data was 1.03×10^{-6} M. ESAT-6 in complex with CFP-10 is also capable of interacting with β2M and the C-terminal six amino acid residues (90-95) of ESAT-6 are found to be crucial for this interaction. CFP-10 and ESAT-6 stabilizes each other through extensive hydrophobic interactions spanning their core helix-loop-helix structure, while the free flexible C-terminal end of ESAT-6 is available for interaction with β2M and possibly other host proteins. The ESAT-6:β2M interaction is not affected by high salt concentration and pH. β2M is an integral part of the functional MHC-I molecules, and our data suggest that ESAT-6 binds only to free β2M, but not to that already in complex with HLA-I heavy chain.

b. ESAT-6 or ESAT-6:CFP-10 is trafficked into the ER:

In order to have a pathophysiological role in the host-pathogen interaction, ESAT-6 and/or ESAT-6:CFP-10 must find its way to the cellular compartments where β2M is present. β2M is known to be present in high concentration within endoplasmic reticulum (ER) where it is synthesized and undergoes necessary post-translational modifications and associates with the alpha chain of the MHC-I, as well as other class I like molecules like CD1 and hemochromatosis protein (HFE) that are transported to the cell surface via the Golgi apparatus. To test whether ESAT-6 is able to enter the ER network, THP-1 macrophages were incubated for 2 hours with FITC-labelled ESAT-6 or ESAT-6:CFP-10 and their localization was tracked along with the ER-specific marker calnexin by confocal microscopy. The ESAT-6:CFP-10 was found to be present in the ER (Fig. 1B). Similarly, FITC-labelled ESAT-6 alone was found to be present in the ER-tracker dye-positive regions of KG-1 dendritic like cells. We next over-expressed FLAG- or GFP-tagged ESAT-6 in cells in an attempt to mimic physiological conditions where ESAT-6 is secreted directly into the cytosol. We transfected HEK-293 cells with pcDNA 3.1(+)-FLAG-esat-6 and staining with anti-FLAG Ab indicated presence of ESAT-6 in the calnexin-positive ER compartments (Fig. 1C) suggesting that intracellular ESAT-6 can also find its way into the ER. Also, we were able to pull down the ESAT-6:β2M complex from the enriched ER fraction of HEK-293 cells transfected with pEGFP-C1-esat-6 but not from cells transfected with the vector alone, indicating that ESAT-6:β2M complex was present inside the ER (Fig. 1D). Thus, once translocated to the ER, ESAT-6:CFP-10 can interact and sequester β2M and thereby reduce the availability of free β2M to form complex with HLA class I molecules. In such situations, the surface levels of both β2M and MHC molecules are likely to be decreased. Expectedly, when THP-1 macrophages were incubated with recombinant ESAT-6:CFP-10 protein, both surface β2M and MHC levels were decreased. The MTT viability assay indicated that reduction in surface β2M levels was not due to cell cytotoxicity of the ESAT-6:CFP-10 complex. ESAT-6:CFP-10 also did not affect intracellular β2M at the protein and mRNA level. The surface expression of other cell surface markers like Mac-1, TLR4, MHC-II and CD14 molecules were found to remain unchanged in ESAT-6:CFP-10 treated cells which indicated that the reduction of surface β2M levels was not due to general trafficking defects but possibly due to physical sequestration of β2M by ESAT-6 inside the ER. These data together suggest that ESAT-6 is not only able to enter the ER but also can interact with ER-resident β2M. Interestingly, the

sandwich ELISA indicate that the ESAT-6:β2M complex can also exist in pathophysiological settings like pleural fluid of individuals suffering from pleural TB.

c. ESAT-6:CFP-10 treatment affects expression of HLA class I molecules:

It is known that β2M molecules form a tri-molecular complex with newly synthesized HLA molecules and antigenic peptide which is transported to the surface to present the peptide to its cognate T cell receptor (TCR). We next checked whether less amount of MHC-1:β2M pool is available in cells treated with ESAT-6:CFP-10. A pull down assay with W6/32 (a monoclonal Ab that recognizes a conformation specific epitope on the HLA class I molecules only when associated with β2M), yielded lesser amount of β2M complexed with MHC-1 in ESAT-6:CFP-10-treated cells, indicating that less amount of β2M was complexed with class I molecules in ESAT-6:CFP-10-treated cells compared to untreated as well as those treated with ESAT-6ΔC:CFP-10 (Fig. 1E). When the surface expression of β2M-complexed HLA class I molecules was measured with the help of W6/32, expectedly we observed a significant decrease in staining in ESAT-6:CFP-10-treated but not in ESAT-6ΔC:CFP-10-treated cells (Fig. 1F). Using HC-10 monoclonal Ab that detects only the free HLA-I heavy chain molecules not complexed with β2M, we observed that the levels of β2M-free HLA class I molecules were increased on the surface (Fig. 1Gi) as well as intracellularly (Fig. 1Gii) after treatment with ESAT-6:CFP-10. These data together indicate that ESAT-6:CFP-10 can sequester free β2M in ER resulting in reduced MHC-I:β2M complex formation and consequently increasing the levels of free HLA class I heavy chain molecules.

d. ESAT-6:CFP-10 inhibits MHC-I presentation of SIINFEKL peptide derived from cytoplasmic and soluble ovalbumin:

When thioglycolate-elicited mouse peritoneal macrophages from C57BL/6 mice (H-2K^b) were cytosolically loaded with native ovalbumin (OVA) and the levels of ovalbumin-derived SIINFEKL peptide (OVA257–264) presented by MHC-I-β2M complex was examined by flow cytometry in the absence or presence of ESAT-6:CFP-10 or ESAT-6ΔC:CFP-10 complex, the surface levels of SIINFEKL in complex MHC-I-β2M complex were found to be significantly reduced in cells treated with ESAT-6:CFP-10 protein when compared with OVA-pulsed cells treated with either medium or ESAT-6ΔC:CFP-10 protein indicating that ESAT-6 reduces class I-mediated antigen presentation (Fig. 1H). We also confirmed the staining experiment with an IL-2 assay as a read out for T cell function (Fig. 1I). The results show that the decreased SIINFEKL staining in cells treated with ESAT-6:CFP-10 actually correlates with a functional defect in presentation of OVA antigen. ESAT-6:CFP-10 also reduces cross-presentation of SIINFEKL peptide derived from the soluble ovalbumin on MHC-I:Δ2M complex (Fig. 1J).

Future plan

We plan to crystallize ESAT-6- β2M complex for designing of small molecule inhibitors and to test the lead molecules in animal model of tuberculosis infection.

Project 2: Studying the TLR2 signaling pathways responsible for induction of anti- and pro-inflammatory responses in tuberculosis.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

The toll-like receptors (TLRs) belong to the family of receptors known as Pattern Recognition Receptors or PRRs, which recognize PAMPs (Pathogen Associated Molecular Pattern). TLRs are shown to be involved in invoking both pro- as well as anti-inflammatory responses. But how these responses are activated and regulated during mycobacterial infection is not well understood. Previous work carried out by us revealed that two PPE proteins of Mycobacterium tuberculosis, PPE17 and PPE18, bind to TLR2. While interaction of PPE17 with TLR2 LRR domain 16~20 induces TNF-α and pro-inflammatory-type responses, binding of PPE18 with TLR2 LRR domain 11~15, results in generation of IL-10 and anti-inflammatory immune responses (Nair et al. J. Immunol. 2011; Bhat et al. J. Biol. Chem. 2012). In the present study, we initiated experiments to understand the mechanism of this differential signalling triggered by PPE17 and PPE18 downstream of TLR2.

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

After the engagement of TLR2 with its ligand, adaptor molecules such as MyD88, IRAK-1, IRAK-4, and TRAF-6 are recruited at the cytosolic domain or TIR domain of the receptor.

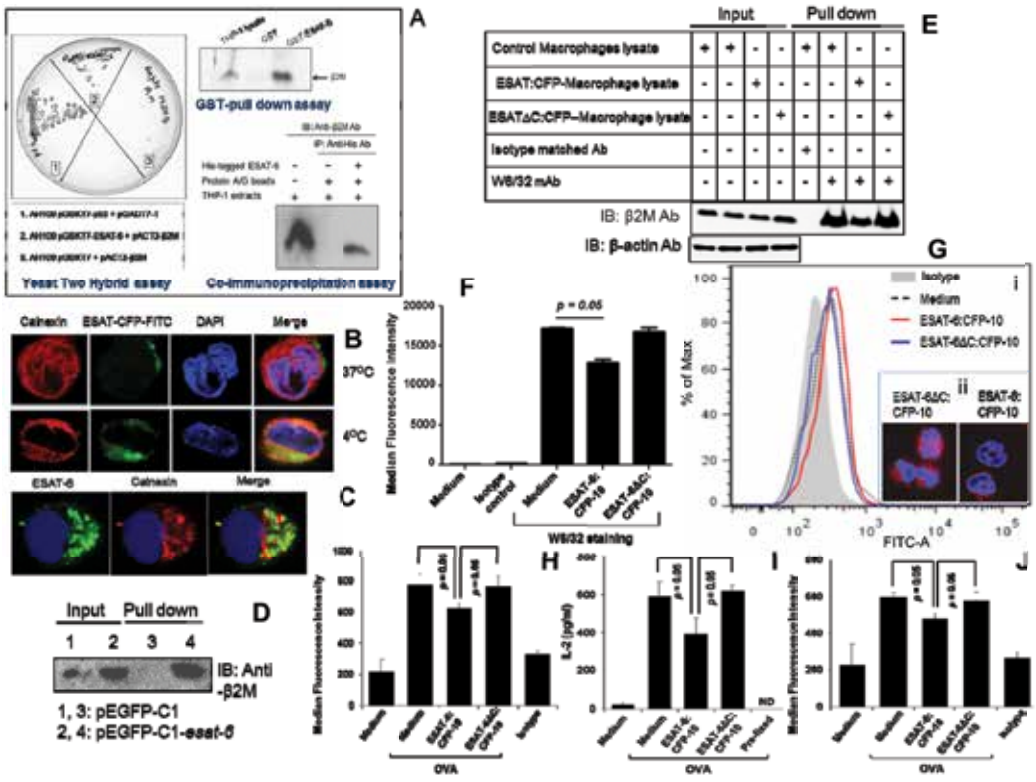

Figure 1. ESAT-6/ESAT-6:CFP-10 protein interacts with β2M and inhibits class I antigen presentation function of macrophages. Yeast two-hybrid assay followed by GST pull-down and co-immunoprecipitation assay indicates a positive interaction between ESAT-6 and β2M (A). The confocal microscopy suggests that exogenously added ESAT-6:CFP-10 (B) as well as intracellularly expressed ESAT-6 (C) gain access into calnexin-positive ER and interacts with β2M present in the ER as ESAT-6:β2M complex was pulled-down from the enriched ER fraction of HEK-293 cells transfected with pEGFP-C1-esat-6 but not from cells transfected with the vector alone (D). Thus less amount of β2M was complexed with HLA class I molecules in ESAT-6:CFP-10-treated macrophages compared to untreated as well as those treated with ESAT-6ΔC:CFP-10 as revealed by co-immunoprecipitation assay using W6/32 (a monoclonal antibody that recognizes a conformation specific epitope on the HLA class I molecules only when associated with β2M) (E) resulting in reduced HLA class I-β2M complex formation on THP-1 macrophages (F) and consequently increasing the levels of levels of β2M-free HLA class I molecules on the macrophage surface (Gi) as well as intracellularly (Gii). Exogenously added soluble ESAT-6:CFP-10 complex was found to reduce the amount of SIINFEKL peptide presented by hypertonically loaded cytoplasmic ovalbumin (H) as well as cross-presented ovalbumin (I) affecting the class I antigen presentation to CD8 T cells (J).

We then hypothesized that whether there was any difference in recruitment of these adaptor molecules (MyD88, IRAKs etc.) as well as activation of downstream kinases such as ERK 1/2 and p38 MAPK. We found that upon treatment with recombinant PPE17 (rPPE17), there was more enrichment of MyD88 when pulled down with IRAK-1 as compared to rPPE18. Also there was a change in the localization of IRAK3 (an inactive kinase and a member of the IRAK family) when macrophages were treated with these two proteins. It was observed that in macrophages treated with either medium (control group) or rPPE18, IRAK3 was spread throughout the cell, and present both in the cytoplasm and in the nucleus. However, in macrophages treated with rPPE17, IRAK3 had redistributed, and localized predominantly in the cytoplasm than in the nucleus. PPE17 when presented in the context of the whole bacillus is also able to ship IRAK3 from the nucleus to the cytosol corroborating the in *vitro* observed data using purified protein. The rPPE17 activated ERK 1/2, but rPPE18 induced p38 MAPK activation.

The polypeptide sequence indicates that IRAK3 has a nuclear export signal. The Leptomycin B (a known inhibitor of nuclear export) prevented export of IRAK3 from the nucleus to the cytosol in PPE17-treated macrophages but did not affect intracellular distribution of IRAK3 in PPE18-treated macrophages. These results indicate that the redistribution of IRAK3 by PPE17 requires nuclear export machinery. Also, the activation of ERK 1/2 and TNF-α by PPE17 was inhibited by Leptomycin B indicating that the export of IRAK3 into the cytoplasm was necessary for inhibition of p38 MAPK activity with simultaneous activation of ERK 1/2 and TNF-α. All these data together suggest that two separate pathways are triggered by PPE17 and PPE18 resulting in subsequent induction of pro- and anti-inflammatory responses downstream of TLR2.

Future plan

We intend to study in detail the TLR2 signaling cascades responsible for regulation of anti- and pro-inflammatory responses in tuberculosis and accordingly designing of small molecule inhibitors to specifically inhibit anti-inflammatory signaling known to favor *M. tuberculosis* infection.

Publications

1. Abraham PR, Latha GS, Valluri VL and Mukhopadhyay S (2014). *Mycobacterium tuberculosis* PPE protein Rv0256c induces strong B cell response in tuberculosis patients. ***Infection, Genetics and Evolution*** 22: 244-249.

2. Sreejit G, Ahmed A, Parveen N, Jha V, Valluri VL, Ghosh S and Mukhopadhyay S (2014). The ESAT-6 protein of *Mycobacterium tuberculosis* interacts with beta-2-microglobulin (ß2M) affecting antigen presentation function of macrophage. ***PLoS Pathogens*** 10: e1004446; doi: 10.1371/journal.ppat.1004446.

3. Bhat KH and Mukhopadhyay S. Macrophage takeover and the host-bacilli interplay during tuberculosis. ***Future Microbiology*** (In press).

LABORATORY OF MOLECULAR GENETICS

(Explanatory note: The Laboratory of Molecular Genetics, which also includes the Centre of Excellence (CoE) in Silkmoth Genetics and Genomics, was headed by CDFD's faculty member Dr. J Nagaraju who unfortunately passed away in December 2012. Subsequently, the activities of his erstwhile group have been continued by Dr K P Arun Kumar and Dr V V Satyavathi with their respective colleagues, whose individual reports are given below. The Director of CDFD is the designated coordinator of the CoE).

A. Report of Dr KP Arun Kumar's group

Faculty	KP Arun Kumar	Scientist
PhD Students	Asha Minz	Senior Research Fellow
	S Suresh Kumar	Senior Research Fellow
	G Gopinath	Senior Research Fellow
Other Members	S Annapurna Bhavani	Technical Officer
	R Lakshmi Vaishna	Technical Assistant
	CVE Rajendra	Research Associate
	Srikeerthana K	Research Associate (Till Dec. 2014)
	Sasi Bhushan S	Research Associate (Since Oct. 2014)
	Kushal Ravindra Kekan	Project Assistant
	Adarsh K Gupta	Project Assistant (Till Jul. 2014)
	Saikat Chakraborty	Project-Junior Research Fellow
	T Vidya	Project-Junior Research Fellow
	Srikakolapu M CH Shekhar	Project-Junior Research Fellow

Objectives

1. Studies on silkworm sex chromosome dosage compensation through large-scale transcriptome sequence analysis.
2. The evolutionary dynamics of *B. mori* Z chromosome in relation to autosomes and sex chromosomes of other animal species.

The progress made in the projects related to sex chromosome dosage compensation in B. mori and evolutionary dynamics of sex chromosome is reported here.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

- Comparison of gonad-specific genes reveals obvious sexual dimorphism: The Z chromosome is defeminized in silkworm.
- Characterization of antiviral and antibacterial activity of *Bombyx mori* seroin proteins.
- bmnpv-miR-3 facilitates BmNPV infection by modulating the expression of viral P6.9 and other late genes in *Bombyx mori.*

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

Objective 1: Studies on silkworm sex chromosome dosage compensation through large-scale transcriptome sequence analysis.

Gene regulation by sex chromosome dosage compensation (DC) prevents the detrimental effects of gene dose differences between sexes due to uneven number of sex chromosomes. Several species have been studied for the occurrence of this biological phenomenon and diverse epigenetic DC mechanisms have been discovered in several male heterogametic systems (XX/XY) as dissimilar as flies, mammals, worms and much progress has been made to understand their evolutionary basis. Gene dose

difference between the sexes is often regulated by the mechanism of DC to normalize the expression of homogametic and heterogametic sex chromosomes to that of autosomes. Studies on ZW systems showed a general lack of DC in birds and reptiles, complete and incomplete DC in the moth species *Manduca sexta* and *Plodia interpunctella* respectively. However, research on the domesticated silkworm, *Bombyx mori* has yielded contradicting results thus far; showing no compensation in the previous studies and a possible existence in a recent report. These observations come from analyses ranging from studies on a confined set of genes to global gene expression studies using microarray.

In this study, we have generated whole transcriptome shotgun sequencing (RNA-seq) data of three early-stage sexed embryos and fifth instar larval heads to show that, Z chromosome is indeed dosage compensated in *Bombyx.* RNA-seq allows addressing DC on a genome-wide scale. To determine whether chromosome-wide Z linked genes in *B. mori* are dosage compensated, we measured mRNA levels of Z linked genes between sexes at different embryonic stages and larval head through RNA-seq data. Our analysis suggests that the overall expression of Z chromosome is lesser than the autosomes and we support the hypothesis of suppression mediated dosage compensation in *B. mori.* The DC mechanism is not established in the two early stages (78h and 96h) but occurs from the later stage (120h) (Fig. 1). We also analyzed the link between expression of *masc* gene and DC to see if any connection indeed exists. As the embryo ages and after the up-regulation of *masc* gene, Z linked genes in males show a lower expression compensating the dosage as those of females. We speculate that DC emerges after 96h in male silkworm. In the embryo samples, 96h was considered as a crucial developmental stage, at which the sex determination and differentiation

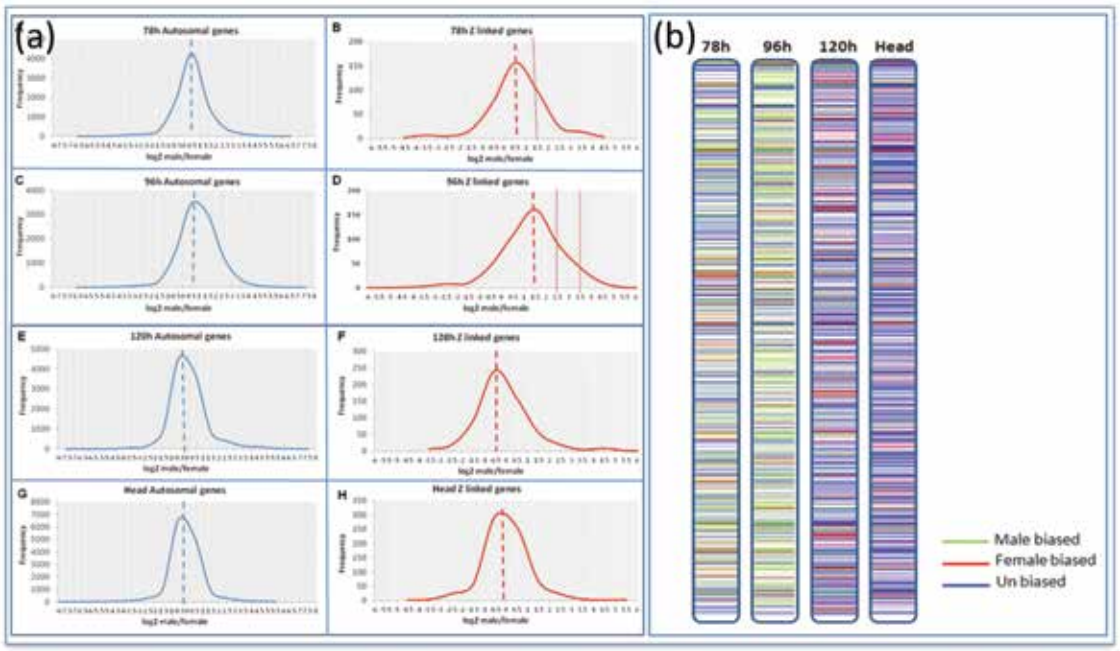

Figure 1: **(a)** The autosomal and Z linked gene expression ratio distribution plots obtained from analyzing the RNA sequencing data of all the samples. These frequency peaks represent the majority of genes falling under certain log2 male/female ratio. For the 78h and 96h stage autosomal genes, this ratio is roughly centred around -0.5 (male biased expression), where as for the 120h and head it is around 0 (equal expression between males and females). **(b)** Schematic representation of Z chromosome biased expression based on fold change. Genes with M/F fold change > 2, < -2 and 2 to -2 are considered male biased (represented in green), female biased (red) and the unbiased (blue) respectively. The percentage of male biased genes decrease from dosage uncompensated early stages (78h, 96h) to the 120h where there is a increase in the percentage of unbiased genes, a pattern that is also found in the dosage compensated head sample, suggesting the establishment of DC in the later embryonic stage of 120h.

are most widely tuned in the embryos, evident by high male biased Z linked gene expression (Fig. 1). Comprehensive analysis of gene expression in different stages reveals that the onset of DC occurs at about 96h, which probably coincides with the initiation of sex specific splicing of sex determining gene *doublesex,* and prevails throughout. Analyses of head RNA-seq data confirms the existence of complete sex chromosomal DC in Bombyx.

To our knowledge, this is the first report verifying the existence of DC in *B. mori* using NGS data, by analyzing the expression pattern of Z linked genes at different stages of sexed embryos and the sexually differentiated head. We conclude that Bombyx Z chromosome is dosage compensated and its expression is just over half to that of the autosomal expression, with males showing a little higher expression than females. The observed DC is possibly because of the reduced expression of genes on Z chromosome in males

Objective 2: The evolutionary dynamics of *B. mori* Z chromosome in relation to autosomes and sex chromosomes of other animal species.

The unique properties of sex chromosomes are predicted to have significant effects on the evolution of sex-linked genes, which has led to numerous studies of patterns of evolution on X chromosomes relative to autosomes in several taxa, as well as limited studies of the Z chromosome in vertebrates. Most notably, the hemizygosity of sex chromosomes in the heterogametic sex significantly affects rates and patterns of evolution in ways that can shed light on the relative importance of drift and selection.

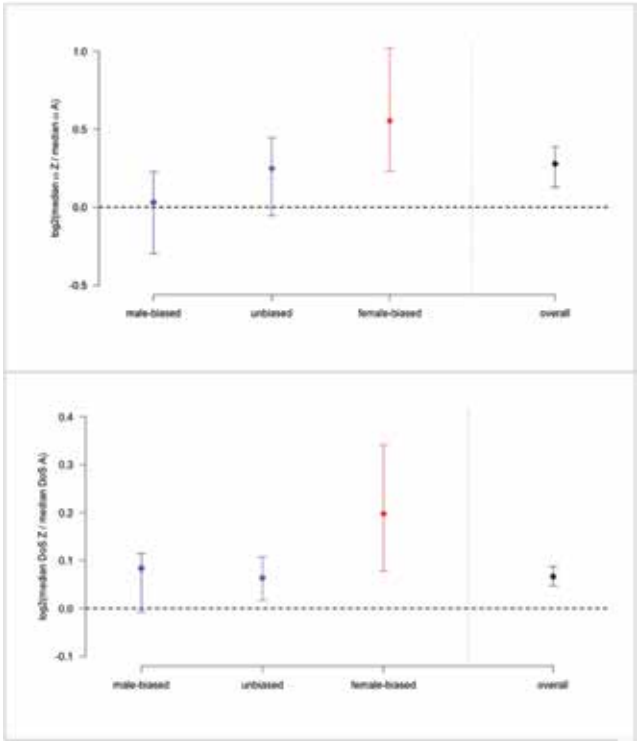

Figure 2. Faster-Z effect in male-biased, unbiased, and female-biased genes. (Top) The faster-Z effect is Z:A ratio of median ω, on a log2 scale, corrected for differences in alignment coverage using a weighted bootstrap. Error bars represent 95% confidence intervals from the weighted bootstrap. The value for female-biased genes is significantly greater than the value for male biased genes based on a permutation test. (Bottom) The faster-Z effect is Z:A ratio of median scaled DoS, on a log2 scale, weighted by the DoS.weight parameter using a weighted bootstrap. Error bars represent 95% confidence intervals from the weighted bootstrap. The value for female-biased genes is significantly greater than the value for male-biased genes based on a permutation test.

Genes linked to X or Z chromosomes, which are hemizygous in the heterogametic sex, are predicted to evolve at different rates than those on autosomes. This "faster-X effect" can arise either as a consequence of hemizygosity, which leads to more efficient selection for recessive beneficial mutations in the heterogametic sex, or as a consequence of reduced effective population size of the hemizygous chromosome, which leads to increased fixation of weakly deleterious mutations due to random genetic drift. Empirical results to date have suggested that, while the overall pattern across taxa is complicated, in general systems with male-heterogamy show a faster-X effect primarily attributable to more efficient selection while the only female heterogamy taxon studied to date (birds) shows a faster-Z effect primarily attributable to increased drift.

In order to test the generality of the faster-Z pattern seen in birds, we sequenced the genome of the lepidopteran insect *Bombyx huttoni,* a close outgroup of the domesticated silkmoth *B. mori,* and use this genome sequence to analyze faster-Z evolution in silkmoths (Lepidoptera). We first show that our *B. huttoni* assembly provides more than adequate coverage for molecular evolutionary studies. Comparing both *dN/dS* ratios and estimates of selection derived from published polymorphism data across expression classes (male-biased, female-biased, and unbiased) indicates a strong faster-Z effect for female-biased genes, an intermediate faster-Z effect for unbiased genes, and no faster-Z effect for male-biased genes. This contrasts with the pattern observed in birds (equal faster-Z effect across all expression classes) and suggests that more efficient selection may be driving the faster-Z effect in silkmoths. We propose that conditions under which drift can predominate in sex chromosome evolution are not universal, even in female-heterogametic taxa (Fig. 2).

Taken together, our results suggest that female heterogamy alone may not be sufficient to explain the discrepancy observed between faster-Z evolution in vertebrates and faster-X evolution in mammals and *Drosophila.* Instead, a combination of several factors, including the ratio of effective population size of the hemizygous chromosome to autosomes and overall effective population size, likely interact to produce the patterns of sex chromosome evolution we observe across taxa. Additional studies of a more diverse array of species will help clarify the role of these forces in faster-Z and faster-X evolution.

B. Report of Dr VV Satyavathi's group

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	SV Seshagiri	APSSRDI, Hindupur

Objectives

1. Introduction of anti-baculoviral property from transgenic silkworms to commercial silkworm strains followed by limited multilocational field trials;
2. Characterization of *Bombyx mori* nucleopolyhedrosis virus (BmNPV) resistant transgenic silkworm strains;
3. Development of baculovirus resistant silkworm strains using marker assisted selection; and
4. Identification and functional characterization of novel genes involved in immune response pathways of silkmoths.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

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

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

Objective 1: Introduction of anti-baculoviral property from transgenic silkworms to commercial silkworm strains followed by limited multilocational contained trials.

Genetically engineered silkworm (*Bombyx mori*) lines resistant to Nuclear Polyhedrosis Virus (NPV) were developed using RNAi technology. The transgenic silkworm lines were initially developed under Nistari genetic background using *piggyBac* transposon-based germline transgenesis. The transgenic lines showed stable resistance against baculovirus and the antiviral property was later transferred to a high yielding, commercial strain CSR2 through repeated backcrossing. The transgenic lines are being maintained at Andhra Pradesh State Sericulture Research and Development Institute (APSSRDI), Hindupur. A work order has been made between CDFD and Biotech Consortium India Limited (BCIL) on preparation of a roadmap for seeking biosafety regulatory approval from Review Committee on Genetic Manipulation

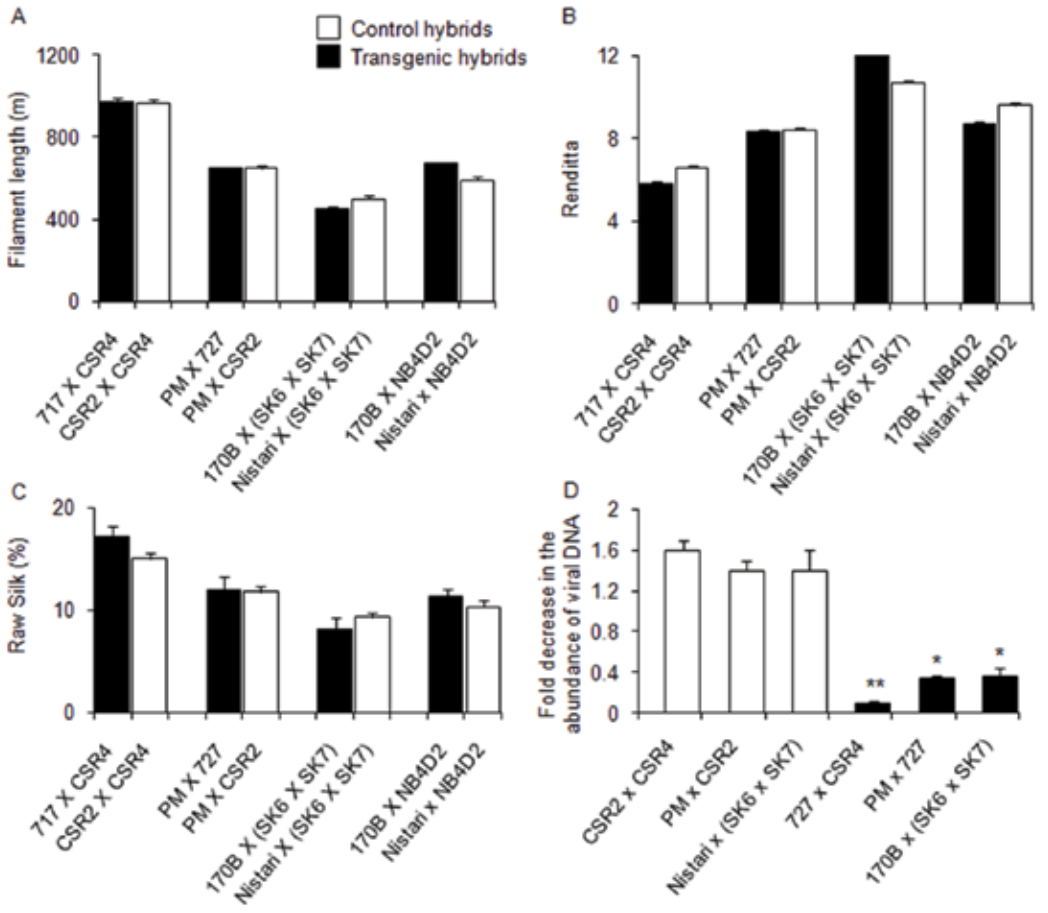

Figure 1. Characteristics of baculoviral resistant *B. mori* transgenic hybrids and their respective controls. Transgenic CSR2 lines (717, 727) and transgenic Nistari line (170B) were crossed with multivoltine (Pure Mysore - PM) and bivoltine (CSR4, NB4D2, SK6 x SK7) breeds. The silk characters shown are (A) Filament length (m), (B) Renditta, and (C) Raw silk (%). (D) Quantitative PCR analysis of *Bm*NPV using *Lef3* gene was performed to determine the viral load in the transgenic and control hybrids infected with *Bm*NPV. The results were normalized against endogenous 18S rRNA gene. Data are mean ± S.E.M (n=3). *p* values are from a Student's t test (* $p < 0.05$; ** $p < 0.01$). Bars in black and white represent transgenic and control hybrids, respectively.

(RCGM) to carry out multilocational contained trials of the transgenic silkworm.

RCGM in its 132nd meeting held on March 2014 has given formal permission to CDFD for the conduct of multilocational trials in contained facilities on genetically engineered *B. mori* lines at 4 locations i.e. three institutions of Central Silk Board i.e. CSR&TI, Mysore, CSR&TI, Berhampore, CSR&TI, Pampore, and Andhra Pradesh State Sericulture Research and Development Institute (APSSRDI), Hindupur. Subsequently, transgenic hybrid layings have been raised four times during the period under report and confined at the APSSRDI for distribution to the selected centres. Since silkworm is the first transgenic insect being considered for commercial scale production in India, a sub-committee on Genetically Engineered Insects (GEI) was set up by RCGM (serviced by BCIL) to formulate guidance to conduct and monitor confined research trials (CRTs) and to develop insect specific supplementary guidelines. Accordingly, all Standard Operating Procedures (SOPs) and recoding formats specific to silkworm on the aspects such as i) Record of transport & transport inventory list, ii) Record of storage, iii) Record of storage inspection & inventory, iv) Record of brushing, v) Record of rearing, vi) Record of harvest/termination, vii) Record of post-rearing activities, and viii) Record of corrective action have been prepared to provide guidance for conducting contained trials. The proposal of CDFD for funds for the conduct of the trials was recommended by the Biotechnology Industry Research Assistance Council (BIRAC) and awaiting for the release of grants.

Objective 2: Characterization of *Bombyx mori* nucleopolyhedrosis virus (BmNPV) resistant transgenic silkworm strains.

For testing at multiple locations, hybrids were generated by crossing Nistari and CSR2 transgenic lines with various commercial local silkworm breeds. The performance of the hybrids was tested against baculovirus infection and the data on their survival was recorded in each season. All the transgenic hybrids showed increased resistance over their nontransgenic control hybrids upon *Bm*NPV infection. Overall, hybrids resulted from the cross involving bivoltine lines showed higher survival rate as compared to polyvoltine transgenic lines. The transgenic and control hybrids were also studied for the silk yield characters. The silk characters, namely, silk filament length, renditta, and raw silk were measured using standard procedures. The silk properties were compared among transgenic hybrids and their controls and the data is presented in Figures 1A-C. Among various hybrids, 717 x CSR4 showed higher filament length, raw silk percentage and lowest value for renditta. The baculovirus accumulation in the transgenic lines was also measured as the concentration of OBs in the hemolymph of larvae after infection. The viral load, as determined by the viral transcript level using qPCR, reduced approximately 6 folds in PM x 727 and 170B x (SK6 x SK7) hybrids and 16 folds in 727 x CSR4 hybrid as compared to their respective controls (Fig. 1D). These results confirm that the virus load is significantly reduced in the transgenic hybrids in comparison to control lines. The transgenic hybrids which indicated their success in inhibiting viral proliferation under laboratory trials need to be further tested at multilocational contained conditions.

Objective 3: Development of baculovirus resistant silkworm strains using marker assisted selection.

We investigated variations in the gene expression of *B. mori* following infection with BmNPV by generating a second generation Illumina sequencing libraries for the midgut and fat body tissues from infected and control larvae of resistant (SBNP1) and susceptible (CSR2) strains. A large number of genes were found to be differentially expressed between the resistant and susceptible silkworm strains. We have also identified several microsatellite markers polymorphic between CSR2 and SBNP1 strains.

In the transcriptome, various serpins were found to express differentially upon BmNPV challenge. Serpins are a superfamily of proteins that perform a broad spectrum of different biological functions. Time course expression analysis revealed high expression of *Serpin 2* in CSR2 strain in the midgut as well as fat body tissue at 72 hours post *Bm*NPV infection (Figs. 2A&B). Interestingly, expression of *Serpin* 2 did not change much in BmNPV-treated resistant strain. We constructed a dsRNA specific for the cDNA sequence of *Serpin 2* and used in a systemic RNAi treatment to reduce mRNA levels of *Serpin* 2. Knockdown of *Serpin* 2 resulted in an increase in viral load in hemolymph of both CSR2 and SBNP1 strains (Fig.2C), indicating a role for *Serpin 2* in antiviral immunity. The multiple sequence alignment of

Serpin 2 with its closet homologues showed that several amino acid residues of serpins were conserved across different species (Fig.2D). Homology model was constructed based on crystal structure coordinates of 1K9O and 3FGQ at a resolution of 2.21Å and the protein structure was visualized using CCP4MG tool. The highly conserved region at reactive centre loop (RCL) of *Serpin 2* was predicted as GAEA (324-327) and FHADRP (347-352) (Fig. 2D). The predicted P1 residue suggests that *Serpin 2* inhibits a protease with trypsin specificity and may regulate trypsin/chymotrypsin like enzymes.

Future work includes identification of markers linked to baculovirus resistance, identification of polymorphism in the genes that are up/ down regulated in the midgut upon baculoviral infection, and incorporation of the identified polymorphic markers linked to baculovirus resistance to susceptible strain by recurrent backcross strategy.

Objective 4: Identification and functional characterization of novel genes involved in immune response pathways of silkmoths.

In a previous study, we identified a novel immune protein Noduler which binds specific bacterial components and hemocytes leading to nodulation response in the wild silkworm, *Antheraea mylitta.* We investigated functional connection between Noduler with various signalling pathways. We consolidated information on the nodulation response in insects and made an analogy with that of vertebrate system.

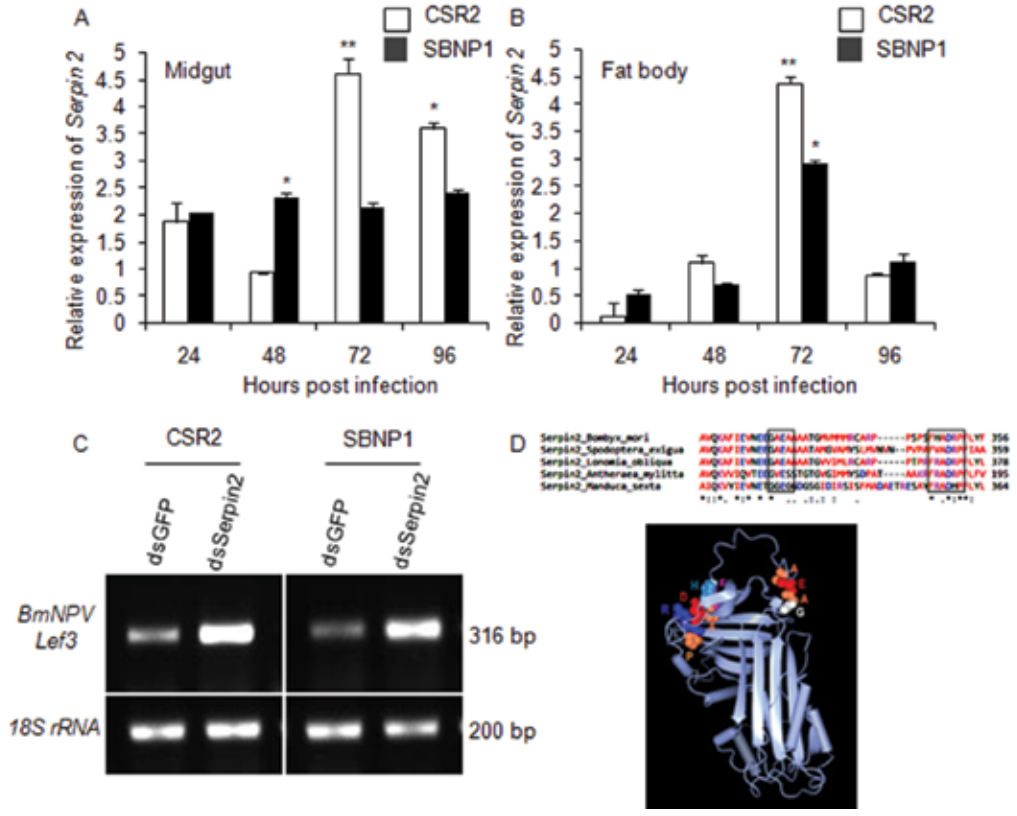

Figure 2. Characterization of *Serpin* 2 gene in *B. mori*. (A) Quantitative PCR analysis of *Serpin* 2 in midgut tissues of fifth instar larvae of CSR2 and SBNP1 strains challenged with *Bm*NPV. Samples were taken at different time points post *Bm*NPV infection and the relative expression levels of the gene were normalized against endogenous 18S rRNA. Data are mean ± S.E.M (n=3). p values are from a Student's t test (* $p < 0.05$; ** $p < 0.01$), (B) same as in (A), and the tissue used is fat body, (C) RT-PCR analysis of *BmNPV* using *Lef3* as a target gene to determine the viral load in the midguts of dsGFP and dsSerpin 2 of CSR2 and SBNP1 strains infected with BmNPV. Endogenous 18S rRNA gene was used as an internal control, and (D) Multiple sequence alignment of reactive center loop (RCL) of *Serpin* 2 homologues. The conserved residues in the RCL region are boxed (Top planel). The predicted secondary structure of *Serpin* 2 is shown in a ribbon format (grey color) and the residues in RCL region (GAEA & FHADRP) are shown as spheres (Bottom panel).

As nodule formation necessitates involvement of hemocytes, we first investigated the effect of bacterial infection on circulating hemocytes in the larval hemolymph of *A. mylitta* larvae. Upon bacterial challenge, the circulating hemocytes showed a significant increase in their number ($P < 0.05$) as compared to uninfected controls. As reported in other lepidopterans, five types of hemocytes - namely, prohemocytes, granulocytes, spherulocytes, plasmatocytes and oenocytoids - were identified in the larval hemolymph. Further analysis of hemocytes by differential staining methods, revealed an increase in the number of prohemocytes, granulocytes and plasmatocytes. Interestingly, Noduler knockdown blocked the increase in hemocyte count induced by bacterial infection. Knockdown of other immune-related genes such as *gloverin* and *prophenoloxidase* had no effect on hemocyte number, suggesting that Noduler has a specific role in hemocyte proliferation. Immunofluorescence analysis using anti-Noduler antibody revealed that Noduler is expressed by all hemocyte subtypes. We speculate that Noduler is a transmembrane protein that is synthesized in the fat body and then transported to the hemocyte surface, where it likely binds directly to pathogens. The binding of Noduler to microbial pathogens results in an interaction and efficient activation of hemocytes. This recognition event generally occurs either directly or indirectly between the secreted proteins and hemocyte receptors. Noduler possesses a reeler domain along its entire length which is highly conserved across species. The reeler domain was initially identified in the mouse reelin protein, as a secreted glycoprotein involved in the development of the central nervous system. Future work includes expression analysis and functional characterization of Noduler homologues in mammalian system, and identification of the molecular components of the signalling pathway.

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1. Arunkumar KP (2014). Book review of the Annual Review of Genetics 2013, Bonnie Bassler *et al.,* (eds) ***Current Science*** 106: 1755-1757.
2. Arunkumar KP (2014). Role of biotechnology in seri-development. ***Indian Silk*** 5: 74-76.

LABORATORY OF MOLECULAR ONCOLOGY

Genomics and molecular genetics of cancer and genetic disorders

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	Pratyusha Bala	Junior Research Fellow
	Ashmala Naz	Junior Research Fellow (Since Aug. 2014)
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Objectives

1. Identification and characterization of important deregulated genes/pathways in cancers prevalent in India; and
2. Identification and characterization of disease causing mutations in genetic disorders.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

Colorectal Cancer (CRC): 50% of early-onset rectal (but not colon) cancer samples did not harbour deregulated canonical Wnt signalling or mismatch repair inactivation though they exhibited extensive chromosomal instability (CIN). Microarray based transcriptome profiling performed on microsatellite stable samples stratified for Wnt status revealed enrichment of the Wnt/β-Catenin pathway in Wnt+ samples, as expected. A combined Gene Set Enrichment Analysis (GSEA) and Significance Analysis of Microarrays (SAM) analyses pointed to the possibility of enrichment of non-canonical Wnt pathway genes in Wnt- samples.

Tongue and oesophageal cancer: Analysis of more than one hundred oral tongue squamous cell carcinoma (SCCOT) samples revealed younger

age and *FHIT* loss to be significantly associated with p53 inactivation especially in patients with no history of tobacco use. P53 inactivation was the only significant prognosticator of SCCOT survival in multivariate analysis. The *TP53* codon 72 Pro allele was significantly associated with SCCOT compared to healthy controls; no such association was detected in oesophageal squamous cell carcinoma (ESCC) samples. Surprisingly, *TP53* DNA binding domain mutation was significantly associated with the Pro allele in ESCC but not in SCCOT.

Genetic disorders: Analysis of twenty five Phenyl ketonuria (PKU) families revealed a significantly low proportion of missense mutations as compared to other world populations. In contrast, a significantly higher proportion of splice, insertion-deletion and nonsense mutations point to a unique *Phenylalanine hydroxylase (PAH)* mutation profile in India. In another study, we identified a significantly higher proportion of autosomal recessive form of hypohidrotic ectodermal dysplasia (HED) due to a founder ectodysplasin A receptor (*EDAR*) mutation unlike other world populations where the X-linked form predominates.

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

CRC: We performed single sample GSEA for identification of pathways differentially

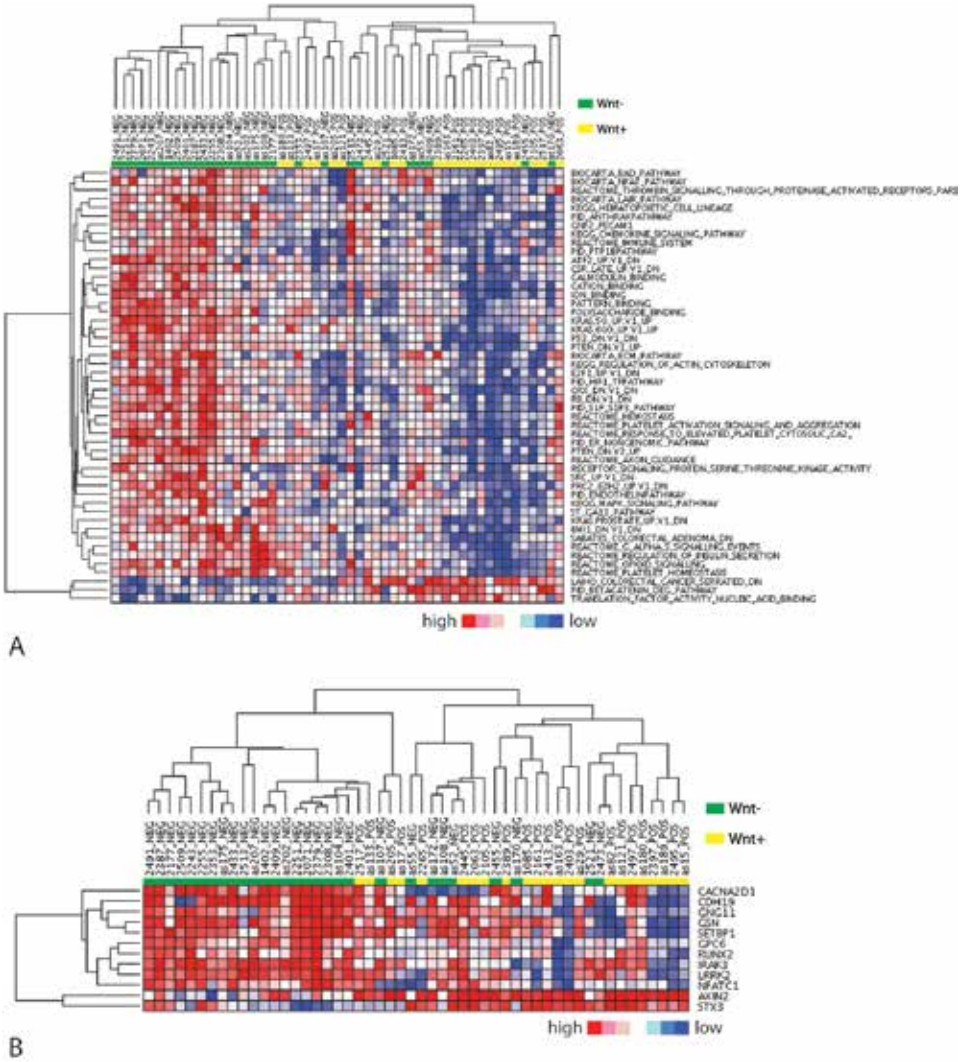

Figure 1. Identification of the first Wnt- gene signature in microsatellite stable CRC. Cellular pathways and genes that are differentially enriched (A) or expressed (B) in Wnt- rectal cancer samples, respectively. Each column represents one sample and each row represents one pathway (A) or gene (B).

expressed between Wnt+ and Wnt- early onset sporadic rectal cancer (EOSRC) samples followed by 'supervised' analysis (comparative marker selection). The pathways were able to faithfully distinguish Wnt+ and Wnt- samples based on 'unsupervised' clustering as expected (Fig. 1A). SAM and leading edge analyses on genes constituting the 50 pathways revealed differentially expressed genes that were further validated using quantitative reverse transcription

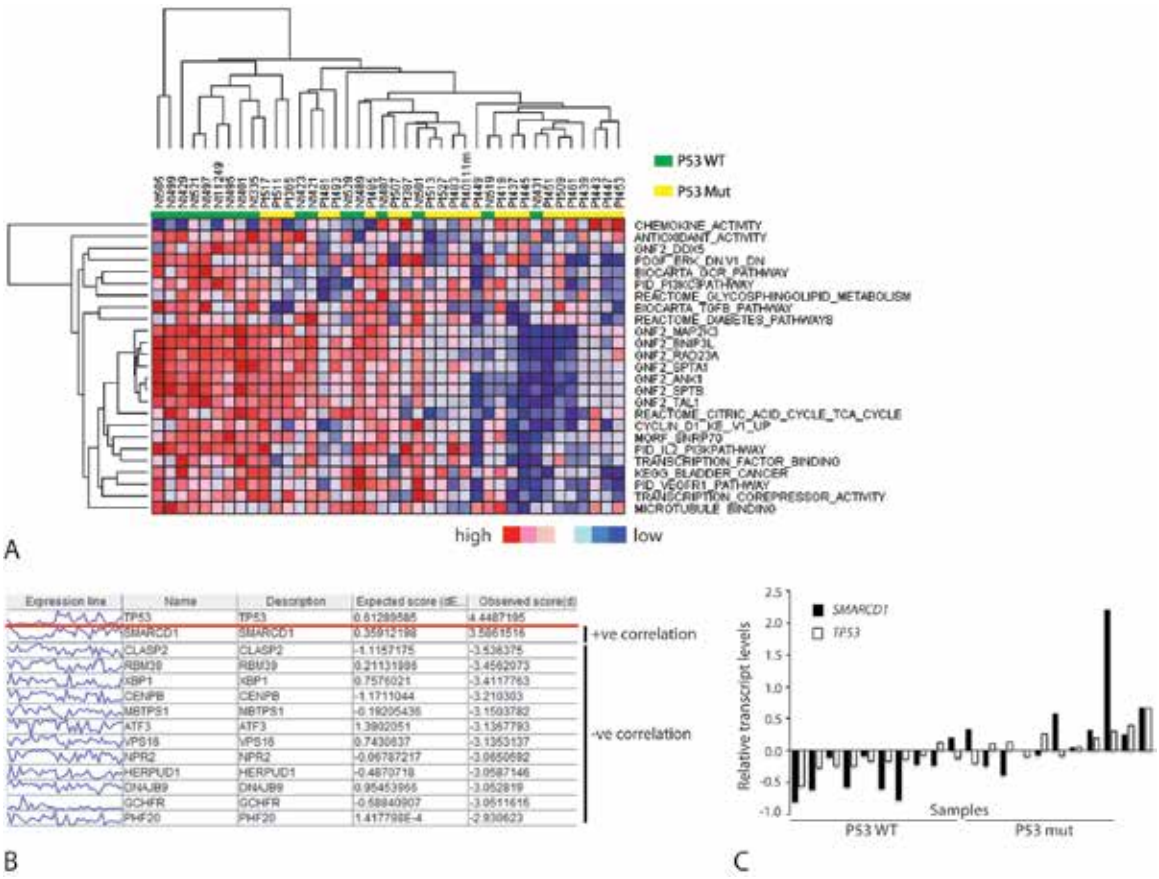

Figure 2. SMARCD1 appears to be a novel transcription target of *TP53*. Panel A shows pathways that are differentially enriched in *TP53* WT (compared to mut) SCCOT samples. Each column represents one sample and each row represents one pathway. Panel B shows genes that are differentially expressed with respect to *TP53* expression status (samples exhibiting higher *TP53* expression are expected to harbour a mutant *TP53*); only *SMARCD1* exhibits positive correlation with *TP53*. Panel C shows Q-RT-PCR based validation of *TP53* and *SMARCD1* expression (mean-centred across all samples); *TP53* ($p<0.01$) and SMARCD1 ($p<0.05$) transcript levels were significantly different between *TP53* WT and mut samples, as determined by Mann-Whitney U test.

PCR (Q-RT-PCR) in independent samples. Twelve validated genes were able to differentiate Wnt+ and Wnt- samples in unsupervised analysis (Fig. 1B). This is the first Wnt- gene signature identified in CRC.

Tongue cancer: We evaluated DNA copy number alterations and transcriptome profile of SCCOT samples stratified by p53 status. Surprisingly, both p53 wild type (WT) and mutant (mut) samples exhibited comparable extent of CIN. Transcriptome profiles were analysed to determine differentially expressed pathways between p53 mut (twenty three) and WT (seventeen) samples (Fig. 2A), as described above for CRC. At 0% false discovery rate (FDR), SAM yielded only one differentially expressed gene namely *TP53* itself thus validating our approach. Interestingly, an FDR of <8% revealed fourteen genes including previously characterized *TP53* targets but only *SMARCD1* exhibited

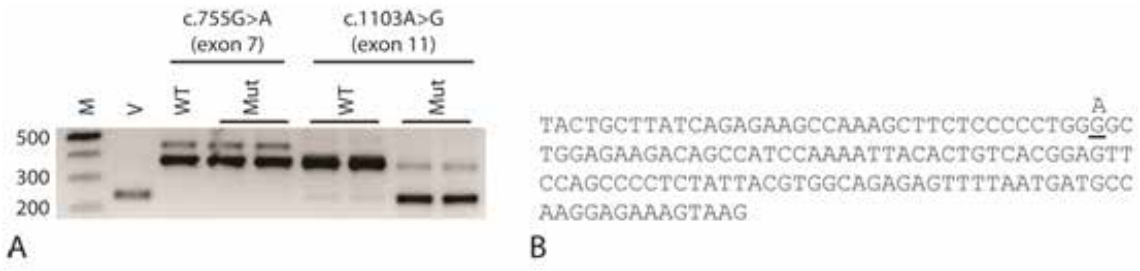

Figure 3. The PAH c.1103A>G mutation disrupts exon 11 splicing. Panel A, Agarose gel analysis of splicing assays performed with pCAS minigene constructs generated for the wild type (WT) and mutant (Mut) *PAH* exon 11. Result for another mutation (c.755G>A) is shown for comparison. M, 100bp DNA ladder. Panel B, complete nucleotide sequence of *PAH* exon 11; the mutant base is underlined and the alternate (wild type) base is shown on top.

positive correlation with *TP53* expression (Fig. 2B). Differential expression of *TP53* and SMARCD1 were confirmed using Q-RT-PCR in *TP53* WT and mut samples (figure 2C) raising the interesting possibility of *SMARCD1* being perhaps transcriptionally activated by mutant *TP53* as shown earlier for other genes. This hypothesis is currently being tested.

Genetic disorders: We evaluated ten (five missense, four non-sense and one silent variant) *PAH* mutations using splicing assays in recombinant pCAS minigene constructs; only one (c.1103A>G (p.E368G); located in exon 11) exhibited a deleterious effect causing skipping of exon 11 (Fig. 3A). The mutation generates a stretch of four 'G' residues (Fig. 3B) suggested to be a signature of exonic splicing silencer elements. In another study, we extended our work on HED to a total of 48 families; mutation was detected in 40 (Ectodysplasin A1 (*EDA-A1*)) in 23 families, *EDAR* in 16 and ectodysplasin A receptor-associated death domain (*EDARADD*) in 1). We detected a novel ~23 Kb deletion in *EDA-A1* extending from *EDA-A1* IVS6+67 till beyond the downstream gene *AWAT2,* We also identified the first splice site mutation ever reported in *EDARADD* (IVS2+1G>A) resulting in autosomal recessive HED.

Future plans and directions

1. Characterization of genes/pathways driving EOSRC in the absence of canonical Wnt signalling.
2. Validation of *SMARCD1* transactivation by *TP53.*
3. Characterization of selected mutations affecting transcript stability/processing identified through screening of various genetic disorders.

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LABORATORY OF NEUROSPORA GENETICS

(1) The search for nucleus-limited genes.
(2) Why are wild-isolated Neurospora strains suppressors of meiotic silencing?

Faculty	DP Kasbekar	Haldane Chair
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Other Members	A Sheeba	Technical Officer
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	S Rekha	Technical Assistant
	Angela Sharma	Research Assistant (Since Jan. 2015)

Objectives

Project 1: The search for nucleus-limited genes

This project represents the first systematic search for nucleus-limited genes. A nucleus bearing a null allele (Δ) of a nucleus-limited gene is not complementable by the wild-type (*WT*) nuclei in a [(*WT*) + (Δ)] heterokaryon. Although no nucleus-limited gene has thus far been reported, sporadic reports in the literature suggest that such genes might exist in fungi. Our approach was to introgress translocations (*T*) from *N. crassa* into *N. tetrasperma* to create [(*T*) + (*N*)] and [(*Dp*) + (*Df*)] heterokaryon strains (for explanation of terms used see below). Ordinarily, in [(*Dp*) + (*Df*)] heterokaryons, we expect the *Df* nuclei to be rescued by the *Dp* nuclei, therefore the [*T* + *N*] and [*Dp* + *Df*] heterokaryons should have the same phenotype. However, if the *Df* were to delete a nucleus-limited gene, then it would not be complemented by the *Dp* nucleus, and the [*Dp* + *Df*] and [*T* + *N*] types should differ in phenotype.

Project 2: Why are wild-isolated Neurospora strains suppressors of meiotic silencing?

Earlier work by us showed that meiotic silencing by unpaired DNA (MSUD) is suppressed in crosses between most wild-isolated *N. crassa* strains and MSUD tester strains made in the standard laboratory Oak Ridge (OR) background. We hypothesized that sequence heterozygosity between the wild and OR genomes might cause one or more MSUD gene to become unpaired and silence itself, thus switching off the MSUD machinery. We have now constructed new MSUD testers in a genetic background derived from the wild-isolated Bichpuri-1 *a* (B) and Spurger *A* (S) strains to ask whether MSUD is seen in tester-heterozygous crosses that otherwise are isogenic for this B/S background.

Project 1. The search for nucleus-limited genes

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

A sexual cross in Neurospora involves the fusion of two haploid nuclei of opposite mating types, *mat-A* and *mat-a*. The resulting diploid zygote nucleus undergoes meiosis and a post-meiotic mitosis to produce eight haploid progeny nuclei, four apiece *mat-A* and *mat-a*. In *N. tetrasperma,* the eight nuclei are partitioned into four ascospores per ascus, each ascospore receiving a non-sister *mat A* + *mat a* pair, whereas in *N.* crassa they are partitioned into eight ascospores (4 *mat A* + 4 *mat a*). *N. tetrasperma* ascospores germinate to produce heterokaryotic mycelium that contains nuclei of both mating types, therefore it is competent to undergo a self-cross. However, by chance the mycelia can also produce some homokaryotic conidia (vegetative spores). Additionally, during ascus development five or more (upto eight) ascospores are occasionally produced, with a pair of smaller homokaryotic ascospores replacing a dikaryotic ascospore. The homokaryotic conidia or ascospores generate self-sterile single-mating-type strains that can cross with like strains of opposite mating type. The dominant *Eight-spore (E)* mutation substantially increases the frequency of >4-spored asci. *N. crassa* ascospores germinate to produce a homokaryotic mycelium in which all nuclei have the same mating type, therefore the mycelia from two different ascospores (one apiece *mat A* and *mat a*) are needed for a sexual cross.

Many chromosome translocations are known in *N. crassa.* Insertional translocations (*IT*) that

transfer a donor chromosome segment into a recipient chromosome are defined by three breakpoint junctions, viz, "A", created by the deletion on the donor chromosome, and "B" and "C" (proximal and distal), created by the insertion in the recipient chromosome. In the cross of an *IT* with a normal sequence strain (*IT* x *N*), alternate segregation generates 4 *N* + 4 *T* ascospores, whereas adjacent 1 segregation generates 4 *Dp* + 4 *Df*, that is, four ascospores with a duplication (*Dp*) of the translocated segment, and four with the complementary deficiency (*Df*). The *N, T* and *Dp* ascospores are viable, whereas *Df* ascospores are inviable. Our laboratory had defined the breakpoint junctions of several *ITs,* therefore *PCR* with junction-specific primers could be used to establish the genotype of progeny from *T* x *N*. This was used to introgress four *ITs (EB4, IBj5, UK14-1*, and *B362i*) from *N. crassa* to *N. tetrasperma.* Introgression is the transfer of genes or genomic regions from one species into another via hybridization and back-crosses. To our best knowledge this was the first introgression of translocations from one species into another.

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

(1) Following the introgressions of the four translocations, we constructed two general types of *N. tetrasperma* heterokaryons with *mat-A* and *mat-a* nuclei of different genotypes. One type being [*T* + *N*] (with one translocation nucleus and one normal sequence nucleus), and the other being [*Dp* + *Df*] (with one nucleus carrying a duplication of the translocation region and the other being deleted for the translocation region). Self-crosses of these heterokaryons again produced [*T* + *N*] and [*Dp* + *Df*] progeny. From conidia produced by the heterokaryotic mycelia we obtained self-fertile (heterokaryotic) and self-sterile (homokaryotic) derivative strains. Homokaryotic conidial derivatives of both mating types were obtained from [*T* + *N*] heterokaryons, but [*Dp* + *Df*] heterokaryons produced viable conidial homokaryons of only the mating type of the *Dp* nucleus. All four [*T* + *N*] heterokaryons, and three [*Dp* + *Df*] heterokaryons, produced both self-sterile and self-fertile conidial derivatives, but the [*Dp(B362i)* + *Df(B362i)*] heterokaryons produced only self-sterile ones. Plausibly, the *Df(B362i)* nuclei may be deleted for a nucleus-limited gene required for nuclear packaging into conidia, and whose deficit is not complemented by the neighboring *Dp(B362i)* nuclei. The work was published in *G3 Genes, Genomes, Genetics* during preparation of this report.

Our search for nucleus-limited genes also led to the serendipitous discovery of a mutation that specifically affected alternate segregation, and apparently had no effect on the adjacent 1 segregation (Fig. 1). The two segregation types are distinguishable only in *T* x *N* crosses. But why would a *T* x *N* cross be homozygous for

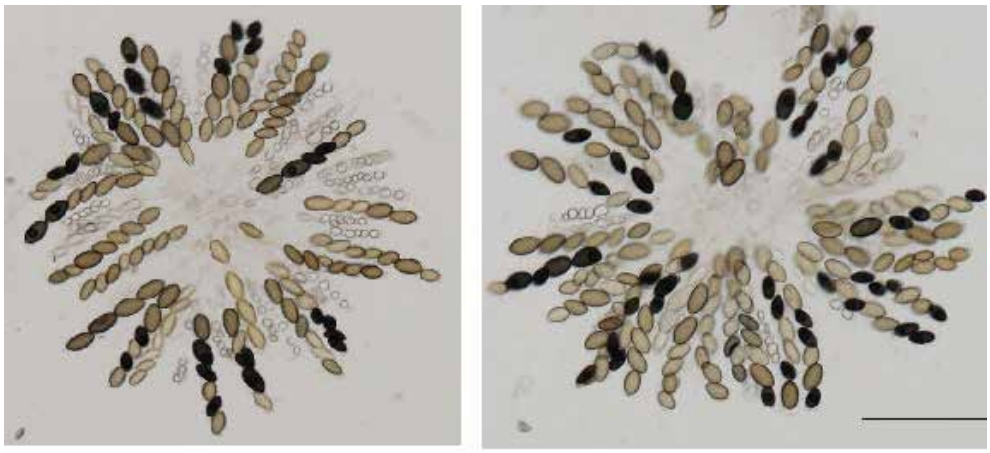

Figure 1. Asci from the cross *T(B362i)A x E a.* The *E (Eight spore)* strain is presumed to be of normal sequence. In an *E*-heterozygous cross, the asci can contain upto eight homokaryotic ascospores, instead of four heterokaryotic ones. Large ascospores are the heterokaryons, the small ones are the homokaryons. Viable ascospores turn black whereas inviable ones remain white. Note that no ascus has more than four black ascospores. This suggests that alternate segregation is blocked, whereas the adjacent 1 segregation is unaffected.

any mutation, let alone one that distinguishes between the two segregations? A 1969 paper provided the clue. It reported that two Neurospora strains, *C4,T4* and *E*, shared the same genetic background. Our group used the *C4*, *T4* strain to construct the *T* parent, and the *E* strain as the normal sequence parent. Consequently, a subset of *T* x *N* crosses apparently had become homozygous for the segregation defect.

Project 2: Why are wild-isolated Neurospora strains suppressors of meiotic silencing?

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

MSUD is an RNAi-mediated process that eliminates the transcripts of any gene that is not properly paired with its homolog in meiosis. The ::*r,* ::*Bml*^{r} and ::*mei-3* tester strains contain a copy of the *r (Round ascospores), Bml*^{r} *(β-tubulin)* or *mei-3* gene inserted ectopically in the *his-3* locus on chromosome 1. In the cross of a tester with an OR strain of opposite mating type, the ectopic copy is unpaired in meiosis and induces the synthesis of MSUD-associated small interfering RNA (masiRNA) which silences it as well as its paired native homologs. Since β-tubulin and MEI-3 protein are essential for ascus development, and the product of the *r* gene is required for the spindle shape of ascospores, the silencing results in ascus or ascospore abnormalities. Homozygous *tester A x tester a* crosses do not show MSUD, nor do crosses of the testers with the semi-dominant *Sad* and *Sms* suppressors of meiotic silencing, and the asci and ascospores develop normally. The suppressor alleles prevent the proper pairing of their wild-type homologues and induce them to autogenously silence themselves. Only eight of 80 wild-isolated strains examined silenced both *bml* and *mei-3* in crosses with the OR-derived testers and they were designated as “OR” type, four failed to silence both genes and were designated the “Sad” type, and the remaining 68 silenced *bml* but not *mei-3* and were designated the “Esm” type. Additional results suggested that MSUD persists throughout the duration of the cross with the OR type, is very fleeting in the cross with the Sad type, and lasts for an intermediate duration in crosses with the Esm type strains. We hypothesized that sequence polymorphism between the tester and wild genomes might cause one or more gene essential for MSUD to become unpaired, silence itself, and thus shorten MSUD duration. To test this idea we decided to make new testers in an isogenic *mat a* and *mat A* background derived from the Sad type wild-isolates Bichpuri-1 *a* and Spurger *A*. Our hypothesis predicts that a tester-heterozygous cross that is otherwise isogenic for the B/S background would show MSUD.

To make the testers we used the RIP mutational process to knock out the *mus-51* gene needed for non-homologous end joining (NHEJ). Consequently, in a *mus-51* mutant strain transforming DNA can integrate only by homologous recombination. This allows the use of targeted integration to create well-defined reporter strains in the B/S line.

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

The native r^{+} gene is 3.3 kb long and located on chromosome I. A 2.3 kb 3' fragment (r^{ef}) was joined to the *hph* cassette by double-joint PCR to create the 4.1 kb r^{ef}*-hph* fusion construct. Use of this construct to transform a *mus-51* mutant strain and selection of transformants on hygromycin medium resulted in the targeted replacement of nucleotides 218849 to 219010 in chromosome 7L with the 4.1 kb r^{ef}*-hph* fusion.

Transformants obtained by us in this way corresponded to the ::*r2* tester made by others in the OR background. When a strain carrying ::*r2* is crossed to an OR strain of opposite mating type, close to 100% of the ascospores are round. This indicates that ::*r2* is detected as unpaired in such crosses, and as a result, the native r^{+} gene on chromosome I is silenced. When a strain carrying ::*r2* is crossed to a strain carrying the same ::*r2*, very few round spores are produced, indicating that the ::*r2* constructs are paired in such crosses and also the native r^{+} gene on chromosome I is expressed at normal levels.

Our model predicts that if we use our new tester to make tester-heterozygous cross that is otherwise isogenic for the B/S background then we will see evidence for MSUD. These crosses are presently under construction.

Other Publications

1. Kasbekar DP (2014). Editorial. Lesser models. *Journal of Biosciences* 39: 1.
2. Kasbekar DP (2014). Sidelights. Are any fungal genes nucleus-limited? *Journal of Biosciences* 39: 341-346.
3. Kasbekar, DP (2015). What have we learned

by doing transformations in Neurospora tetrasperma? In: Genetic Transformation Systems in Fungi, Volume 2. Edited by M. A. van den Berg and K. Maruthachalam, Springer, Switzerland. Pages 47-52.

4. Kasbekar DP (2015). Editorial. Via media. ***Journal of Biosciences*** 40: 1.

LABORATORY OF PLANT-MICROBE INTERACTIONS

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

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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 role in virulence; and
4. Role of PAMP in pathogen recognition and plant defense response

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

We are trying to understand the virulence mechanisms of important *Xanthomonas* pathogens like, *Xanthomonas campestris pv. campestris* (Xcc; a pathogen of crucifers), *Xanthomonas oryzae pv. oryzae and Xanthomonas oryzae pv. oryzicola* (Xoo, Xoc; pathogens of rice). In *Xanthomonas*, cell-cell (quorum sensing) is mediated by the production and sensing of fatty acid like signaling molecule known as Diffusible Signaling Factor. We have shown that DSF in Xoo plays an impotant role in trasition of planktonic to biofilm lifestyle. Our studies have shown that DSF in closely related phytopathogens regulate virulence associated traits in a contrasting fashion. To understand the role of DSF in adaptation to different lifestyle we have characterized the role of DSF in the virulence of Xoo and Xoc. Charaterization of DSF deficient *ΔrpfF* mutant of Xoc revealed that DSF promotes in *planta* growth by regulating ferric iron uptake. We are presently studying the role of DSF in regulating virulence associated function in *Xanthomonas* oryzae pv. *oryzicola* (Xoc) and its contribution to adaptation to host environment.

To understand the dynamics of quorum sensing, we have previously constructed several biosensor strains to study quorum sensing response in individual cells in the population. We have used also an *E. coli* system to reconstitute the AHL mediated QS system to study QS in a heterologous host. Our study has indicated that bacteria exhibit non genetic phenotypic heterogeneity in social behavior and may contribute to bet hedging strategy to changing environmental condition. Overall, these results support the model that bacteria maintain QS-responsive and non-responsive subpopulations at high cell densities in a bet-hedging strategy to simultaneously perform functions that are both positively and negatively regulated by QS to improve their fitness in fluctuating environments. Our results have shown that bacteria maintain

stochastic reversible phenotypic heterogeneity during a widely conserved QS-response that is involved in coordinating multiple social behaviors

Details of progress made in the current reporting year (April 1, 2014 – March 31, 2015)

Project 1: Role of quorum sensing and heterogeneity in environmental adaptation of bacteria.

Bacteria coordinate their social behavior in a density dependent manner by production of diffusible signal molecules by a process known as quorum sensing (QS). We have shown that bacteria exhibit reversible non gebnetic heterogeneity in QS. We have proposed a model based on our studies and evolutionary theory, which predicts that maintaining phenotypic heterogeneity in performing social tasks is advantageous as it can serve as a bet-hedging survival strategy. To gain more understanding of the role of QS in adaptation to different environmental conditions, we performed co-inoculation and competition experiments using mixed population of wild type and QS deficient mutants (Fig. 1). Coinoculation studies indicate that under rich media condition, there is no significant difference in the growth rate of wild type and QS – mutants. However, in coculture, the QS- mutant exhibited significant growth advantage which indicates that cost of signal production may be disadvantageous for the wild type strain when nutrients are available in sufficient amount. In recent experiments we have observed that the wild type cells exhibit increased viability during late stationary-phase, which is generally associated with nutrient limitation, compared to the QS- mutants. In general, it appears that QS- mutants exhibit growth disadvantage at early log phase and compromised viability at late stationary phase. Our transcriptome analysis by microarray and translation assays indicate that QS promotes transition to stationary phase by slowing down the metabolism (transcription and translation), as an anticipation of stationary-phase stress. In future, we are interested to study the role of QS in stationary–phase adaptation and contribution of QS heterogeneity in this process (Fig. 2).

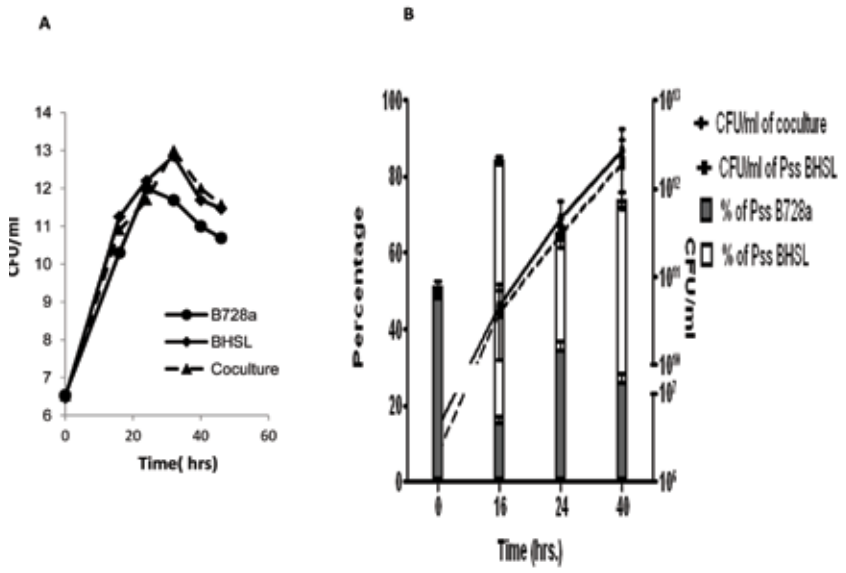

Figure 1. QS- mutants exhibit growth advantage over wild type strain in co culture... *Pss*B728a (closed circles), *PssBHSL* (closed diamond) and co culture (closed triangle) of both the strain inoculated at a ratio of 1:1 in Kings B broth. At times indicated, samples were taken and dilution plated to obtain single colonies on selection plate and CFU/ml of each respective strain and total CFU/ml in the co culture was determined. (B) Enrichment of QS cheater (*Pss*BHSL*; ahlI*). Co cultures were inoculated with a wild type strain *Pss*B728a to *Pss*BHSL (*ahlI*) mutant ratio of 1:1 in Kings B Broth. At times indicated, samples were taken and dilution plated to obtain single colonies. Shown are CFU/ml of the entire culture (closed circles) and *Pss*BHSL (*ahlI*::Km^r; open circles) as determined by plating on selective and nonselective media. Approximately, 200-300 colonies were replica patched in selective and nonselective media to determine the percent of wild type and PssBHSL in the co culture. Percent of the wild type and the *pss*BHSL are shown by closed and open bars. Values presented are mean and standard deviation (+S.D) from two experiments each consisting of two replicates.

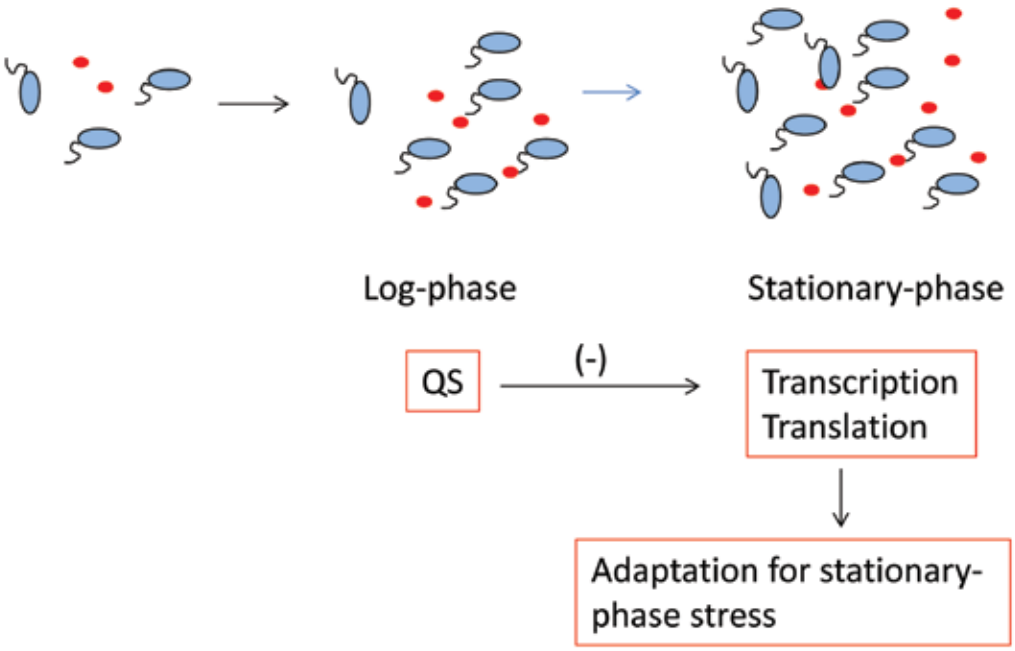

Figure 2. A proposed model for the role of QS as a signal for anticipation of stationary-phase. At high cell-density, the concentration of QS signal increases and mediate down regulation of transcription and translation, as a preparative step to counter long-term survival under stationary-phase stress. QS- mutants exhibit increased production of ribosomal proteins, protein synthesis, metabolic enzymes and poor survival under prolong stationary-phase stress.

Project 2: Role of DSF mediated cell-cell signaling in iron uptake and virulence of *Xanthomonas oryzae pv. oryzicola.*

Cell –cell communication mediated by diffusible signal factor (DSF) plays an important role in virulence of several *Xanthomonas* group of plant pathogens. In the bacterial pathogen of rice, *Xanthomonas oryzae pv. oryzicola,* DSF is required for virulence and *in planta* growth. In order to understand the role of DSF in promoting in planta growth and virulence, we have characterized the DSF deficient mutant of *X. oryzae pv. oryzicola.* Mutant analysis by expression analysis, radiolabelled iron uptake studies and growth under low-iron conditions indicated that DSF positively regulates ferric iron uptake. Further, the DSF deficient mutant of *X. oryzae pv. oryzicola* exhibited a reduced capacity to use ferric form of iron for growth under low-iron conditions. Exogenous iron supplementation in the rice leaves rescued the *in planta* growth deficiency of the DSF deficient mutant. In this work we have also shown that *Xanthomonas* produces vibrioferrin siderophore and DSF positively regulate vibrioferrin production by down regulating expression of repressor FUR (Fig. 3). This is the first report which demonstrates that the *Xanthomonas* group of plant pathogens produces siderphore vibrioferrin for the uptake of ferric form of iron. In this study we also showed that siderophore production *in planta* is very critical for growth and virulence of Xoc. Our results also indicate that requirement of iron uptake strategies to utilize either Fe^{3+} or Fe^{2+} form of iron for colonization may vary substantially among closely related members of the *Xanthomonas* group of plant pathogens. Apart from iron, we have identified novel role of DSF in regulating Type III secretion system which is required for pathogenicity of *Xanthomonas*. DSF deficient *rpfF* mutant are exhibit reduced Hypersensitive Response (HR) and reduced expression of Type III secretion components and effectors.

In future, we want to study the mechanism of DSF sensing which controls iron uptake and regulatory mechanisms, which are involved in DSF regulated traits such as Type III secretion, attachment and biofilm formation.

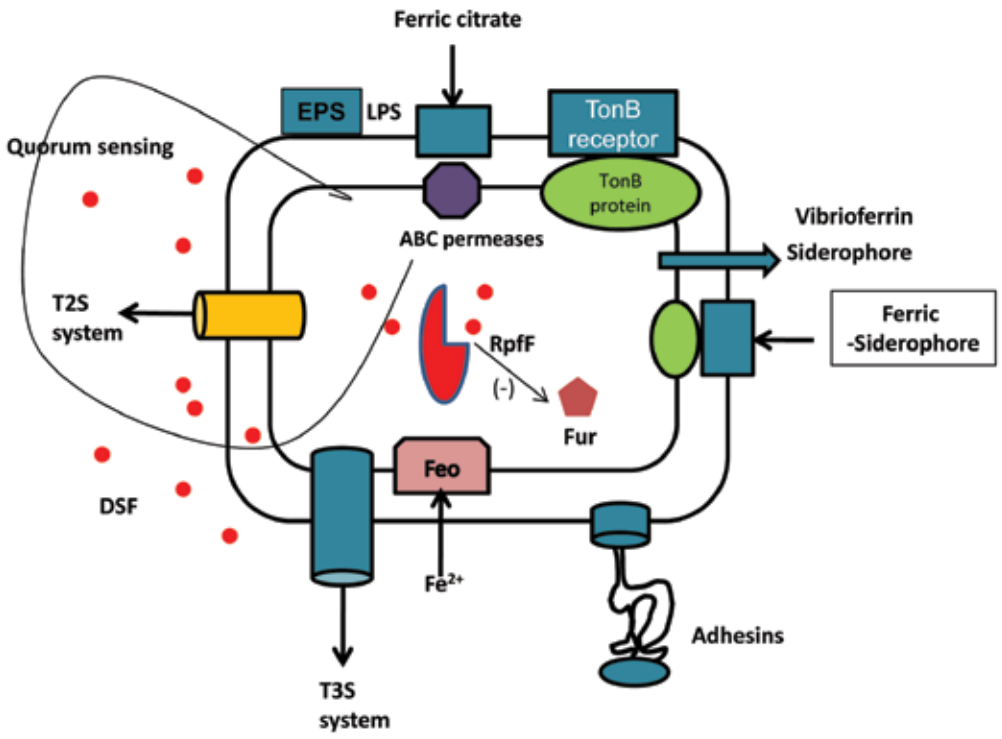

Figure 3. Diffusible signal factor (DSF) mediated quorum sensing in the rice pathogen *Xanthomonas oryzae pv. oryzicola* (Xoc) regulate ferric iron uptake and diverse virulence associated factors to promote pathogenesis. DSF synthase RpfF produces DSF in a density dependent fashion and up regulate vibrioferrin siderophore biosynthesis and uptake under low –iron conditions. DSF negatively regulate *fur* expression, which is a suppressor of iron uptake vibrioferrin biosynthetic and uptake genes. Apart from siderophore production, DSF positively regulate production of ferric-citrate transporter which transports ferric-citrate complex. Siderophore vibrioferrin is produced *in planta* and is required for virulence of Xoc.

Publications

1. *Lindow S, Newman K, Chatterjee S, Baccari C, Lavarone AT and Ionescu M (2014). Production of Xylella fastidiosa diffusible signal factor in transgenic grape causes pathogen confusion and reduction in severity of Pierce's disease. ***Molecular Plant Microbe Interactions*** 27: 244-254.
2. Pradhan BB and Chatterjee S (2014). Reversible non-genetic phenotypic heterogeneity in bacterial quorum sensing. ***Molecular Microbiology*** 92: 557-569.
3. Sundaram RM, Chatterjee S, Oliva R, Laha GS, Cruz CV, Leach JE and Sonti R (2014). Update on bacterial blight of rice: fourth international conference on bacterial blight. ***Rice*** 7: 12-14.
4. Rai R, Javvadi S and Chatterjee S. Cell–cell signalling promotes ferric iron uptake in *Xanthomonas* oryzae pv. oryzicola that contribute to its virulence and growth inside rice. ***Molecular Microbiology.*** doi: 10.1111/mmi.12965 (In press).

*Work done elsewhere

LABORATORY OF TRANSCRIPTION

Mechanism of transcription termination and antitermination in Escherichia coli

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	V Vishalini	Senior Research Fellow
	Gairika Ghosh	Senior Research Fellow
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	Amitabh Ranjan	Research Associate (Till Aug. 2014)
	B Sudha Kalayni	Research Associate
	Shweta Singh	Research Associate (Since Sep. 2014)
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	Sonia Agrawal	Project-Junior Research Fellow (Since Feb. 2015)
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	V Nagaraja	IISc., Bangalore
	Jayanta Mukhopadhyay	Bose Institute, Kolkata
	Akira Ishihama	Hosei University, Japan

Objectives

Fundamental questions in the area of mechanism of transcription termination and antitermination processes is still not very clear and offers an exciting subject for study. In my laboratory, we have undertaken following studies. 1) Mechanism of action of transcription termination factor, Rho. 2) Molecular basis of Rho-NusG interaction. 3) Mechanism of conversion of NusA into an antiterminator by N. 4) Mechanism of action of transcription antitermination of Rho-dependent termination by an anti-rho factor, Psu. 5) In vivo cross-talks between Rho-dependent termination and other biological processes. 6) Designing transcription modulators using synthetic biology approaches.

Summary of work done until the beginning of this reporting year (upto March 31, 2014)

- We showed that sequence specific recognition of the *rut* site in majority of the Rho-dependent terminators can be compromised to a great extent without seriously affecting the genome-wide termination function as well as the viability of *E.coli*. These terminators function optimally only through a NusG-dependent assisted recruitment and activation of Rho (NAR, 2014).
- We made a detailed characterization of the *Mycobacterium tuberculosis (M. tb.)* protein Rv0639 that has been annotated as a homologue of *Escherichia coli (E. coli)* NusG. It exhibited dramatically different conformational

and functional properties, and hence, we re-annotated Rv0639 as a paralogue of NusG, instead of a homologue (Microbiology, 2015

Details of progress made in the current reporting year (April 1, 2014- March 31, 2015)

1) Transcription elongation factor, NusA, is a negative regulator of Rho-dependent termination in *Escherichia coli.*

NusA is an essential protein that binds to RNA polymerase (RNAP) and also to the nascent RNA, and influences transcription by inducing pausing and facilitating transcription termination / antitermination. Its involvement in Rho-dependent transcription termination has been perceived, but the molecular nature of this involvement is not known.We hypothesized that as both the Rho and the NusA are RNA-binding proteins and has the potential to target the same mRNA, the latter is likely to influence the global Rho-dependent termination. Analyses of the nascent RNA-binding properties and consequent effects on the Rho-dependent termination functions of specific NusA-RNA binding domain mutants revealed an existence of Rho-NusA direct competition for the overlapping *nut* (NusA-binding site) and *rut* (Rho-binding site) sites on the mRNA. This leads to delayed entry of Rho at the *rut* site, thereby inhibiting the latter's RNA release process. High density tiling micro-array profiles of these NusA mutants revealed that a significant number of operons are up-regulated, and majority of the genes present in these operons are also up-regulated when Rho function was compromised. This indicated the existence of NusA-binding sites in different operons, which are also the targets Rho-dependent terminations. Our data strongly argue for a direct competition between NusA and Rho for the access of specific sites on the nascent transcripts in several operons. We propose that this competition enables NusA to function as a global negative regulator of Rho function, which is unlike its role as a facilitator of hairpin-dependent termination.

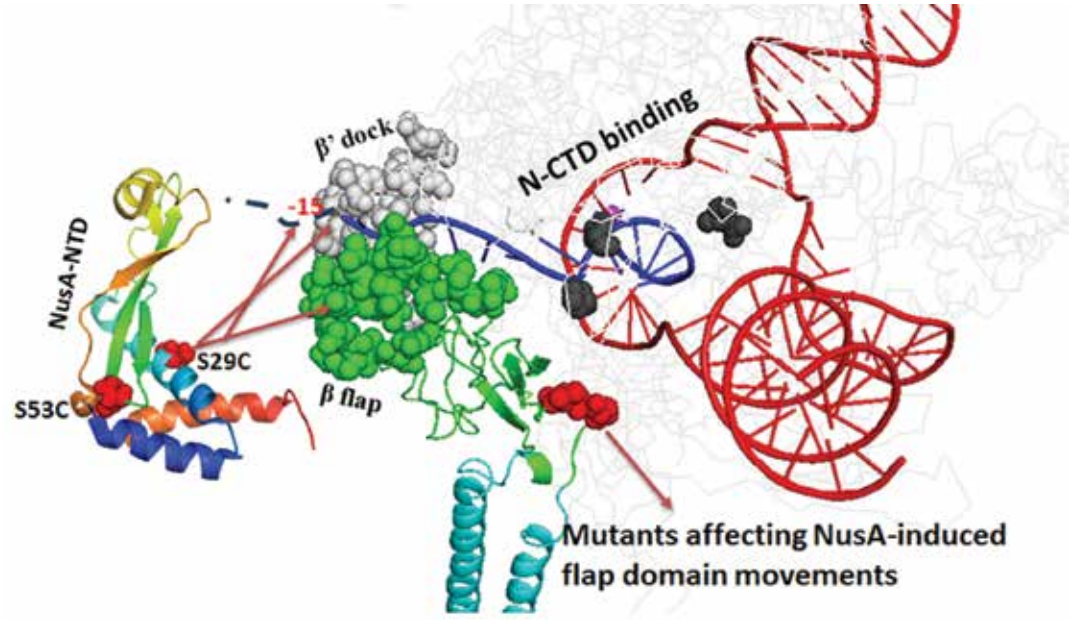

Figure 1. Model of transcription elongation complex indicating the regions of β-flap and β'-dock domains affected due to the N-NusA interaction on the surface of the EC. The regions that were cleaved from S29C of NusA are indicated by arrows. DNA template and the emerging RNA are shown in red and blue. Part of RNA outside the exit channel is shown as dashed line. Flap domain is in green. The flap-tip helix is indicated as spheres on the ribbon diagram of flap domain. The dock domain is in grey. The flap domain mutants affecting the N-antitermination via hindering the domain movement are shown in red sphere. β'- mutants directly affected N –CTD binding are shown as black spheres inside the RNA exit channel.

2) N protein from lambdoid phages transform NusA into an antiterminator by modulating NusA- RNA polymerase flap domain interactions.

Interaction of the lambdoid phage N protein with the bacterial transcription elongation factor NusA is the key component in the process of transcription antitermination. A convex surface of NusA-NTD, located opposite to its RNA polymerase-binding domain (the β-flap domain), directly interacts with N in the antitermination complex. We hypothesized that this N-NusA interaction induces allosteric effects on the NusA-RNAP interaction leading to transformation of NusA into a facilitator of the antitermination process. Here we showed that mutations in β-flap domain specifically defective for N antitermination exhibited altered NusA-nascent RNA interaction and have widened RNA exit channel indicating an intricate role of flap domain in the antitermination. Presence of N, reoriented the RNAP binding surface of NusA-NTD, which changed its interaction pattern with the flap domain. These changes caused significant spatial rearrangement of the β flap as well as the β' dock domains to form a more constricted RNA exit channel in the N-modified elongation complex (EC), which might play key role in conertingNusA into a facilitator of the N antitermination (Fig. 1). We propose that in addition to affecting the RNA exit channel and the active center of the EC, β-flap domain rearrangement is also a mechanistic component in the N antitermination process.

3) Mechanism of NusG-mediated stimulation of Rho-dependent termination.

Transcription elongation factor NusG is an interacting partner of the transcription terminator Rho that stimulates the latter's RNA release process. 21 kDNusG has two domains; the N-terminal domain (NTD) interacts with the elongating RNAP, whereas its C-terminal domain (CTD) binds to Rho. We are now in the process of elucidating the mechanism of NusG-mediated stimulation of Rho-dependent termination.

We have isolated three Rho mutants, I168V, R221C and P235H, that can function in vivo independent of NusG. This indicated that they have acquired properties, which are usually imparted by NusG. Purified Rho mutants exhibited following in vitro properties in the absence of NusG. 1) Early termination with stimulation in RNA release from the stalled elongation complex. 2) Have higher rate of ATPase activity and higher affinity for RNA at the secondary RNA binding site (SBS). However, their primary RNA binding is not affected. These mutations induce altered conformations at the secondary RNA binding domain allosterically. We propose that NusG enhances the stability of Rho SBS-RNA interactions and accelerates the open to close conformations of Rho so that the translocase competent form is attained faster (Fig. 2).

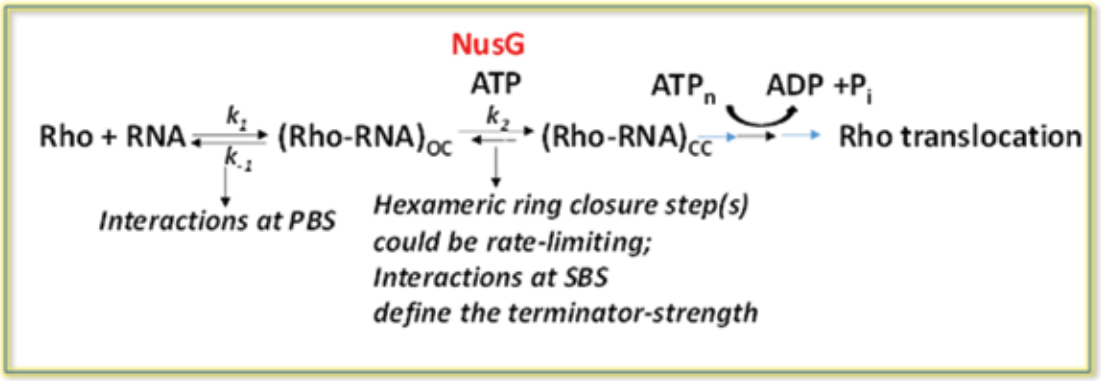

Figure 2. A kinetic scheme for the formation of translocase competent form of Rho upon interaction with the nascent RNA. NusG acts on the isomerization step that converts Rho open state (OC) to its closed state (CC).

Future Plans/directions

The following projects, being pursued in my lab, are in different stages of completion. 1) Mechanism of NusG mediated stimulation of Rho, ii) Involvement of Rho in transcription coupled repair process, iii) global analyses of Rho-dependent termination in different operons, iii) design of peptide inhibitor(s) for Rho and iv) isolation and characterization of different transcription regulators from mycobacteriophages.

Publications

1. Sen R, Chalissery J, Qayyum MZ, Vishalini V and Muteeb G (2014). Nus factors of Escherichia coli. ***EcoSal Plus*** ESP-0008-2013; doi:10.1128.
3. Shashni R, Qayyum MZ, Vishalini V, Dey D and Sen R (2014). Redundancy of primary RNA-binding functions of the bacterial transcription terminator Rho. ***Nucleic Acids Research*** 42: 9677-9690.
3. Kalyani BS, Kunamneni R, Wal M, Ranjan A and Sen R (2015). A NusG paralogue from *Mycobacterium tuberculosis*, Rv0639, has evolved to interact with ribosomal protein S10 (Rv0700) but not to function as a transcription elongation-termination factor. ***Microbiology*** 161: 67-83.

अन्य वैज्ञानिक सेवाएँ / सुविधाएँ
other scientific services / facilities

LABORATORY ANIMAL FACILITY

Faculty Coordinators	Rashna Bhandari	Staff Scientist& WT-DBT IndiaAlliance Senior Fellow
	Sanjeev Khosla	Staff Scientist
Other Members	Hole Jayant Pundalikrao	Officer In-Charge
	Sridhar Kavela	Technical Officer
	Suman Komjeti	Technical Assistant (Till May 2014)

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 (upto March 31, 2014)

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 2014, the facility housed approximately 900 mice of five different strains, and in 2013-14, users were supplied with 821 mice for IAEC approved experimentation.

Details of progress made in the current reporting year (April 1, 2014- March 31, 2015)

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 users' requirements. Currently this facility has approximately 450 adult and 170 newborn mice housed in 300 IVC cages (Table 1). During the year, 896 mice were supplied to users for IAEC approved experimentation.

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 2014-15 are highlighted below:

Strains	Total (Male+Female)	Under Breeding (Male+Female)	Supplied during 2013-14
Ip6k1	40+44	15+25	65
Nnat	20+29	06+11	28
Balb/c	35+54	07+12	630
C57BL/6	24+37	05+10	125
Foxn1^{nu}	31+28	05+12	48

Table 1. Strain-wise break up of adult mice housed at LAF as on March 31,2015, and supplied to users during 2014-15.

- 54 Balb/c mice and 2 Sprague Dawley rats were injected subcutaneously with protein antigens and polyclonal antibodies were generated successfully.
- 65 I*p6k1* and 10 C57BL/6 mice were used for tumour susceptibility analysis, histopathology, and generation of mouse embryonic fibroblasts (MEFs) for further research.
- 239 Balb/c mice were injected intravenously with *Candida glabrata* for studies on comparative bio-burden of different *Candida* strains.
- 28 *Nnat*1 mice were used for measurement of biochemical parameters.
- 177 Balb/c mice were used to study the effect of *Mycobacterium tuberculosis* protein PPE18 on LPS-induced endotoxaemia.
- 48 *FoxN1*^{nu} athymic mice were injected with oncogenic cell lines to study tumour progression and metastasis.
- 86 Balb/c and 35 C57BL/6 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.
- 37 Balb/c mice were used to study the immunomodulatory roles of recombinant purified proteins of *Mycobacterium tuberculosis.*
- 80 C57BL/6 and 37 Balb/c mice were injected with thioglycolate by intra-peritoneal route for the generation of macrophages.

We are currently involved in setting up CDFD's

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 *Ip6k1* knockout mice–version 2
3	Signal transduction pathway in immune cells regulating their innate and effecter functions during oxidative stress
4	Protocol for comparative bio-burden study of fifteen strains of *Candida glabrata* 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 PPE 18(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– version2
9	Isolation of macrophages from Balb/c mice
10	Cryopreservation of mouse embryos by vitrification
11	Understanding the role of Rab711 in phagosome maturation and immune effector signalling
12	Establishment and histopathological characterization of *Ip6k2* knockout mice
13	Establishment of transgenic mouse model to study the role of *Ip6k1* in tumorigenesis
14	Studying the immunomodulatory role of some candidate recombinantly purified proteins of mycobacteria
15	Studying the *in vivo* immunomodulatory role of some candidate PE/PPE proteins of *Mycobacterium tuberculosis* recombinantly over expressed in the non pathogenic Mycobacterial strain of *M. smegmatis*
16	Studying the in vivo epigenetic role of some candidate proteins of *Mycobacterium tuberculosis* recombinantly over expressed in the non pathogenic Mycobacterial strain of *M. smegmatis*
17	Protocol for testing tumorogenic and metastatic potential in nude mice

own Experimental Animal Facility which is under construction in the upcoming CDFD campus at Uppal, Hyderabad. We have provided inputs towards the design of the facility, including layout, ventilation system and planned workflow. We have initiated procurement of essential equipment to be installed in this facility, including double door autoclaves, a cage washer, IVC systems, and cage changing stations. An application for registration of the CDFD Experimental Animal Facility was submitted to CPCSEA, MoEF&CC, and a preliminary inspection by CPCSEA nominated inspectors was conducted successfully. We are working towards the completion of the facility, its registration with CPCSEA, and the start of operations in the near future.

Future direction

Once the CDFD Experimental Animal Facility is operational, we aim to develop cryopreservation, archiving and retrieval of transgenic mouse strains for future use. Novel methods such as the CRISPR/Cas system will be developed to generate our own transgenic and knockout mice.

BIOINFORMATICS

Head	HA Nagarajaram	Staff Scientist
Other Members	R Chandra Mohan	Technical Officer
	K Prashanthi	Technical Assistant

Objectives

1. To maintain various servers, workstations, PCs, printers and other peripheral devices;
2. To maintain the CDFD website, to provide web based services and e-mail services;
3. To maintain Institute-wide LAN as well as the internet connectivity;
4. To secure the CDFD network from security threats;
5. To integrate Institute's network into National and International grid computing networks; and
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, 2014)

- Activities related to installation, administration and maintenance of servers which providevarious services, databases and computational jobs were undertaken.
- Internet, web, email-services were provided with enhanced functionalities.
- High-end PCs, workstations, laptops, scanners and printers were procured and installed.
- Existing PC Annual Maintenance Contract was renewed.
- Awarded Annual Maintenance Contract of Zimbra email server to M/s Callippus Solutions Private Ltd.
- Configured Zimbra Email server with a failsafe server.
- Initiated the process of procuring next generation firewall, high end intelligent switches.
- Renewed the MoU with CDAC for availing GARUDA-grid facility.
- Coordinating the process of procurement and setup of server with workstations and backup facility for CODIS project.

Details of progress made in the current reporting year (April 1, 2014 - March 31, 2015)

- Activities related to installation, administration and maintenance of servers which provide various services, databases and computational jobs were undertaken.
- Internet, web, email-services have been provided with enhanced functionalities.
- High-end PCs, workstations, laptops, scanners and printers were procured and installed.
- PC Annual Maintenance Contract was awarded to a new vendor M/s Accel Frontline Limited.
- Existing AMC of Zimbra email server with M/s Callippus Solutions Private Ltd. was renewed.
- Upgraded zimbra email server to the latest version.
- Coordinating the process of procurement and completed the installation setup of server with workstations and backup facility for CODIS project.
- Renewed the MoU with CDAC for availing GARUDA-grid facility.
- Procurednext generation firewall and is currently getting installed.
- Upgraded the BSNL internet leased line bandwidth to 25Mbps.

INSTRUMENTATION

Head	Raghavendrachar J	Staff Scientist
Other Members	RN Mishra	Senior Technical Officer
	SD Varalaxmi	Technical Officer
	M Laxman	Technical Officer
	Satyanarayana	Technical Officer
	T Ramakrishna Reddy	Technical 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 (upto March 31, 2014)

During the year 2013-14, we have installed 61 new equipments like inverted microscopes, PCR machines, refrigerated centrifuges, shakingwaterbaths, -20^{0}C freezers, cooled incubator, refrigerators etc. and we have also completed 498 work orders for repair & maintenance of various laboratory equipments.

We were involved in re-organizing the first floor Lab area and have shifted and re-installed many instruments including Illumina Bead Xpress Next Generation Genotyping System, pyrosequencer, laminar hoods, fume hood etc.

In addition, we were involved in organizing the audio & visual requirements for presentations in various seminars, lectures and workshops, Foundation Day lectures, distinguished scientist lectures. We were actively involved in conducting the Guha Research Conference at ArakuVally and Vizag from 7$_{th}$ to 10 December 2013 and "Young Investigator Meeting" at Ramoji Film city, Hyderabad from 8th to 12th February 2014. 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.

Details of progress made in the current reporting year (April 1, 2014 – March 31, 2015)

During the year 2014-15, we have installed 57 new equipmentslike color doppler ultrasound scanner at NIMS, automatic vertical autoclaves, IP-Star automated robotic work station, upright microscopes, 2 Nos. of laser scanning confocal microscopes, FLA 9500 phosphor imaging system, bio-ruptors, PCR machines, refrigerated centrifuges, shaking water baths, -200C freezers, cooled incubator, refrigerators etc. and we have also completed 503 work orders for repair & maintenance of various laboratory equipments.

We were involved in re-organizing and installing the lab tables for the "Laboratory for Genomics and Profiling Applications"(LGPA) in the basement and install small equipments also.

We were involved in organizing the CODIS software installation and training to the Laboratory of DNA Fingerprinting Services (LDFS) at CDFD Library from 5th October 2014 to 12th October 2014.

In addition, we were involved in organizing the audio & visual requirements for presentations in various seminars, lectures and workshops, Foundation Day lectures, distinguished scientist lectures. 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

* Publications of adjunct faculty of CDFD in which CDFD's affiliation is included.

** Work done elsewhere

A. Publications during the year 2014

1. Abraham PR, Latha GS, Valluri VL and Mukhopadhyay S (2014). *Mycobacterium tuberculosis* PPE protein Rv0256c induces strong B cell response in tuberculosis patients. ***Infection, Genetics and Evolution*** 22: 244-249.

2. Adduri RSR, Katamoni R, Pandilla R, Madana SN, Paripati AK, Kotapalli V and Bashyam MD (2014). TP53 Pro72 allele is enriched in oral tongue cancer and frequently mutated in esophageal cancer in India. ***PLoS One*** e114002. doi: 10.1371/journal.pone.0114002.

3. Adduri RSR, Kotapalli V, Gupta NA, Gowrishankar S, Srinivasulu M, Ali MM, Rao S, Uppin SG, Nayak UK, Dhagam S, Chigurupati MV and Bashyam MD (2014). P53 nuclear stabilization is associated with FHIT loss and younger age of onset in squamous cell carcinoma of oral tongue. ***BMC Clinical Pathology*** 14:37.

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5. Ali A, Veeranki SN and Tyagi S (2014). A SET-domain-independent role of WRAD complex in cell-cycle regulatory function of mixed lineage leukemia. ***Nucleic Acids Research*** 42:7611-7624.

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51. Satyavathi VV, Raju PJ, Basha KI, Basavaraja HK and Nagaraju J (2014). Genesis and performance evaluation of baculovirus resistant transgenic silkworm hybrids. ***Sericologia*** 54: 181-187.

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57. Singh CP, Vaishna RL, Kakkar A, Arunkumar KP and Nagaraju J (2014). Characterization of antiviral and antibacterial activity of *Bombyx mori* seroin proteins. ***Cellular Microbiology*** 16: 1354-1365.

58. Sinha A and Nagarajaram HA (2014). Nodes occupying central positions in human tissue specific PPI networks are enriched with many splice variants. ***Proteomics*** 14: 2242-2248.

59. Sreejit G, Ahmed A, Parveen N, Jha V, Valluri VL, Ghosh S and Mukhopadhyay S(2014). The ESAT-6 protein of *Mycobacterium tuberculosis* interacts with beta-2-microglobulin (β2M) affecting antigen presentation function of macrophage. ***PLoS Pathogens*** 10: e1004446; doi: 10.1371/journal.ppat.1004446.

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B. Publications in 2015 (Till March 31, 2015)

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69. Dalal AB, Ranganath P, Phadke SR, Kabra M, Danda S, Puri RD, Sankar VH, Gupta N, Patil SJ, Mandal K, Tamhankar P, Aggarwal S and Agarwal M (2015). Prenatal diagnosis in India is not limited to sex selection. ***Genetics in Medicine*** 17: 88.

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71. Delma CR, Somasundaram ST, Srinivasan GP, Khursheed M, Bashyam MD and Aravindan N (2015). Fucoidan from Turbinaria conoides: a multifaceted 'deliverable' to combat pancreatic cancer progression. ***International Journal of Biological Macromolecules*** 74: 447-457.

72. Dutta UR, Hansmann I and Schlote D (2015). Molecular cytogenetic characterization of a familial pericentric inversion 3 associated with short stature. ***European Journal of Medical Genetics*** 58: 154-159.

73. Ghosh C, Prakash NR, Manna SK, Bishayi B. (2015) Presence of toll like receptor-2 in spleen, lymph node and thymus of Swiss albino mice and its modulation by *Staphylococcus aureus* and bacterial lipopolysaccharide. ***Indian Journal of Experimental Biology*** 53: 82-92.

74. Gowrishankar J (2015). End of the beginning: elongation and termination features of alternative modes of chromosomal replication initiation in bacteria. ***PLoS Genetics*** 11: e1004909.

75. *Gupta D, Gupta V, Singh V, Chawla S, Ranganath P and Phadke SR (2015). Study of polymorphisms in CFH, ARMS2

and HTRA1 genes as potential risk factors for age-related macular degeneration in Indian patients. ***International Journal of Bioassays*** 4: 3747- 3752.

76. Kalyani BS, Kunamneni R, Wal M, Ranjan A and Sen R (2015). A NusG paralogue from *Mycobacterium tuberculosis,* Rv0639, has evolved to interact with ribosomal protein S10 (Rv0700) but not to function as a transcription elongation-termination factor. ***Microbiology*** 161: 67-83.

77. Kiran S, Anwar T, Kiran M and Ramakrishna G (2015). Sirtuin 7 in cell proliferation, stress and disease: rise of the seventh sirtuin. ***Cellular Signalling*** 27: 673-682.

78. Kiran S, Oddi V and Ramakrishna G. (2015). Sirtuin 7 promotes cellular survival following genomic stress by attenuation of DNA damage, SAPK activation and p53 response. ***Experimental Cell Research*** 331: 123-141.

79. Parine NR, Lakshmi P, Kumar D, Shaik JP, Alanazi M and Pathan AAK (2015). Development and characterisation of nine polymorphic microsatellite markers for *Tephrosia calophylla* Bedd. (Fabaceae). ***Saudi Journal of Biological Sciences*** 22: 164–167.

80. Rachita HR and Nagarajaram HA (2015). Molecular principles of human virus protein-protein interactions. ***Bioinformatics*** 31: 1025-1033.

81. Rai MN, Sharma V, Balusu S and Kaur R (2015): An essential role for phosphatidylinositol 3-kinase in the inhibition of phagosomal maturation, intracellular survival and virulence in *Candida glabrata.* ***Cellular Microbiology*** 17: 269-287.

82. *Ramasarma T, Rao AV, Devi MM, Omkumar RV, Bhagyashree KS and Bhat SV (2015). New insights of superoxide dismutase inhibition of pyrogallol autoxidation. ***Molecular and Cellular Biochemistry*** 400: 277-285.

83. Stephen J, Girisha KM, Dalal A, Shukla A, Shah H, Srivastava P, Kornak U and Phadke SR (2015). Mutations in patients with osteogenesis imperfecta from consanguineous Indian families. ***European Journal of Medical Genetics*** 58: 21-27.

84. Thota SG, Unnikannan CP, Thampatty SR, Manorama R and Bhandari R (2015). Inositol pyrophosphates regulate RNA polymerase I-mediated rRNA transcription in Saccharomyces cerevisiae. ***Biochemical Journal*** 466: 105-114.

85. Yousuf S, Angara R, Vindal V and Ranjan A (2015). Rv0494 is a starvation-inducible, auto-regulatory FadR like regulator from *Mycobacterium tuberculosis.* ***Microbiology*** 161: 463-476.

C. Publications in Press (as on March 31, 2015)

86. *Aggarwal S and Phadke SR. Medical genetics and genomic medicine in India: current status and opportunities ahead. ***Molecular Genetics and Genomic Medicine.***

87. Anusha U, Ranganath P, Jamal Md NJS, Krishna Prasad C, Anupam S, Verma IC, Phadke SR, Puri RD, Danda S, Muranjan MN, Jevalikar G, Nagarajaram HA and Dalal AB. Novel mutations of the ARSB gene in Indian patients with Mucopolysaccharidosis Type IV. ***Indian Journal of Medical Research.***

88. Bashyam MD, Kotapalli V, Raman R, Chaudhary AK, Yadav BK, Gowrishankar S, Uppin SG, Kongara R, Sastry RA, Vamsy M, Patnaik S, Rao S, Dsouza S, Desai D and Tester A. Evidence for presence of mismatch repair gene expression positive Lynch syndrome cases in India. ***Molecular Carcinogenesis*** doi: 10.1002/mc.22244.

89. Bhat KH and Mukhopadhyay S. Macrophage takeover and the host-bacilli interplay during tuberculosis. ***Future Microbiology.***

90. Bidchol AM, Dalal A, Trivedi R, Shukla A, Nampoothiri S, Sankar VH, Danda S, Gupta N, Kabra M, Hebbar SA, Bhat RY, Matta D, Ekbote AV, Puri RD, Phadke SR, Gowrishankar K, Aggarwal S, Ranganath P, Sharda S, Kamate M, Datar CA, Bhat K, Kamath N, Shah H, Krishna S, Gopinath PM, Verma IC, Nagarajaram HA, Satyamoorthy K and Girisha KM. Recurrent and novel GLB1 mutations in India. ***Gene.***

91. Dalal A, Aneek Das Bhowmik, Divya Agrawal and Phadke SR. Exome sequencing and homozygosity mapping help in identification of genetic etiology for spastic ataxia in a consanguineous family. ***Indian Journal of Medical Research.***

92. Pasha J, Kandagatla B, Sen S, Seerapu GPK, Bujji S, Haldar D, Nanduri S and

Oruganti S (2015). Amberlyst-15 catalyzed Povarov reaction of N-arylidene-1H-indazol-6-amines and indoles: a greener approach to the synthesis of exo-1,6,7,7a,12,12a-hexahydroindolo[3,2-c]pyrazolo[3,4-f] quinolines as potential sirtuin inhibitors. ***Tetrahedron Letters.***

93. Rai R, Javvadi S and Chatterjee S. Cell-cell signalling promotes ferric iron uptake in *Xanthomonas* oryzae pv. oryzicola that contribute to its virulence and growth inside rice. ***Molecular Microbiology*** doi: 10.1111/mmi.12965.
94. Shantibala T, Victor T, Luikham R, Arunkumar KP, Debaraj Sharma H, Lokeshwari RK and Kim I. Complete mitochondrial genome of the wild eri silkworm, *Samia canningi* (Ledpidoptera: Saturniidae). ***Mitochondrial DNA.***
95. Sharma G, Upadhyay S, Srilalitha M, Nandicoori VK and Khosla S. The interaction of mycobacterial protein Rv2966c with host chromatin is mediated through non-CpG methylation and histone H3/H4 binding. ***Nucleic Acids Research.***
96. Singh S, Kumar PU, Thakur S, Kiran S, Sen B, Sharma S, Rao VV, Poongothai AR and Ramakrishna G. Expression/localization patterns of sirtuins (SIRT1, SIRT2, and SIRT7) during progression of cervical cancer and effects of sirtuin inhibitors on growth of cervical cancer cells. ***Tumor Biology.***
97. Srivastava VK, Suneetha KJ and Kaur R. The mitogen-activated protein kinase CgHog1 is required for iron homeostasis, adherence and virulence in *Candida glabrata.* ***The FEBS Journal.***

D. Other Publications

1. Arunkumar KP (2014). Book review of the Annual Review of Genetics, 2013. Bonnie L. Bassler et al. (eds). ***Current Science*** 106: 1755-1757.
2. Arunkumar KP (2014). Role of Biotechnology in seri-development. ***Indian Silk*** 5: 74-76.
3. Dalal A (2014). Phenylketonuria: Past, present and future. ***Genetics Clinics*** 7: 19-24.
4. Dutta UR (2014). Precision in chromosome identification with leads in molecular cytogenetics: an illustrated review. ***Journal of Pediatric Genetics*** doi: 10.3233/PGE-14083.
5. Kasbekar DP (2014). Editorial. Lesser models. ***Journal of Biosciences*** 39: 1.
6. Kasbekar DP (2014). Sidelights. Are any fungal genes nucleus-limited? ***Journal of Biosciences*** 39: 341-346.
7. Ranganath P and Dalal A (2014). Quality issues in medical genetics. ***Genetics in Clinical Practice*** (1st Edition) 237-243.
8. Ranganath P (2014). Approach to Intellectual Disability. ***Genetic Clinics*** 7: 12-18.
9. Ranganath P (2014). Approach to a child with dysmorphism/ congenital malformation. ***Genetic Clinics*** 7: 11-17.
10. Ranganath P and Rai GK (2014). Marfan Syndrome: Recent advances in diagnosis and management. ***Genetic Clinics*** 7: 6-10.
11. Satyavathi VV (2014). International exposure to GM research. ***South Asia Biosafety Program Newsletter*** 11: 2.
12. Kasbekar DP (2015). What have we learned by doing transformations in Neurospora tetrasperma? Genetic transformation systems in fungi. ***Springer,*** Switzerland 47-52.
13. Kasbekar DP (2015). Editorial. Via media. ***Journal of Biosciences*** 40: 1.
14. Ranganath P (2015). Patterns of Inheritance. In: Postgraduate Textbook of Pediatrics. Ed. Piyush Gupta. Jaypee Brothers Medical Publishers 24-31.

E. Patents granted

1. Hasnain SE. Antigenic peptides.

European Patent Application No. 05779727.6

Patent No.: 1809658

Date of grant: June 25, 2014

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 MBBS or Masters degree in any branch of Science, Technology or Agriculture from a recognized University or Institute. Candidates (other than MBBS graduates) must have cleared National Eligibility Test (NET) with valid CSIR-JRF or UGC-JRF or DBT-JRF or ICMR-JRF or ICAR-JRF or INSPIRE-PhD or JEST or GATE (All India top 50 ranks of all Chemistry, Life Sciences and Biotechnology steams). Those who have appeared for their final semester examination, but are awaiting results, are also eligible to apply. Those with independent Senior Research Fellowships (SRF) from CSIR can also apply. 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, 2015 the Centre has 108 Research Scholars working for their doctorates in different areas of research. In the reporting year 4 of the 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 selectedcompetitively by the DST fast track young scientist scheme, 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-onexperience in modern biology. In the reporting year, 5 students were given the opportunity to avail training under this programme.

Research Scholars Conferred PhD Degree During the Reporting Period

Scholar	Supervisor	Date of *viva voce* examination	Title of thesis
Maruti Nandan Rai	Rupinder Kaur	15.04.2014	Study of host-pathogen interaction in *Candida glabrata*
Ratheesh R	MD Bashyam	13.05.2014	A comprehensive analysis of tumorigenesis pathways driving early and late onset colorectal cancer in India
Anupam Sinha	HA Nagarajaram	05.01.2015	Computational analysis of the effects of alternative splicing on protein-protein interaction networks
Sapan Borah	Rupinder Kaur	03.02.2015	A molecular analysis of antifungal drug susceptibility in the human opportunistic pathogen *Candida glabrata*

पुरस्कार एवं सम्मान
Awards and Honours

AWARDS & HONOURS

FACULTY & STAFF	
Dr Ashwin B Dalal	Selected under Exchange of Scientists Programme between INSA and Royal Society, Edinburgh (2015) – visit to Laboratory of Dr Andrew Jackson, Medical Research Council Laboratory, University of Edinburgh, Scotland, UK for 4 weeks from 6th June to 6th July, 2015.
Dr Murali Dharan Bashyam	Awarded the National Bioscience Award for Career Development-2013 from the DBT
Dr Rupinder Kaur	Invited as Plenary Lecturer in the "6th FEBS Advanced Lecture Course on Human Fungal Pathogens" to be held at Nice, France during May 2015
Dr Sangita Mukhopadhyay	Awarded ICMR Basanti Devi Amir Chand Prize-2011
PhD STUDENTS & PROJECT PERSONNEL	
Ms. Anusha Uttarilli	DST travel grant to attend 64th Annual Meeting of the American Society of Human Genetics (ASHG) in California, USA (October 2014)
Mr Aushaq Bashir Malla	Poster Presentation Award at the Chromosome Stability Conference-2014 held at Jawaharlal Nehru Centre for Advanced Scientific Research (JNCASR), Bengaluru (December 2014)
Mr Bhavik Sawhney	Poster Award at the 25th tRNA Conference held at Kyllini, Greece (September 2014)
Mr Kundan Kumar	Shyama Prasad Mukherjee Fellowship (SPMF) in the CSIR-UGC National Eligibility Test (NET) held in June 2014
Mr Imtiyaz Yaseen	Poster Prize from the Biochemical Journal, UK at 5th meeting of Asian Forum of Chromosome and Chromatin Biology held at JNCASR, Bangalore (January 2015)
Dr Nagender Rao R	"DST-SERB" Fast track Young Scientist Award (2015)
Ms. Narmadha Reddy	Dr KV Rao Research Award (2013-2014)
Ms. Neelam Chaudhary	Best Poster Award at International conference on genome architecture and cell fate regulation held at University of Hyderabad (December 2014)
Mr Philip Raj Abraham	(i) ICMR Travel Grant from ICMR, New Delhi and (ii) Prof GP Talwar Travel Bursary from Immunology Foundation, New Delhi to attend the 12th International Conference on Molecular Epidemiology and Evolutionary Genetics of Infectious Diseases (MEEGID XII) held at Bangkok, Thailand (2014)
Mr Rathan Singh Jadav	Poster Presentation Award at the Chromosome Stability Conference-2014 at Jawaharlal Nehru Centre for Advanced Scientific Research (JNCASR), Bengaluru (December 2014)
Dr Ratheesh Raman	Young Scientist Award-2014 from the AP Akademi of Sciences, Hyderabad
Ms. Rikky Rai	ASM Travel Award for attending ASM Conference on Cell-Cell Communication in Bacteria at San Antonio, Texas, USA (October 2014)
Mr Vishwanath Jha	Best Poster Award at 83rd Annual Meeting of the Society of Biological Chemists of India, Bhubaneshwar (December 2014)

व्याख्यान, बैठक, कार्यशाला व अन्य महत्वपूर्ण कार्यक्रम

Lectures, Meetings, Workshops and Important Events

DISTINGUISHED VISITORS AND LECTURES

Visitor	Title of Lecture	Date
Dr Kunal Rai Department of Genomic Medicine, University of Texas MD Anderson Cancer Center, Houston, TX USA	Functional epigenomics of melanoma progression	11.04.2014
Dr Pramod Kaitheri Kandoth Division of Plant Sciences, Interdisciplinary Plant Group, University of Missouri, Columbia, MO, USA	Elucidating function of a SHMT in plant nematode resistance	28.04.2014
Dr Umasankar K. Perunthottathu Department of Cell Biology and Physiology University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania, USA	Traffic block: Sort it all out in one minute	07.05.2014
Prof Paturu Kondaiah MRDG, Indian Institute of Science,Bangalore	Identification of novel gene expression profiles that predict prognosis in Indian Breast cancer patients	13.05.2014
Dr Prashanth Kumar Bajpe Department of Molecular Carcinogenesis, Netherlands Cancer Institute, Plesmanlaan 121, 1066CX, Amsterdam, The Netherlands	Understanding resistance to cancer drugs through functional genetics	05.06.2014
Dr Aravind Penmatsa Vollum Institute, Oregon Health and Science University, Portland, OR, USA	Structure of the dopamine transporter unravels mechanism of neurotransmitter transport inhibition	11.06.2014
Dr Souvik Bhattacharjee University of Notre Dame, Center for Rare and Neglected Diseases, Notre Dame, USA	From malaria to Irish potato famine: Reprograming the host through PI(3)P	12.06.2014
Dr Dipanjan Roy Department of Neurology, Charite, Chariteplatz 1, Berlin, Germany	Traces of learning in the spontaneous activity in the human cortex: Experimental and modeling insights	21.08.2014
Dr Sandeep M. Eswarappa Dept. of Cellular and Molecular Medicine, Cleveland Clinic Lerner Research Institute, Cleveland, USA	Translation beyond the stop codon	12.09.2014

Visitor	Title of Lecture	Date
Dr Raghu Padinjat National Centre for Biological Sciences, Bangalore	Regulation of insulin signalling and cell size by a novel phosphoinositide kinase	25.09.2014
Dr Debabrata Mandal NIPER-Hajipur, Bihar	Transfer RNA and genetic code: A modified view	29.09.2014
Dr Gaurav Das Centre for Neural Circuits and Behaviour (CNCB), Anatomy and Genetics, Oxford University	Neural circuit basis of learning, memory and behavior: Drosophila learn opposing components of a compound food stimulus	31.10.2014
Prof Upender Manne Comprehensive Cancer Center and Minority Health & Health Disparities Research Center, University of Alabama, Birmingham, USA	Importance of population-based studies in developing cancer molecular biomarkers	11.12.2014
Dr Prasanna K Devaraneni Department of Biochemistry and Molecular Biology, School of Medicine, Oregon Health & Sciences University, Portland, OR-USA	Membrane protein biogenesis: Topogenesis, assembly and function	15.12.2014
Dr Devanjan Sinha Indian Institute of Science, Bangalore	Mitochondrial chaperones: Bridging the gap between the organeller function and disease biology	09.02.2015
Dr Virupakshi Soppina University of Michigan Medical School, Ann Arbor, USA	Kinesin-3 motors are marathon runners of the cellular world	12.02.2015
Dr Parthasarathy Sampathkumar Albert Einstein College of Medicine, Bronx, NY, USA	TbPEX5, MtbThyX, and a peek into the inner ring of nuclear pore complex: A tale of Nup192	16.02.2015
Dr Amit Ghosh Lawrence Berkeley National Laboratory, California, USA	Quantitative systems and synthetic biology for microbial engineering	20.02.2015
Prof Stephane Genin INRA, France	*Ralstonia solanacearum* pathogenesis and adaptation to the plant environment	04.03.2015
Prof Philippe Bouloc Laboratoire Signalisation et Réseaux de Régulations Bactériens, Institute for Integrative Biology of the Cell (I2BC), CEA, CNRS, Université Paris-Sud, Orsay Cedex, France	Trapping sRNA targets in *Staphylococcus aureus* to decipher sRNA-dependent networks	24.03.2015

IMPORTANT EVENTS

Event	Date
Anti -Terrorism Day	21.05.2014
Press Meet on “DNA test for victims of Uttarakhand Tragedy”	23.06.2014
Summer Trainees’ Colloquium	25.06.2014
Mock fire drill	26.06.2014 – 27.06.2014
16th meeting of CDFD Research Area Panels-Scientific Advisory Committee (RAP-SAC)	04.07.2014 – 05.07.2014
Video conference with the Hon’ble President using NKN	04.08.2014 or 05.08.2014
Independence Day Celebrations	15.08.2014
Sadbhavana Diwas Pledge	20.08.2014
Hindi Pakhwada Celebrations	01.09.2014 – 15.09.2014
Meeting of the Institutional Biosafety Committee (IBSC) of CDFD	10.09.2014
Hindi Day Celebrations	17.09.2014
Swachh Shapath (Cleanliness Pledge)	02.10.2014
Installation and training program on CODIS Software (Visit of Hon’ble Consul General of USA Mr Michael Mullins for Valedictory function on 10.10.2014)	07.10.2014 – 11.10.2014
30th Meeting of the CDFD Finance Committee	13.10.2014
23rd Meeting of the CDFD Building Committee	13.10.2014
Vigilance Awareness Week	27.10.2014
Visit of Dr Tomohiro Shimada Tokyo Institute of Technology, Japan under the DST-JSPS collaborative research project	30.10.2014 – 06.11.2014
Indo-US symposium on Genomic insights into Human morphogenesis – 1st Annual meeting of Society for Indian Academy of Medical Genetics-2014	07.11.2014 – 09.11.2014
Visit of Meritorious students of Madhya Pradesh State under Vigyan Manthan Yatra organized under Madhya Pradesh Mission Excellence Programme by Madhya Pradesh Council of Science & Technology, Bhopal	12.11.2014
36th meeting of the Governing Council of CDFD	13.11.2014
19th Annual General Body meeting of the CDFD Society	13.11.2014
Visit of Prof K VijayRaghavan, Secretary, DBT for interaction with faculty	23.11.2014
Visit of Hon’ble newly recruited District Judges from Andhra Pradesh Judicial Academy, Secunderabad as a part of their foundation course	26.11.2014
Visit of Prof Kazuei Mita Genome Biology Southwest University, China	05.12.2014
Visit of Shri Santosh Sarangi Chairman APEDA, New Delhi for Mitochondrial chaperones: Bridging the gap between the organeller function and disease biology	05.12.2014
Visit of Dr SK Saxena Director Export Inspection Council (EIC), Delhi for Discussion Meeting on the activities of APEDA-CDFD Centre for Basmati DNA Analysis	23.12.2014
Republic Day celebrations	26.01.2015
CDFD Foundation Day Celebrations	28.01.2015

Observance of silence on ‘Martyrs day’	30.01.2015
Productivity Week Celebrations-2015	12.02.2015 – 18.02.2015
CDFD Institutional Biosafety Committee meeting	24.02.2015
Unicode Hindi Software Workshop	20.03.2015
Visit of Prof Sylvie Rimsky Director of Research Ecole Nationale Superieure (ENS), Cachan, France under Indo-France collaborative research project	23.03.2015-24.03.2015

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

DEPUTATIONS ABROAD – FACULTY & STAFF

Faculty/Staff	Period	Country of Visit and Purpose
J Gowrishankar	01.04.2014 to 04.04.2014	**France & Belgium:** (i) to meet the groups of his research collaborators Drs. Sylvie Rimsky and Philippe Bouloc in Paris, France for scientific discussions (ii) to participate and to speak in the workshop titled "Implementing a global research agenda for AMR" and also to attend a joint workshop with the European Commission on "Antibiotics and their alternatives" being organized by the Joint Programming Initiative on Antimicrobial Resistance (JPIAMR) in Brussels, Belgium.
	01.08.2014 to 09.08.2014	**USA:** (i) to visit the laboratories of Prof. Stanley N Cohen, Stanford University and Prof. Carol A Gross, University of California, San Francisco (ii) to attend the "2014 Molecular Genetics of Bacteria and Phages Meeting" at University of Wisconsin, Madison, Wisconsin, USA
	24.02.2015 to 10.03.2015	**Japan:** (i) to visit the laboratory of Dr. Tomohiro Shimada (Japanese Investigator), Tokyo Institute of Technology, in connection with implementation of the joint India-Japan research project with Dr. Tomohiro titled "Analysis of co-regulation between DNA replication and amino acid homeostasis by transcription factor IciA / ArgP in *Escherichia coli"* (ii) to visit Prof. Nobuo Shimamoto at Kyoto Sangyo University, Kyoto
Ranjan Sen	31.08.2014 to 06.09.2014	**UK:** To attend the Total Transcription Conference at Wellcome Trust Conference Centre, Hinxton, Cambrideshire, UK.
Murali Dharan Bashyam	01.04.2014 to 10.04.2015	**USA:** (i) To visit (a) UCLA Jonsson Comprehensive Cancer Centre at Los Angeles, CA, USA (b) Moore's Cancer Centre, UCSD, San Diego, CA, USA (c) Beckman Facility at San Diego and (ii) To attend the "American Association for Cancer Research (AACR) Annual Meeting" during 5-9 April, 2014 at San Diego, CA, USA (iii) To visit the laboratory of Dr. Ramana Davuluri at The Robert H. Lurie Comprehensive Cancer Centre of Northwestern University, Chicago and Prof. Ananda Chakrabarty at University of Illinois, Chicago to perform next generation sequencing data analysis and to discuss collaboration on cancer genomics.

Rupinder Kaur	24.09.2014 to 02.10.2014	**Germany:** To present her work in the EMBL Conference on "Frontiers in Fungal Systems Biology" scheduled to be held at Heidelberg, Germany.
Rashna Bhandari	09.10.2014 to 12.10.2014	**France:** To attend and submit an abstract at the 39th European Symposium on Hormones and Cell Regulation which will focus on "Inositol Lipid Signaling: from molecular mechanisms to human pathologies" at Mont Ste Odile, France.
	13.10.2014 to 18.10.2014	**Germany:** To visit Laboratory of Prof. Georg Mayr, Institute of Biochemistry and Signal Transduction, University Medical Center, Hamburg – Eppendorf, Hamburg, Germany.
N Madhusudan Reddy	01.06.2014 to 12.07.2014	**Germany:** To conduct research as Guest Scientist in the laboratory of Prof. Mark Stoneking, Professor for Biological Anthropology, Department of Evolutionary Genetics, Max Planck Institute for Evolutionary Anthropology (MPI-EVA), Leipzig, Germany against his fourth 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.
	15.08.2014 to 15.12.2014	**USA:** to visit the laboratory of Prof. Arthur Eisenberg, Professor and Chairman, Department of Molecular and Medical Genetics, University of North Texas Health Science Center, Fort Worth, Texas, USA for conducting research work in the area of forensic DNA profiling as a part of Indo-US Research Fellowship.
Shweta Tyagi	05.12.2014 to 13.12.2014	**USA:** (i) To attend "The American Society of Cell Biology (ASCB / IFCB) meeting at Philadelphia, Pennsylvania, USA (ii) To meet Dr. Iain Cheeseman in Massachusetts Institute of Technology, Boston and Dr. Michael Cosgrove in Upstate Medical University, Syracuse, New York to discuss results and explore future collaborations.
Subhadeep Chatterjee	06.06.2014 to 15.06.2014	**China:** (i) To attend the 13th International Conference on Plant Pathogenic Bacteria (ICPPB) and give a talk on the work on Xanthomonas - plant interaction at Shanghai Jiao Tong University, Shanghai, China.

		(ii) To visit Dr. Goungyou Chen's laboratory and to visit department of Plant Pathology for exploring future collaboration and scientific discussion in Shanghai Jiao Tong University, Shanghai.
Sardesai Abhijit Ajit	30.01.2015 to 11.02.2015	**Japan:** To visit the Tokyo Institute of Technology (Titech), Tokyo, Yokohama, Japan under the Indo-Japan Collaborative Research Project between the Laboratory of Bacterial Genetics, CDFD and the Laboratory of Dr. Tomohiro Shimada.
R Harinarayanan	04.08.2014 to 11.08.2014	**USA:** To attend a conference titled "Molecular Genetics of Bacteria and Phages" at Madison, Wisconsin, USA and to visit the laboratory of Dr. Michael Cashel for scientific interaction at NIH, Bethesda, USA.
Arun Kumar KP	01.04.2014 to 11.04.2014	**Italy:** (i) To visit the Laboratory of Prof. Giuseppe Saccone at the University of Naples, Italy to discuss on the collaborative research activities on insect sex determination. (ii) To attend the Final Research Coordination Meeting (FRCM) on "Development and evaluation of improved strains of insect pests for SIT" at the International Atomic Energy Agency (IAEA), Capri, Italy.
	28.09.2014 to 13.10.2014	**France:** As a part of his collaborative research work under the Indo French Centre for the Promotion of Advanced Research (IFCPAR) project on "Global transcriptomics of sex-specific splicing" as an exchange visit to the laboratory of Prof. Leonard Rabinow at the Centre de Neurosciences de Paris Sud, France.
Venkata Satyavathi	14.09.2014 to 17.09.2014	**Sri Lanka:** To participate in the 2nd Annual "South Asia Biosafety Conference" at Tay Samudra Hotel, Colombo, Sri Lanka.

DEPUTATIONS ABROAD – STUDENTS

Name of the Scholar	Period	Country of Visit and Purpose
Adduri Sita Rama Raju	05.04.2014 to 09.04.2014	**USA:** To attend American Association for Cancer Research (AACR) Annual Meeting-2014
Saurabh Mishra	26.04.2014 to 30.04.2014	**USA:** To attend 2014 ASBMB Annual Meeting on Experimental Biology (EB 2014)
Atul Udgata	01.06.2014 to 06.06.2014	**Greece:** To attend 11th International Conference on Innate Immunity
Garima Sharma	08.06.2014 to 13.06.2014	**USA:** To attend Gordon Research conference titled "Chromatin structure and function"
Mugdha Singh	01.07.2014 to 10.08.2014	**Germany:** Visiting Scholar / Guest Researcher as part of the Max Planck Partner Group Programme
Rachita HR	12.07.2014 to 15.07.2014	**USA:** To attend International Conference ISMB-2014
Arpita Goswami	26.07.2014 to 01.08.2014	**USA:** To attend Gordon Research Conference and Gordon Research Seminar on "Diffraction methods in Structural Biology (GRC and GRS 2014)"
Aanisa Nazir	05.08.2014 to 09.08.2014	**USA:** To attend 2014 Molecular Genetics of Bacteria and Phages Meeting
Amit Pathania	05.08.2014 to 09.08.2014	**USA:** To attend 2014 Molecular Genetics of Bacteria and Phages Meeting
Amitava Basu	09.09.2014 to 13.09.2014	**USA:** To attend Epigenetics and chromatin conference
Bhavik Sawhney	21.09.2014 to 25.09.2014	**Greece:** To attend 25th tRNA conference
Anusha Uttarilli	18.10.2014 to 22.10.2014	**USA:** To attend 64th Annual meeting of the American Society of Human Genetics (ASHG)
Rikky Rai	18.10.2014 to 21.10.2014	**USA:** To attend 5th ASM Conference on Cell-Cell Communication in Bacteria
Neelam Chaudhary	11.01.2015 to 16.01.2015	**USA:** To attend Keystone meeting on "The biological code of cell singalling: a tribute to Tony Pawson(F1)"
Jadav Rathan Singh	07.02.2015 to 13.02.2015	**USA:** To attend Gordon Research Seminar and Gordon Research Conference on "Mammalian DNA Repair: Controlling Traffic on the Streets and at the Crossroads of DNA Repair Pathways"
Tarique Anwar	08.03.2015 to 12.03.2015	**USA:** To attend Keystone Symposia Conference on "Biology of Sirtuins (C3)"

सीडीएफडी के संकाय एवं अधिकारी
Faculty and Officers of CDFD

SCIENTIFIC GROUP LEADERS (FACULTY)

Dr J Gowrishankar

Dr DP Kasbekar

Dr Ranjan Sen

Dr Sangita Mukhopadhyay

Dr MD Bashyam

Dr Sunil Kumar Manna

Dr Nagarajaram HA

Dr Akash Ranjan

Dr Rupinder Kaur

Dr Sanjeev Khosla

Dr Ashwin B Dalal

Dr Rashna Bhandari

Dr Devyani Haldar

Dr N Madhusudan Reddy

Dr Shweta Tyagi

Dr MV Subba Reddy

Dr Subhadeep Chatterjee

Dr Sardesai Abhijit Ajit

Dr Rohit Joshi

Dr R Harinarayanan

Dr Arun Kumar KP

ADJUNCT FACULTY

Dr EA 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

Mr S Ayub Basha

Mr B Jagannathacharyulu

केन्द्र की समितियाँ

(31.03.2015 तक)

Committees of the Centre

(As on 31.03.2015)

MEMBERS OF CDFD SOCIETY

Dr Harsh Vardhan Hon'ble Minister for S&T and Earth Sciences	-	President
Prof K VijayRaghavan Secretary, DBT, New Delhi	-	Member (Ex-officio)
Dr PS Ahuja Director General, CSIR, New Delhi	-	Member (Ex-officio)
Dr Manoj S Rohilla Scientist 'C', DBT (Nominee of Dr (Mrs) Suman Govil, Adviser, DBT, New Delhi)	-	Member (Ex-officio)
Ms Kusum Lata Sharma Deputy Secretary, DBT (Nominee of JS & FA, DBT, New Delhi	-	Member (Ex-officio)
Joint Secretary (PM) Ministry of Home Affairs, New Delhi	-	Member (Ex-officio)
Shri Devkant Deputy Legal Adviser (Nominee of Joint Secretary & Legal Adviser, Ministry of Law & Justice, New Delhi)	-	Member (Ex-officio)
Dr JR Gaur PSO, BPR&D, New Delhi (Nominee of Director General, BPR&D, New Delhi)	-	Member (Ex-officio)
Prof P Balaram IISc, Bangalore Chairman of Scientific Advisory Committee, CDFD	-	Member (Ex-officio)
Prof VS Chauhan Visiting Scientist, ICGEB, New Delhi	-	Member
Prof Dipankar Chatterji IISc, Bangalore	-	Member
Dr J Gowrishankar Director, CDFD, Hyderabad	-	Member Secretary

MEMBERS OF CDFD GOVERNING COUNCIL

Prof K VijayRaghavan Secretary, DBT, New Delhi	-	Chairperson
Dr PS Ahuja Director General, CSIR, New Delhi	-	Member (Ex-officio)
Dr Manoj S Rohilla Scientist 'C', DBT (Nominee of Dr (Mrs) Suman Govil, Adviser, DBT, New Delhi)	-	Member (Ex-officio)
Ms Kusum Lata Sharma Deputy Secretary, DBT (Nominee of JS & FA, DBT, New Delhi	-	Member (Ex-officio)
Joint Secretary (PM) Ministry of Home Affairs, New Delh	-	Member (Ex-officio)
Shri Devkant Deputy Legal Adviser (Nominee of Joint Secretary & Legal Adviser, Ministry of Law & Justice, New Delhi)	-	Member (Ex-officio)
Dr JR Gaur PSO, BPR&D, New Delhi (Nominee of Director General, BPR&D, New Delhi)	-	Member (Ex-officio)
Prof P Balaram IISc, Bangalore Chairman of Scientific Advisory Committee, CDFD	-	Member (Ex-officio)
Prof VS Chauhan Visiting Scientist, ICGEB, New Delhi	-	Member
Prof Dipankar Chatterji IISc, Bangalore	-	Member
Dr J Gowrishankar Director, CDFD, Hyderabad	-	Member Secretary

MEMBERS OF CDFD RESEARCH AREA PANELS – SCIENTIFIC ADVISORY COMMITTEE (RAP-SAC)

Prof P Balaram Director, IISc, Bangalore	-	Chairman
Dr Vijay Kumar ICMR, New Delhi (ICMR representative)	-	Member
Shri V Venugopal Director, CFSL, Hyderabad (MHA representative)	-	Member
Dr (Mrs) Suman Govil DBT representative	-	Member
Dr KV Prabhu IARI, New Delhi (ICAR representative)	-	Member
Dr Ghanshyam Swarup CCMB, Hyderabad (CCMB representative)	-	Member
Dr Veena K Parnaik CCMB, Hyderabad	-	Member
Dr SK Apte BARC, Mumbai	-	Member
Dr Usha Vijayraghavan IISc, Bangalore	-	Member
Prof Umesh Varshney IISc, Bangalore	-	Member
Dr Sandhya S Visweswaraiah IISc, Bangalore	-	Member
Dr Jaya Sivaswami Tyagi AIIMS, New Delhi	-	Member
Prof MK Mathew NCBS, Bangalore	-	Member
Prof Sanjeev Galande IISER, Pune	-	Member
Dr Joyoti Basu Bose Institute, Kolkata	-	Member
Dr Debasisa Mohanty NII, New Delhi	-	Member
Dr Shubha R Phadke SGPGI, Lucknow	-	Member
Dr Ramakrishna Ramaswamy UoH, Hyderabad	-	Member
Dr J Gowrishankar Director, CDFD, Hyderabad	-	Member Secretary

MEMBERS OF CDFD ACADEMIC COMMITTEE

Prof AS Raghavendra Dean, School of Life Sciences University of Hyderabad, Hyderabad	-	Chairman
Prof Anil K Tyagi University of Delhi, South Campus, New Delhi	-	Member
Dr K Satyamoorthy Director, Manipal Life Sciences Centre Manipal University, Manipal	-	Member
Dr DP Kasbekar Haldane Chair, CDFD, Hyderabad	-	Member
Dr Ranjan Sen Staff Scientist, CDFD, Hyderabad	-	Member
Dr Sanjeev Khosla Staff Scientist & Coordinator (Academics) CDFD, Hyderabad	-	Member Convenor

MEMBERS OF THE INSTITUTIONAL BIO-SAFETY COMMITTEE

Dr DP Kasbekar Haldane Chair, CDFD, Hyderabad (Nominee of Director, CDFD)	-	Chairman
Dr Rupinder Kaur Staff Scientist, CDFD, Hyderabad	-	Member Secretary
Dr Ashwin B Dalal Staff Scientist, CDFD, Hyderabad	-	Biosafety Officer
Dr Murali Dharan Bashyam Staff Scientist, CDFD, Hyderabad	-	CDFD Expert
Dr Subhadeep Chatterjee Staff Scientist, CDFD, Hyderabad	-	CDFD Expert
Dr Ashok Khar Former Director, CMBRC, Appollo Hospitals Educational and Research Foundation	-	Outside Expert
Dr Manjula Reddy Senior Principal Scientist, CCMB, Hyderabad	-	DBT Nominee

MEMBERS OF THE INSTITUTIONAL BIOETHICS COMMITTEE

Prof G Manohar Rao Former Principal, PG College of Law, Osmania University, 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
Mrs Amita Kasbekar VP, Deloitte Consulting India Pvt. Ltd. RMZ, Hitech City, Hyderabad	-	Member
Dr Murali Bashyam Staff Scientist, CDFD, Hyderabad	-	Member
Dr Ashwin B Dalal Staff Scientist, CDFD, Hyderabad	-	Member Secretary

MEMBERS OF CDFD BUILDING COMMITTEE

Prof VS Chauhan Visiting Scientist, ICGEB, New Delhi	-	Chairman
Dr J Gowrishankar Director, CDFD, Hyderabad	-	Member
Joint Secretary DBT, New Delhi	-	Member
Shri VH Rao Hyderabad	-	Member
Shri J Sanjeev Rao Head-Administration, CDFD, Hyderabad	-	Member
Shri BJ Acharyulu Head-F&A, CDFD, Hyderabad	-	Member
Shri S Ayub Basha Staff Scientist-V (Engg), CDFD, Hyderabad	-	Member-Convenor

MEMBERS OF CDFD MANAGEMENT COMMITTEE

Dr J Gowrishankar Director, CDFD, Hyderabad	-	Chairman
Dr DP Kasbekar Haldane Chair, CDFD, Hyderabad	-	Member
Dr Rupinder Kaur Staff Scientist, CDFD, Hyderabad	-	Member (for a 2 year period)
Dr Subhadeep Chatterjee Staff Scientist, CDFD, Hyderabad	-	Member (for a 2 year period)
Shri BJ Acharyulu Head-F&A, CDFD, Hyderabad	-	Member
Shri J Sanjeev Rao Head-Administration, CDFD, Hyderabad	-	Member-Convenor

MEMBERS OF CDFD FINANCE COMMITTEE

Prof VS Chauhan Visiting Scientist, ICGEB, New Delhi	-	Chairman
Dr Dipankar Chatterji IISc, Bangalore	-	Member
Ms Anuradha Mitra JS&FA, DBT, New Delhi	-	Member
Dr Suman Govil Advisor, DBT, New Delhi	-	Member
Dr J Gowrishankar Director, CDFD, Hyderabad	-	Member
Shri BJ Acharyulu Head-F&A, CDFD, Hyderabad	-	Member Convenor

MEMBERS OF SEXUAL HARASSMENT COMPLAINTS COMMITTEE

Dr Sangita Mukhopadhyay Staff Scientist, CDFD, Hyderabad	-	Chairperson
Mr J Sanjeev Rao Head – Administration, CDFD, Hyderabad	-	Member
Ms V Naga Sailaja Technical Officer, CDFD, Hyderabad	-	Member
Ms MV Sukanya Technical Officer, CDFD, Hyderabad	-	Member
Mr MSA Zaman Khan Section Officer, CDFD, Hyderabad	-	Member
Ms P Jamuna Gramya Resource Centre for Women (representing an NGO)	-	Member

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

IMPLEMENTATION OF RIGHT TO INFORMATION (RTI) ACT, 2005

Appellate Authority : J Sanjeev Rao

Central Public Information Officer : M Kavita Rao

Details about the RTI applications and appeals received in CDFD

As received under RTI Act 2005	**Opening Balance as on 1.4.2014**	**Received during the year 2014-15**			**Disposed of during the year 2014-15**				**Closing Balance as on 31.3.2015**
		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/ appeals rejected	Transferred to other Public Authorities [u/s 6(3) of Act]	Total	
Applications	4	15	8	27	24	3	0	27	0
Appeals	0	2	Not applicable	2	1	1	Not applicable	2	0

बजट एवं वित्त
Budget and Finance

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS HYDERABAD

Budget & Finance 2014-15

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 2014-15

Particulars	Amount in Lakhs	Percentage - %
Plan Grant in Aid	4100.00	72.46
Sponsored Projects	1080.91	19.10
CDFD Services	164.82	2.91
Misc Receipts	312.67	5.53
Total	**5658.40**	**100.00**

I. Application of Funds during 2014-15 (Plan Grant in Aid)

S No	Particulars	Amount in Lakhs	Percentage- %
1	**Recurring**		
	GIA- Salaries	1239.38	28.72
	GIA-General	1604.49	37.18
	Total	**2843.87**	**65.90**
2	**Non-Recurring**		
	GIA- Capital	1471.79	34.10
	Total	**1471.79**	**34.10**
	Grand Total	**4315.66**	**100.00**

II. Application of Funds during 2014-15 (Extra Mural Projects)

S No	Particulars	Amount in Lakhs	Percentage- %
1	**Recurring**		
	Salaries	286.43	29.82
	General	579.09	60.29
	Total	**865.52**	**90.11**
2	**Non-Recurring**		
	GIA- Capital	94.97	9.89
	Total	**94.97**	**9.89**
	Grand Total	**960.49**	**100.00**

लेखा परिक्षक की रिपोर्ट
Auditor’s Report

K R Srinivasan & Co

Chartered Accountants

AUDITOR'S REPORT

Date: 15-05-2015

The Director,
Centre for DNA Fingerprinting and Diagnostics,
Nampally,
Hyderabad – 500 001

We have audited the attached Balance Sheet of CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, Hyderabad, as at 31st March 2015 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 2015 and

   b) In so far as it relates to the Income & Expenditure account excess of expenditure over income for the year ended on 31st March 2015.

for **K R Srinivasan & Co**
Chartered Accountants
[K R SRINIVASAN]

Place: Hyderabad
Date: 15/05/2015

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

BALANCE SHEET AS ON 31st MARCH 2015

(Amount - Rs.)

	Schedule	Current Year	Previous Year
CORPUS/CAPITAL FUND AND LIABILITIES			
Corpus / Capital Fund	1	1212702539	1169815289
Reserves and Surplus	2	0	0
Earmarked / Endowment funds	3	0	0
Secured Loans & Borrowings	4	0	0
Unsecured Loans & Borrowings	5	0	0
Deffered Credit Liabilities	6	0	0
Current Liabilities and Provisions	7	70028009	70814398
TOTAL		**1282730548**	**1240629687**
ASSETS			
Fixed Assets	8	1090185109	966768793
Investments- From Earmarked / Endowment Funds	9	35098273	19398273
Investments - Others	10	33593376	23131298
Current Assets, Loans, Advances etc.	11	123853790	231331323
Miscellaneous Expenditure		0	
TOTAL		**1282730548**	**1240629687**
Significant Accounting Policies	24		
Contingent Liabilities and Notes on Accounts	25		

DIRECTOR
CDFD

For K R SRINIVASAN & CO
CHARTERED ACCOUNTANTS
(K R SRINIVASAN)

HEAD - FINANCE & ACCOUNTS
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

INCOME & EXPENDITURE FOR THE YEAR ENDED 31st MARCH 2015 **(Amount - Rs.)**

INCOME	**Schedule**		**Current Year**		**Previous Year**
Income from Sales/Services	12		16481871		6408041
Grants/Subsides	13		260000000		250932400
Fees/Subscriptions	14		0		0
Income from Investments	15		27138910		23220086
Income from Royality, Publications etc.	16		0		0
Interest Earned	17		2104450		43238
Other Income	18		3609182		3620866
Increase/(decrease) in stock of Finished goods and works-in-progress	19		0		0
TOTAL (A)			**309334413**		**284224631**
EXPENDITURE					
Establishment Expenses	20		128443061		106712459
Administrative Expenses	21		207784973		177267101
Expenditure on Grants, Subsides etc.	22		0		0
Interest	23		0		0
Depreciation (Net Total at the year-end -corresponding to Schedule 8)		81320619		84513447	
Less:Transferred to Grants-in-Aid		81320619	0	84513447	
Provision For Salaries			8395162		3392051
TOTAL (B)			**344623196**		**287371611**
Balance being excess of Income over Expenditure (A-B)			**-35288783**		**-3146980**

DIRECTOR
CDFD

For K R SRINIVASAN & CO
CHARTERED ACCOUNTANTS
(K R SRINIVASAN)

HEAD - FINANCE & ACCOUNTS
CDFD

Transfer to Special Reserve (Specify each)					
Transfer to/from General Reserve					
BALANCE BEING SURPLUS/(DEFLICT) CARRIED TO CORPUS/CAPITAL FUND					
SIGNIFICANT ACOUNTING POLICIES	24				
CONTINGENT LIABILITIES AND NOTES ON ACCOUNTS	25				

DIRECTOR
CDFD

For K R SRINIVASAN & CO
CHARTERED ACCOUNTANTS
(K R SRINIVASAN)

HEAD - FINANCE & ACCOUNTS
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

RECEIPTS AND PAYMENTS ACCOUNT FOR THE YEAR ENDED 31st MARCH 2015

(Amount - Rs.)

RECEIPTS	Current Year	Previous Year	PAYMENTS	Current Year	Previous Year
1.Opening Balances			**1. Expenses**		
a) Cash in hand			a) Establishment Expenses (corresponding to Schedule 20)	128443061.00	106712459.00
b) Bank Balances			b) Administrative Expenses (corresponding to Schedule 21)	207784973.05	177267101.00
i) In current accounts	26417751.96	12223805.10	c) Schedule 22	0.00	0.00
ii) In deposit accounts					
iii) Savings accounts	4383078.10	20909457.77			
2. Grants Received			**2. Payments made against funds for various projects**		
a) From Government of India	410000000.00	340932400.00	(Name of the fund or project should be shown along with		
b) From State government			the particulars of payments made for each project)		
c) From other sources (details)			Projects (Annexure F)	96048982.00	106664828.00
(Grants for capital & revenue			CSIR(Stipend)	10088151.00	10743372.00
exp. To be shown seperately)			DBT(Stipend)	5571185.00	4534065.00
Research Associates - CSIR(Stipend)	11093876.00	5567737.00	DST(Stipend)	1340375.00	739200.00
Research Associates - DBT(Stipend)	5623475.00	5292736.00	ICMR(Stipend)	2785432.00	2901926.00
Research Associates - DST(Stipend)	85239.00	250400.00	IISC(Stipend)	813334.00	1578752.00
Research Associates - ICMR(Stipend)	1589055.00	2830106.00	UGC(Stipend)	8242741.00	5150772.00
Research Associates - IISC(Stipend)	1029961.00	424400.00			
Research Associates - UGC(Stipend)	16736506.00	0.00			
			3. Investments and deposits made		
Projects (Annexure - C)	108091285.00	74360025.00	a) Out of Earmarked/Endowement funds	189000000.00	203000000.00
			b) Out of Own Funds (Investments-Others)	0.00	

DIRECTOR
CDFD

For K R SRINIVASAN & CO
CHARTERED ACCOUNTANTS
(K R SRINIVASAN)

HEAD - FINANCE & ACCOUNTS
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

RECEIPTS AND PAYMENTS ACCOUNT FOR THE YEAR ENDED 31st MARCH 2013

(Amount - Rs.)

RECEIPTS	Current Year	Previous Year	PAYMENTS	Current Year	Previous Year
3. Income on Investments from					
a) Earmarked/Endow. Funds	9126414.00	23220086.49	**4. Expenditure on Fixed Assets & Capital Work-in-Progress**		
b) Own Funds (Oth. Investment)			a) Purchases of Fixed Assets:		
Investments EnCashed	162000000.00	246000000.00	Books & Journals	895458.00	562565.00
			Equipment -Lab/Office/Furniture	74499419.00	33772456.00
			b) Expenditure on Capital Work-in-Progress:	119845405.00	59875026.00
4. Interest Received					
a) On Bank deposits	0.00	0.00			
b) Loans, Advances etc			**5. Refund of surplus money/Loans**		
Interest on LC	2104449.40	43238.00	a) To the Government of India	0.00	
Interest on Computer Advance, Conveyance Advance and HBA	17526.00	12488.00	b) To the State Government	0.00	
			c) To other providers of funds	0.00	
5. Other Income(Specify)					
a) Analysis Charges	16481871.00	6408041.00	**6. Finance Charges (Interest)**	0.00	
6. Any Other Receipts(Give Details)			**7. Other Payments (Specify)**		
I-Remittances (Annexure-A)	23453753.00	20611562.00	Advances (Annexure-D)	172463825.00	55068123.00
			I-Remittances (Annexure-E)	23186242.00	19987907.00
CPF-SUB,Arrears and adv.Refund	10645544.00	12641274.70	CPF A/c	18257438.00	8630042.00
Sundry Receipts	3254256.00	3150268.00	New Pension Scheme	3136300.00	2935894.00
Application Fee	235800.00	384730.00			
Provident Fund Salwage	0.00	0.00	**8. Closing Balances**		
Free Gifts - Donations	0.00	0.00	a) Cash in hand		
Sale OF Tender Forms	47000.00	19500.00	b) Bank Balances		

DIRECTOR
CDFD

For K R SRINIVASAN & CO
CHARTERED ACCOUNTANTS
(K R SRINIVASAN)

HEAD - FINANCE & ACCOUNTS
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS,HYDERABAD

RECEIPTS AND PAYMENTS ACCOUNT FOR THE YEAR ENDED 31st MARCH 2013

(Amount - Rs.)

RECEIPTS	Current Year	Previous Year	PAYMENTS	Current Year	Previous Year
Leave Salary-Pension Contribution	0.00	0.00	i) In current accounts	13313616.81	26417751.96
License Fee	54600.00	53880.00	**ii) In deposit accounts**		
Welfare Fund	0.00	0.00	iii) Savings accounts	9433617.60	4383078.10
NPS	3040743.00	2935894.00			
Advance/Refunds/Recovery/Adj(Annexure-B)	269637372.00	52653289.00			
TOTAL	**1085149555.46**	**830925318.06**	**TOTAL**	**1085149555.46**	**830925318.06**

DIRECTOR
CDFD

For K R SRINIVASAN & CO
CHARTERED ACCOUNTANTS
(K R SRINIVASAN)

HEAD - FINANCE & ACCOUNTS
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
BALANCE SHEET AS ON 31st MARCH 2015

(Amount - Rs.)

		Current Year		Previous Year
SCHEDULE 1 - CORPUS/CAPITAL FUND :				
Balance as at the begining of the year		1169815289.00		1142536939.00
Add : Contribution towards Corpus/Capital Fund				
CDFD Core - Plan (Non-Recurring)	150000000.00		90000000.00	
Capitalised portion of Capital Expenditure of projects	9496652.00	159496652.00	24938777.00	114938777.00
Less : Depreciation For the Year 2014-2015		81320619.00		84513447.00
Less : Excess of Expenditure over Income		35288783.00		3146980.00
BALANCE AS AT THE YEAR - END		**1212702539.00**		**1169815289.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	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			0.00	
Addition during the year				
Less : Deductions during the year	0.00	0.00	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 2015

(Amount - Rs.)

		Current Year		Previous Year
SCHEDULE 3 - EARMARKED/ENDOWMENT FUNDS :				
(Refer Annexures)				
(a) Opening balance of the Funds		**-25773781.00**		**6531021.00**
(b) Additions to the Funds :				
i. Donations /grants	108091285.00		74360025.00	
ii. Income from investments made on account of funds	0.00		0.00	
iii. Other additions	0.00	108091285.00	0.00	74360025.00
TOTAL (a+b)		**82317504.00**		**80891046.00**
(c) Utilisation/Expenditure towards objective of funds				
(i) Capital Expenditure (Refer Annexures I & II)				
- Fixed Assets	9200996.00		24642024.00	
- Others	295656.00	9496652.00	296753.00	24938777.00
- Total				
(ii) Revenue Expenditure (Refer Annexures I & II)				
- Salaries, Wages and allowances etc.	28642978.00		31884970.00	
- Rent	0.00		0.00	
- Other Expenses	57909352.00	86552330.00	49841081.00	81726051.00
Total				
TOTAL (c)		96048982.00		106664828.00
NET BALANCE AS AT THE YEAR-END [(a + b)-c]		**-13731478.00**		**-25773782.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	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 accured and due	0	0	0	0
4. Banks :				
a) Terms Loans	0	0	0	0
- Interest accured and due	0		0	
b) Other Loans	0		0	
- Interest accured and due	0	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 2015

(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	0
5. Other Institutions and Agencies		0		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 2015

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 6 - DEFFERED 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 2015

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 7 - CURRENT LIABILITIES AND PROVISIONS :				
A. CURRENT LIABILITIES				
1. Acceptances				
2. Sundry Creditors				
3. Advances Received				
4. Interest accured but not due on:				
5. Statutory Liabilities:				
6. Other current Liabilities				
CDFD.CP Fund A/C(Annexure-G)	40638533.00		37788350.00	
Collaboration -Workshop Funds	0.00		11300000.00	
DG Set Maintenance [Advance]	0.00		42000.00	
EMD	2378534.00		2357734.00	
GSLI	30785.00		263362.00	
Honorarium [Advance]	0.00		8000.00	
House Building Advance	129831.00		129831.00	
Human Resource Develpment - Training of Staff - Conferences [Advance]	0.00		199000.00	
Income Tax	97088.00		57955.00	
Lab Security Deposit & Hostel Security Deposit	1242716.00		1170310.00	
LIC	2550.00		2550.00	
Medical [Advance]	0.00		238481.00	
Others (I-Remittances)	296555.00		269095.00	
Others [Maintenance Advance]	0.00		1000.00	
Out Standing Liabilities	11845456.00		8453405.00	
Postage-Courier [Advance]	0.00		1264.00	
PPF EMPLOYER SHARE	34566.00		0.00	
Professional Tax	99742.00		96927.00	
Public Provident Fund	124630.00		116345.00	
Royalty & Consultancy	1654142.00		2254142.00	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 7 - CURRENT LIABILITIES AND PROVISIONS :				
Scientific Workshops - Symposiums - Seminars [Advance]	0.00		25000.00	
Security Deposit	1691275.00		1803775.00	
Service Tax	247331.00		0.00	
TA Abroad [Advance]	65249.00		0.00	
TDS	800515.00		604383.00	
Works Tax	253349.00		239439.00	
Workshop & Conference	0.00	61632847.00	0.00	67422348.00
TOTAL (A)		**61632847.00**		**67422348.00**
B.PROVISIONS				
1. For Taxation				
2. Gratuity				
3. Superannuation/Pension				
4. Accumulated Leave Encashment				
5. Trade Warranties/Claims				
6. Others (Specify)	8395162.00	8395162.00		0
TOTAL (B)		**8395162.00**		**0**
TOTAL (A+B)		**70028009.00**		**67422348.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	GROSS BLOCK				DEPRECIATION				NET BLOCK	
	Cost/valuation As at begining of the the year	Addition during during the year	Deductions during the year	Cost/valuation at at the year end	As at the begining the year	On additions during the year	On Deductions during the year	Total up to the uo to the year end	As at the Current current year end	As at the prevous 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
b) Leasehold	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	47036194.00	25952426.00	0.00	72988620.00	147063749.00	173016175.00
b) On Leasehold Land	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
c) Ownership Flats/Premises	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
3. PLANT MACHINERY & EQUIPMENT	590733681.05	83549478.00	0.00	674283159.05	283882780.00	52879689.00	0.00	336762469.00	337520690.05	306850901.05
4. VEHICLES	4153026.00	0.00	0.00	4153026.00	3488747.00	99642.00	0.00	3588389.00	564637.00	664279.00
5. FURNITURE, FIXTURES	16458656.00	10906.00	0.00	16469562.00	10197477.00	630093.00	0.00	10827570.00	5641992.00	6261179.00
6. OFFICE EQUIPMENT	11567465.00	83851.00	0.00	11651316.00	8728288.00	432166.00	0.00	9160454.00	2490862.00	2839177.00
7. COMPUTER/PERIPHERALS	75843.00	56180.00	0.00	132023.00	0.00	0.00	0.00	0.00	132023.00	75843.00
8. ELECTRIC INSTALLATIONS	0.00				0.00	0.00	0.00	0.00		
9. LIBRARY BOOKS	16826120.00	1191114.00	0.00	18017234.00	16460997.00	1219652.00	0.00	17680649.00	336585.00	365123.00
10. TUBEWELLS & WATER SUPPLY	0.00				0.00	0.00	0.00	0.00		
11. OTHER FIXED ASSETS	8857898.00	0.00	0.00	8857898.00	7892048.00	106951.00	0.00	7998999.00	858899.00	965850.00
Airconditioning works		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		
DG Set		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		
Typewriters		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		
Other Assets		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		
TOTAL	872625058.05	84891529.00	0.00	957516587.05	377686531.00	81320619.00	0.00	459007150.00	498509437.05	494938527.05
B. CAPITAL WORK-IN-PROGRESS	471830266.70	119845405.00	0.00	591675671.70	0.00	0.00	0.00	0.00	591675671.70	471830266.70
TOTAL	**1344455324.75**	**204736934.00**	**0.00**	**1549192258.75**	**377686531.00**	**81320619.00**	**0.00**	**459007150.00**	**1090185108.75**	**966768793.75**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	Current Year	Previous Year
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)	35098273.00	19398273.00
TOTAL	**35098273.00**	**19398273.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	Current Year	Previous Year
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	33593376.00	23131298.00
TOTAL	**33593376.00**	**23131298.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 11 - INVESTMENTS - OTHERS :	**Current Year**		**Previous Year**	
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	
b) Others-Life Membership Fees	165935.00	165935.00	165935.00	165935.00
3. Cash balances in hand (including cheques/drafts and imprest)				
4. Bank Balances:				
a) With Scheduled Banks:				
-On Current Accounts	13313616.81		26417751.96	
-On Deposit Accounts (includes margin money)	0.00		0.00	
-On Savings Accounts	9433617.60	22747234.41	4383078.10	30800830.06
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)		**22913169.41**		**30966765.06**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	Current Year		Previous Year	
SCHEDULE 11 - INVESTMENTS - OTHERS :				
B. LOANS, ADVANCES AND OTHER ASSETS				
1. Loans:				
a) Staff	0.00		0.00	
b) Other Entities engaged in activities/objectives similar to that of the Entity	0.00	0.00	0.00	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)	51994904.06		151473164.51	
b) Prepayments - Deposits (Annexure-I)	17201742.00		23117612.00	
c) Others	0.00	69196646.06	0.00	174590776.51
3. Income Accured:				
a) On Investments from Earmarked/Endowments Funds	0.00		0.00	
b) On Investments - Others	18012496.00		0.00	
c) On Loans and Advances	0.00		0.00	
d) Others	0.00	18012496.00	0.00	0.00
4. Claims Receivable		13731478.00		25773782.00
TOTAL (B)		100940620.06		200364558.51
TOTAL (A+B)		**123853789.47**		**231331323.57**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	Current Year	Previous Year
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)	16481871.00	6408041.00
c) Agency Commission and Brokerage	0.00	0.00
d) Maintenance Services (Equpiment/Property)	0.00	0.00
e) Others (Specify)	0.00	0.00
TOTAL	**16481871.00**	**6408041.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 13 - GRANTS/SUBSIDES :		
(Irrevocable Grants & Subsides Received)		
1) Central Government (DBT Plan Grant-in-Aid)	260000000.00	250932400.00
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	**260000000.00**	**250932400.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	Current Year	Previous Year
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 2015

(Amount - Rs.)

	Current Year		Previous Year	
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	0.00	0.00
2) Dividends:				
a) On Shares	0.00	0.00	0.00	0.00
b) On Mutual Fund Securities	0.00	0.00	0.00	0.00
3) Rents	0.00	0.00	0.00	0.00
4) Others (Specify) STDRs	27138910.00		23220086.00	0.00
TOTAL	**27138910.00**		**23220086.00**	**0.00**
TRANSFERRED TO EARMARKED/ENDOWMENT FUNDS				

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 16 - INCOME FROM ROYALITY, PUBLICATION ETC. :		
1) Income from Royality	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 2015

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 17 - INTEREST EARNED :		
1) On Term Deposits		
a) With Schedule Banks	2104449.88	43238.00
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	0.00	0.00
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		
b) Others	0.00	0.00
4) Interest on Debtors and Other Receivables	0.00	0.00
TOTAL	**2104449.88**	**43238.00**
Note :- Tax deducted at source to be indicated		

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

SCHEDULE 18 - OTHER INCOME	Current Year	Previous Year
1) Profit on Sale/disposal of Assets:	0.00	0.00
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		
5) Other Receipts		
Sundry Receipts	3254256.00	3150268.00
Application Fee	235800.00	384730.00
Sales Of Tender Forms	47000.00	19500.00
Licence Fee	54600.00	53880.00
Interest On Computer Advance,Conveyance Advance And HBA	17526.00	12488.00
Leave Salary-Pension Contribution	0.00	0.00
Provident Fund Salwage	0.00	0.00
Free.Gifts-Donations	0.00	0.00
TOTAL	**3609182.00**	**3620866.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

SCHEDULE 19 - INCREASE/(DECREASE) IN STOCK OF FINISHED GOODS & WORK IN PROGRESS :	Current Year	Previous Year
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 2015

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 20 - ESTABLISHMENT EXPENSES :		
a) Salaries and Wages	68828459.00	54269773.00
b) Allowances and Bonus	50691650.00	41267382.00
c) Contribution to Provident Fund	2619770.00	3213621.00
d) Contribution to Other Fund (NPS)	2358636.00	1736649.00
e) Staff Welfare Expenses - Medical charges	2136167.00	2195107.00
f) Expenses on Employees Retirement and Terminal Benefits	1808379.00	4029927.00
g) Others (specify) - Staff leased House	0.00	0.00
TOTAL	**128443061.00**	**106712459.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 21 - OTHER ADMINISTRATIVE EXPENSES :		
a) Purchases	77276637.48	54146570.00
b) Electricity and power	21857964.00	20703811.00
c) Water charges	898347.00	592058.00
d) Insurance	90857.00	106691.00
e) Repairs and maintenance	16452976.00	13737055.00
f) Rent, Rates and Taxes	18919374.00	18691350.00

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 21 - OTHER ADMINISTRATIVE EXPENSES :		
g) Vehicles Running and Maintenance	1254153.00	949931.00
h) Postage, Telephone and Communication Charges	3037666.00	2198082.00
i) Printing and Stationary	1151024.00	1701402.00
j) Travelling and Conveyance Expenses	9897639.57	9099650.00
k) Expenses on Seminar/Workshops	316177.00	654385.00
l) Subscription Expenses	38693.00	60872.00
m) Expenses on Fees	80874.00	322746.00
n) Auditors Remuneration	56180.00	71326.00
o) Hospitality Expenses	772072.00	826450.00
p) Professional Charges	5985002.00	3722520.00
q) Advertisement and Publicity	3034697.00	2821705.00
r) Bank Charges	4818.00	14931.00
s) Security & Cleaning Contract Charges	21011830.00	18839558.00
t) Training Course /Symposia	-88482.00	211800.00
u) Other Contingencies	1881362.00	1502085.00
v) Liveries & Blankets	30819.00	102830.00
w) Other Research Expenses	22011273.00	26140605.00
x)Office Books	13020.00	48688.00
y)Over Heads	1800000.00	0.00
TOTAL	**207784973.05**	**177267101.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS
SCHEDULES FORMING PART OF BALANCE SHEET AS AT 31st MARCH 2015

(Amount - Rs.)

	Current Year	Previous Year
SCHEDULE 22 - EXPENDITURE ON GRANTS, SUBSIDES, 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 2015

(Amount - Rs.)

	Current Year	Previous Year
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 31/03/2015

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	Head- Finance & Accounts CDFD	for K R Srinivasan & Co Chartered Accountants [K R SRINIVASAN]

Place: Hyderabad
Date: 15/05/2015

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

CLARIFICATION ON NOTES ON ACCOUNTS: 2014-15

- 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.

B J ACHARYULU
Head Finance & Accounts
CDFD

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

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

For the Year Ended 31st MARCH 2015

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
-13869143	COE1	COE1	-13242813
-23581573	COE2	COE2	-13991880
-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 raective oxygen species on T-Cell immune response: An approach to understand the molecular mechanism of immunosuppression during tuberculosis - National Bioscience Award	-576590
3727878	P-101	Role of inositol pyrophosphates in cell physiology: Investigating the biochemical significance of protein pyrophosphorylation - Senior Fellowship	6859801
-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
-3307223	P-104	Virtual Centre of Excellence on Epigenetics	-1160508
-862685	P-105	Cloning, Characterization and analysis of chromosomal rearrangements in human genetic disorders	-862685
-227909	P-106	Clinical, Biochemical and molecular analysis of treatable lysosomal storage disorders	0
15400	P-107	IYBA Project - Mechanism and role of bacterial cell-cell signaling molecules in plant defense response	1036691
-454643	P-108	Establishment of EBV transformed cell lines from families with rare genetic disorders	-454643
57690	P-109	Molecular dissection of PI3-Kinase/Akt pathway by suing proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors	3351336
-191391	P-110	India-Japan research project title"Identification and analysis of sex determining genes in silkmoths"	-191391
450416	P-111	Ramalingaswami Fellowship - Refractoriness mechanism in Mosquito: cracking molecular codes at genomic scale	1169677
-450859	P-114	Evaluating the Calcineurin-NFAT Pathway and its regulators superoxide dismutase (SOD) AND RCAN1 (regular 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 esophaeal cancer	-2892
-1474723	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
4377125	P-122	Understanding the role of Hox genes in anterior-posterior axis determination of the central nervous system	388692
513310	P-123	Establish a Max Planck Partner Group for Genetic Diversity Studies at CDFD	1402135
-549916	P-124	Preparation and characterization of peroxometal compounds and studies and their biological significance in cellular signalling	-748411
172619	P-125	Mechanistic studies on the role of protein kinase Snfilk in cell cycle and cancer	0
35390	P-126	Rho-dependent transcription termination machinery: mechanism of action	442524
283993	P-127	Systematic studies on the functional network of phosphatases in cell life and death	-294516
-608942	P-128	Mechanism of iron acquisition and iron homeostasis in an opportunistic human pathogen Candida glabrata	-77108
6737	P-13	"Programme to delineate gene functions in the post – genomics era by a systematic two gene knockout method"	3947
2865531	P-130	Comparative genetic analysis of sex chromosomes and sex determining genes in silkmoths	-2550050

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

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

For the Year Ended 31st MARCH 2015

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
-1245339	P-131	Structural and functional studies of Acyl CoA Binding proteins from plasmodium falciparum	398632
-2166471	P-132	Characterization of tumor suppressor function of ARIDIB, a component of the human SWI/SNF chromatin remodelling complex	-640003
534614	P-133	Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster	460117
-156437	P-134	Exploration of wild silk moth biodiversity in Manipur and their genetic characterization using molecular markers	-77061
-298323	P-135	Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Phthogen Interaction in TB Infection	-357268
13618	P-136	Raf Kinase - a key target for modem-day theraphy against tumors	-292334
44141	P-137	Signaling pathways involved in down regulation of proinflammatory responses by PPE18 protein of Mycobacterium tuberculosis: Implication of PPE18 as therapeutics	759474
-638079	P-138	Co-evaluation of Dnmt3l and Genomic imprinting	-1353238
20000	P-139	Evaluating the role of Sirtuins and epigenetic changes during cellular senescense in context of p53 status	20000
146091	P-140	Development of baculovirus resistant silkworms strains through synthetic miRNA based knockdown of essential viral genes	-403336
-223537	P-141	Evaluating the functional role of PTEN interacting proteins in cell survival signaling and tumor suppression	-125000
-401878	P-142	Identification of H3K4 TRI Demethylase involved in erasing H3K4 trimethylation marks at E2F Responsive promoters	-280596
-751303	P-143	Microarray based characterisation of squamous cell carcinoma of the tongue occuring in non smokers	-534504
0	P-144	Tri-National Training Program for Psychiatric Genetics	424130
-1064782	P-145	"H3K4 HMT family regulatescell cycle progression	-1112243
763439	P-146	"Role of MLL in ribosomal RNA transcription	433858
41311	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	-677839
270865	P-149	"Role of SUMOylation in the pathobiology of Candida Glabrata	-1016335
-28096	P-150	"Genetic and genomic basis of the evolution of bombycid and sturniid silkmoths	0
594981	P-151	"Human Exome Sequencing to Identify Novel Genes for Medelian Disorders	-601366
1114145	P-152	"Global transcriptomics of sex specific spilicing	29100
3613562	P-153	"An attractive and promising stragegy for early cancer diagnosis through the assembly of the human cancer volatome""	641552
87432	P-154	"Rational design, synthetic strategies for developing organometallic anticancer compounds based on organotin and organoiron	30832
335194	P-155	"Studies on thecellular roles of calcium signalling proteins in Neurospora crassa	335194
926632	P-156	"Targeting microbial quorum sensing to demonstrate potential application of cell-cell signaling molecules from Xanthomonas group of plant pathogen in diesease control	-175165
944665	P-157	"Identification of novel antifungal drug and delineation of drug resistance mechanisms in an opportunistic human fungal pathogen Candida glabrata	204372
621787	P-158	"Modulation of host immune responses by a PPE Protein of Mycobacterium tuberculosis: Understanding its role in host - pathogen cross-talk	-1379658
300000	P-159	"Gene Targeting of microbial isolates to demonstrate potential plant growth promoting (PGP) traits by third generation sequencing	0
363884	P-160	"Understanding the role of novel adhesins of Xanthomonas oryzae PV orzae in Virulence and colonization in Rice	208333
350000	P-161	"Analysis of co-regulation between DNA replication activity and amino acid homeostatis by transcription factor IciA/ArgP in Eschericia coli	84656
235671	P-162	Characterization and design of inhibitors of Mycobacterium tuberculosis transcription	-316464
2006048	P-163	Unravelling new functions for the H-NS family of proteins in Gram-negative bacterial pathogens	1052471

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

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

For the Year Ended 31st MARCH 2015

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
-26671	P-164	"A Yeast based screen for discovery of novel sirtuin inhibitors as anticancer agents	-24671
1569682	P-165	"Identification and functional characterization of immune response genes in silkmoths	330135
0	P-166	"Sequencing analysis of transcriptome variants in early-onset sporadic rectal cancer	2165638
0	P-167	"To elucidate the role of MLL complex in epigenetic specification of centromeres	633780
0	P-168	"A Search for nucleus -limited genes in Neurospora	788623
0	P-169	"Implementation of 3 year DNB Program in Medical Genetics by Department of Biotechnology in colloboration with National Board of Examination ag SGHR, NIBMG&CDFD	1758108
-687887	P-17	“Studies on inosital-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV” – Transfer from IMTECH, Chandigarh	-687887
0	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""	277449
0	P-171	"Role of vesicle-mediated transport and chromatin remodelling in the virulence of Candida glabrata	1754447
0	P-172	"Molecular Characterization of early onset sporadic rectal cancer	1461747
0	P-173	"Development and application of a next generation sequencing approach for molecular genetic analysis of lysosomal storage disorders	584882
0	P-174	"Is non-canonical Wnt signalling a major player in early-onset sporadic rectal cancer	500000
0	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""	-509714
0	P-176	International Atomic Energy Agency	200103
-274286	P-18	“Mapping of receptor binding site on the Eythrocyte binding of malaria parasyte”	-274286
-1888111	P-20	“Genomic Micro array R&D Programmes on infectious diseases and Neurological Disorders”	-1888111
0.5	P-22	“Biotechnology for leather – towards cleaner processing”	0.5
-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 protien 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
2045696	P-30	Transcription termination and anti termination in E-coli	2045696
746453	P-31	Role of K-ras in Lung type II epithelial cells	746453
-234000	P-33	“Molecular and Epidemiological characterisation of cryptosporidium – An enteric protozoon parasite”	-234000
26334	P-34	“Molecular analysis of lepidopteran – specific immune protiens from silkmoths”	26334
-283883	P-35	“Identification, Characterization and Physical mapping of Z-Chromosome linked genes of the silk worm, Bombyxmori”	-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
-2237285	P-42	“Structural and functional studies on Mycobacterium tuberculosis heat shock proteins”.	-2237285
685906.7	P-43	“A generalized mechanism of transcription termination in prokaryotes: a quest for mechanism based transcription inhibitors for microbial pathogens”.	685906.7
-457538	P-44	“Understanding of role of Ras and NO / iNOS signalling in promotion of hepatocellular carcinomas with persistent HBV infection”	-457538
605714	P-45	Specialized chromatin structures as epigenetic imprints to distinguish parental alleles”.	605714
-1586965	P-47	Research cum Training for DRDO Programme	-1586965

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

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

For the Year Ended 31st MARCH 2015

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
151826	P-48	'Molecular characterization of human liver stem cells for use in the treatment of hepatic diseases'.	151826
804660	P-49A	International Atomic Energy Agency (IAEA)	1041952
-284065	P-51	"Understanding the mechanism of doxorubicin resistance in breast cancer celline MCF-7"	-284065
-1231118	P-52	"Nucleo Cytoplasmic transport of HIV – 1 Vpr"	-1231118
-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 nucleoied protein H-NS"	-280000
-278928	P-62	"HIV – 1 Pathogenesis: Role of Integrase in Reverse Transciption and Nuclear Transport of Viral Genome"	-278928
-837574	P-63	"Upgradation of the existing computing infrastructure at the Bioinformatics facility at CDFD"	-837574
-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 Helicobater pylori"	-582647
20617169	P-65A	APEDA-CDFD Centre for Basmati DNA Analysis	21828405
-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
463453	P-81A	Financial assistance for award of J C Bose Fellowship to Dr J Gowrishankar	62620
-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

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-I

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

For the Year Ended 31st MARCH 2015

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
-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 silkmoths	-65698
218818	P-88	Introduction of anti-baculoviral property in commercial silkworm strains by expression of multiple RNAi viral targets	0
-636286	P-90	Role of Yapsins in the Pathobiology of Candida Glabrata	-636286
-1098900	P-91	DMMT3L: epigenetic correlation with cancer	-1098900
268823	P-92	Swarnajayanti fellowship proj on "Designing transcription anti-terminators: a novel approach for making new inhibitors of gene expression"	268823
-605745	P-93/ A1	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against tuberculosis	-611833
-2469833	P-93/ A2	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis	-3025061
0	P-93B2 (II)	Evaluation of peptides / small molecules targeting ESAT-6:B2M interaction and PPE18-TLR2 interaction as potent anti tuberculosis therapautics	1110000
-276552	P-97	Proteome-wide Analysis of Serine pyrophosphorylation by inositol pyrophosphates	-276552
-203419	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
-25773781.8			**-13731478.8**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-II

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

For the Year Ended 31st MARCH 2015

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
11713327	COE-I	COE for Genetics and Genomics of silkmoths	11713327
10000000	COE-II	DBT Centre of Excellence for Microbial Biology	10156100
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 raective oxygen species on T-Cell immune response: An approach to understand the molecular mechanism of immunosuppression during tuberculosis - National Bioscience Award	17784
12024311	P-101	Role of inositol pyrophosphates in cell physiology: Investigating the biochemical significance of protein pyrophosphorylation - Senior Fellowship	13084732
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
915278	P-109	Molecular dissection of PI3-Kinase/Akt pathway by suing proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors	915968
206800	P-111	Ramalingaswami Fellowship - Refractoriness mechanism in Mosquito: cracking molecular codes at genomic scale	206800
0	P-112	Ramanujan Fellowship	0
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 (regular 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
9889367	P-122	Understanding the role of Hox genes in anterior-posterior axis determination of the central nervous system	10824792
540436	P-123	Establish a Max Planck Partner Group for Genetic Diversity Studies at CDFD	1022127
402016	P-126	Rho-dependent transcription termination machinery: mechanism of action	591694
6281319	P-127	Systematic studies on the functional network of phosphatases in cell life and death	6755620
1609427	P-128	Mechanism of iron acquisition and iron homeostasis in an opportunistic human pathogen Candida glabrata	1690360
1334600	P-13	"Programme to delineate gene functions in the post – genomics era by a systematic two gene knockout method"	1334600
81500	P-130	Comparative genetic analysis of sex chromosomes and sex determining genes in silkmoths	81500
964215	P-133	Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster	1018512

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-II

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

For the Year Ended 31st MARCH 2015

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
5500000	P-135	Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Phthogen Interaction in TB Infection	5500000
130979	P-137	Signaling pathways involved in down regulation of proinflammatory responses by PPE18 protein of Mycobacterium tuberculosis: Implication of PPE18 as therapeutics	815232
565518	P-138	Co-evaluation of Dnmt3l and Genomic imprinting	565518
500000	P-139	Evaluating the role of Sirtuins and epigenetic changes during cellular senescense in context of p53 status	500000
5163243	P-14	“Comparitive and functional genomics approaches for the identification and characterization of genes responsible for multi drug resistance of mycobacterium tuberculosis”	5163243
0	P-140	Development of baculovirus resistant silkworms strains through synthetic miRNA based knockdown of essential viral genes	500000
624495	P-142	Identification of H3K4 TRI Demethylase involved in erasing H3K4 trimethylation marks at E2F Responsive promoters	651933
1546279	P-145	"H3K4 HMT family regulatescell cycle progression	1868000
686219	P-146	"Role of MLL in ribosomal RNA transcription	1000000
468720	P-149	"Role of SUMOylation in the pathobiology of Candida Glabrata	468720
6000000	P-15	“The Helicobacter Pylori genome programme – Genome sequencing, functional analysis and comparitive genomics of the strains obtained from Indian patients”	6000000
0	P-153	"An attractive and promising stragegy for early cancer diagnosis through the assembly of the human cancer volatome""	3000000
0	P-154	"Rational design, synthetic strategies for developing organometallic anticancer compounds based on organotin and organoiron	132495
0	P-155	"Studies on thecellular roles of calcium signalling proteins in Neurospora crassa	0
0	P-156	"Targeting microbial quorum sensing to demonstrate potential application of cell-cell signaling molecules from Xanthomonas group of plant pathogen in diesease control	0
380852	P-157	"Identification of novel antifungal drug and delineation of drug resistance mechanisms in an opportunistic human fungal pathogen Candida glabrata	992265
0	P-158	"Modulation of host immune responses by a PPE Protein of Mycobacterium tuberculosis: Understanding its role in host - pathogen cross-talk	299941
1814901	P-16	NMITLI Project on – Latent M.Tuberculosis: New targets, Drug delivery systems, Bio enhancers & Therapeutics	1814901
0	P-167	"To elucidate the role of MLL complex in epigenetic specification of centromeres	39304
0	P-168	"A Search for nucleus -limited genes in Neurospora	31450
244400	P-17	“Studies on inosital-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV” – Transfer from IMTECH, Chandigarh	244400
344020	P-18	“Mapping of receptor binding site on the Eythrocyte binding of malaria parasyte”	344020
7246511	P-19	“Construction of Integrated RAPD, RFLP and Microsatellite linkage map of the Silkworm, Bombyx mori and its corelation 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 protien X (VPX)”	600000
500000	P-26	Occurrence of Mutations in Non dividing cells of Escherichia Coli”	500000

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-II

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

For the Year Ended 31st MARCH 2015

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
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 protien(s) interacting with macrophase 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 Transciption and Nuclear Transport of Viral Genome"	4308314
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 Helicobater pylori"	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

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS Annexure-II

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

For the Year Ended 31st MARCH 2015

(Amount in Rs.)

Previous year	Proj No	Particulars	Current Year
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
195728	P-81A	Financial assistance for award of J C Bose Fellowship to Dr J Gowrishankar	205073
1480220	P-82	Functional genomic analysis of Candida Glabrata-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 Candida Glabrata	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
840648	P-93/ A2	Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis	900000
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
2783795	P-98	Role of cell - cell signaling mediated by Diffusible signaling factor (DSF) in Xanthomonas virulence	2816418
2963482	P-99	Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosomae biogenesis	2963482
289524651			**299021303**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	I-Remittances	
4872379.00	TDS	5410533.00
6930770.00	Income Tax	7678934.00
4751.00	Works Tax	13910.00
1501203.00	LIC	1732202.00
450115.00	GSLI	275017.00
2201735.00	Public Provident Fund	2686575.00
568281.00	Professional Tax	573726.00
2739240.00	Service Tax	3453615.00
1142963.00	Others (I-Remittances)	998280.00
200125.00	Health Insurance	411095.00
0.00	CCS	185300.00
0.00	PPF EMPLOYER SHARE	34566.00
20611562.00		**23453753.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Advance refunds/recovery/Adjst.	
432548.00	Advance for purchases by Staff	478737.00
34547.00	AMC for Equipment [Advance]	255558.00
0.00	CDFD Staff reserve Fund	0.00
25209032.00	Chemicals [Advance]	54643035.00
83449.00	Computer Advance [Research Fellows]	70453.00
35900.00	Computer Advance [Staff]	85330.00
33400.00	Consumables, glassware and Spares [Advance]	3123522.00
47140.00	Conveyance Advance	80600.00
42000.00	DG Set Maintenance [Advance]	0.00
339200.00	EMD	168000.00
13093454.00	Equipment [Advance]	76669827.00
103105.00	Festival Advance	132375.00

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.
1999431.00	General Deposits And Advances	5915870.00
23000.00	Honorarium [Advance]	0.00
34744.00	House Building Advance	0.00
199000.00	Human Resource Develpment - Training of Staff - Conferences [Advance]	0.00
0.00	Inter Bank Transfer	120836854.00
141500.00	Lab Security Deposit & Hostel Security Deposit	174000.00
864513.00	LTC [Advance]	1358506.00
538481.00	Medical [Advance]	0.00
28090.00	Other Research Expenses [Advance]	9166.00
405179.00	Others [Advances]	304927.00
1000.00	Others [Maintenance Advance]	0.00
1264.00	Postage-Courier [Advance]	0.00
319669.00	Revolving Advance	440208.00
0.00	Royalty & Consultancy	0.00
25000.00	Scientific Workshops - Symposiums - Seminars [Advance]	0.00
95300.00	Security Deposit	30000.00
157412.00	TA Abroad [Advance]	1266313.00
4352911.00	TA With in India [Advance]	2024892.00
13000.00	Trainee Security Deposit	12000.00
4000000.00	Workshop & Conference	1557199.00
52653269.00		**269637372.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Projects - Receipts	
3814000.00	COE1/CORE	9102000.00
750000.00	COE1/P-I	732000.00
643000.00	COE1/P-II	459000.00
1009000.00	COE1/P-III	1090000.00
0.00	COE2-II/P-1	2186000.00
0.00	COE2-II/P-A	1093000.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
0.00	COE2-II/P-B	500000.00
0.00	COE2-II/P-C	1093000.00
0.00	COE2-II/P-D	500000.00
0.00	COE2-II/P-E	1093000.00
0.00	COE2-II-Core	11236000.00
450000.00	COE-I/P-IV	463000.00
6230314.00	P-101	9098800.00
457596.00	P-102	0.00
300000.00	P-103	0.00
0.00	P-104	2898000.00
0.00	P-106	227909.00
0.00	P-107	1854000.00
0.00	P-109	5056000.00
1490000.00	P-111	1635000.00
1419047.00	P-113	0.00
0.00	P-114	0.00
0.00	P-115	0.00
1328000.00	P-119	0.00
0.00	P-120	828000.00
4986110.00	P-122	1213195.00
1203108.00	P-123	2449811.00
1374000.00	P-125	0.00
1780400.00	P-126	1433700.00
6910824.00	P-127	4990612.00
0.00	P-128	807800.00
4300000.00	P-130	0.00
1768900.00	P-131	1902500.00
0.00	P-132	3046200.00
981000.00	P-133	867000.00
425000.00	P-134	235000.00
2057700.00	P-135	2371000.00
759000.00	P-136	570000.00
473256.00	P-137	2500000.00
0.00	P-138	0.00
520000.00	P-139	520000.00
394000.00	P-140	835000.00
300000.00	P-141	600000.00
211000.00	P-142	935920.00
0.00	P-143	1144199.00
0.00	P-144	424130.00
0.00	P-145	1870600.00
872000.00	P-146	809000.00

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.
500000.00	P-147	0.00
0.00	P-148	0.00
1059500.00	P-149	0.00
0.00	P-150	153846.00
0.00	P-151	0.00
2872300.00	P-152	2562571.00
937000.00	P-153	621000.00
1030000.00	P-154	943000.00
0.00	P-155	0.00
2104400.00	P-156	1076500.00
2760800.00	P-157	1317000.00
1933141.00	P-158	0.00
300000.00	P-159	0.00
382000.00	P-160	531649.00
350000.00	P-161	0.00
799600.00	P-162	0.00
2006048.00	P-163	0.00
0.00	P-164	188000.00
1569682.00	P-165	0.00
0.00	P-166	4383200.00
0.00	P-167	1700000.00
0.00	P-168	1400000.00
0.00	P-169	1890000.00
0.00	P-170	820000.00
0.00	P-171	2415730.00
0.00	P-172	2100000.00
0.00	P-173	699782.00
0.00	P-174	500000.00
0.00	P-176	200103.00
0.00	P-40	0.00
496299.00	P-49A	237292.00
1062000.00	P-65A	1211236.00
1360000.00	P-81A	1360000.00
0.00	P-83A	0.00
0.00	P-88	0.00
4000000.00	P-92	0.00
645000.00	P-93/A1	0.00
985000.00	P-93/A2	0.00
0.00	P-93B2 (II)	1110000.00
0.00	P-99	0.00
74360025.00		**108091285.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	Advances	
340050.00	Advance for purchases by Staff	538638.00
0.00	AMC for Equipment [Advance]	251855.00
6938893.00	Chemicals [Advance]	4139900.00
32400.00	Computer Advance [Research Fellows]	168592.00
90000.00	Computer Advance [Staff]	270000.00
10610261.00	Consumables, glassware and Spares [Advance]	9467022.00
180768.00	Conveyance Advance	30000.00
0.00	DG Set Maintenance [Advance]	42000.00
880000.00	EMD	147200.00
23577545.00	Equipment [Advance]	28608232.00
600700.00	GDA [Others]	0.00
124875.00	Festival Advance	161250.00
10000.00	General Deposits And Advances	0.00
10000.00	Honorarium [Advance]	8000.00
0.00	Human Resource Develpment - Training of Staff - Conferences [Advance]	199000.00
0.00	Inter Bank Transfer	120836854.00
127000.00	Lab Security Deposit & Hostel Security Deposit	101594.00
31000.00	Liveries & Blankets [Advance]	99351.00
1417120.00	LTC [Advance]	1519510.00
0.00	Medical [Advance]	238481.00
9166.00	Other Research Expenses [Advance]	0.00
1023456.00	Others [Advances]	6230.00
0.00	Others [Maintenance Advance]	1000.00
0.00	Postage-Courier [Advance]	1264.00
370500.00	Revolving Advance	392500.00
598.00	Royalty & Consultancy	600000.00
0.00	Scientific Workshops - Symposiums - Seminars [Advance]	25000.00
0.00	Security Deposit	142500.00
614715.00	TA Abroad [Advance]	743761.00
3847364.00	TA With in India [Advance]	1731760.00
13500.00	Trainee Security Deposit	11000.00
4218212.00	Workshop & Conference	1981331.00
55068123.00		**172463825.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	I-Remittances	
0.00	CCS	185300.00
231143.00	GSLI	507594.00
334610.00	Health Insurance	558782.00
6910170.00	Income Tax	7639801.00
1501203.00	LIC	1732202.00
1052853.00	Others (I-Remittances)	970820.00
0.00	PPF EMPLOYER SHARE	0.00
570541.00	Professional Tax	570911.00
1999815.00	Public Provident Fund	2678290.00
2640829.00	Service Tax	3128141.00
4746743.00	TDS	5214401.00
0.00	Works Tax	0.00
19987907.00		**23186242.00**

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	
8440715.00	COE1/CORE	8700539.00
687200.00	COE1/P-I	637866.00
409739.00	COE1/P-II	491226.00
964758.00	COE1/P-III	1059200.00
8433772.00	COE2/CORE	4606321.00
0.00	COE2/P-1	0.00
552410.00	COE2/P-2	343200.00
612089.00	COE2/P-A	269100.00
612089.00	COE2/P-B	269100.00
553032.00	COE2/P-C	0.00
0.00	COE2-II/P-1	114735.00
0.00	COE2-II/P-A	289700.00
0.00	COE2-II/P-B	200000.00
0.00	COE2-II/P-C	289700.00
0.00	COE2-II/P-E	16774.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
0.00	COE2-II-Core	1712677.00
387200.00	COE-I/P-IV	330839.00
0.00	P-100	0.00
6866703.00	P-101	5966877.00
55498.00	P-102	0.00
0.00	P-103	0.00
1289348.00	P-104	751285.00
17739.00	P-105	0.00
38698.00	P-106	0.00
-14965.00	P-107	832709.00
61678.00	P-108	0.00
36736.00	P-109	1762354.00
0.00	P-110	0.00
1590000.00	P-111	915739.00
382293.00	P-113	0.00
0.00	P-114	0.00
-5.00	P-115	0.00
0.00	P-116	0.00
198263.00	P-119	0.00
874505.00	P-120	122761.00
0.00	P-121	0.00
13698667.00	P-122	5201628.00
1841767.00	P-123	1560986.00
0.00	P-124	198495.00
720400.00	P-125	172619.00
1059582.00	P-126	1026566.00
10789369.00	P-127	5569121.00
1146713.00	P-128	275966.00
0.00	P-129	0.00
0.00	P-13	2790.00
1900442.00	P-130	5415581.00
2245570.00	P-131	258529.00
937991.00	P-132	1519732.00
1415875.00	P-133	941497.00
440000.00	P-134	155624.00
7732589.00	P-135	2429945.00
823362.00	P-136	875952.00
1114135.00	P-137	1784667.00
1542023.00	P-138	715159.00
1723583.00	P-139	520000.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
804000.00	P-140	1384427.00
525000.00	P-141	501463.00
973026.00	P-142	814638.00
897587.00	P-143	927400.00
0.00	P-144	0.00
3272988.00	P-145	1918061.00
920770.00	P-146	1138581.00
774331.00	P-147	719150.00
20326.00	P-148	0.00
2558921.00	P-149	1287200.00
192802.00	P-150	125750.00
1398219.00	P-151	1196347.00
1758155.00	P-152	3647616.00
323438.00	P-153	3593010.00
942568.00	P-154	999600.00
1177768.00	P-156	2178297.00
1816135.00	P-157	2057293.00
1311354.00	P-158	2001445.00
0.00	P-159	300000.00
18116.00	P-160	687200.00
0.00	P-161	265344.00
563929.00	P-162	552135.00
0.00	P-163	953577.00
26671.00	P-164	186000.00
0.00	P-165	1239547.00
0.00	P-166	2217562.00
0.00	P-167	1066220.00
0.00	P-168	611377.00
0.00	P-169	131892.00
0.00	P-170	542551.00
0.00	P-171	661283.00
0.00	P-172	638253.00
0.00	P-173	114900.00
0.00	P-175	509714.00
0.00	P-49A	0.00
179652.00	P-65A	0.00
0.00	P-71	0.00
1459167.00	P-81A	1760833.00
1300.00	P-82	0.00
0.00	P-88	218818.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
640922.00	P-92	0.00
589291.00	P-93/A1	6088.00
1007836.00	P-93/A2	555228.00
129682.00	P-97	0.00
-52425.00	P-98	32623.00
251736.00	P-99	0.00
106664828.00		**96048982.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	CDFD C.P.F ACCOUNT	
35805402.00	Opening Balance	37788349.37
	Add:	
4801908.00	Employee subscription/ refunds	5433264.00
0.00	Transfer from other departments	0.00
0.00	Institute contribution (inc. Projects staff)	0.00
980582.00	Interest received	208230.00
3799542.00	Less Advances/withdrawals/Transfer/Adjst	2791310.00
37788350.00		**40638533.37**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	LOANS AND ADVANCES	
146340.50	Advance for purchases by Staff	206241.50
4310.00	Advances [Previous Years]	4310.00
3703.00	AMC for Equipment [Advance]	0.00
61056531.00	Chemicals [Advance]	10553396.00
16860.00	Computer Advance [Research Fellows]	114999.00
142600.00	Computer Advance [Staff]	327270.00

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
11291561.00	Consumables, glassware and Spares [Advance]	17635061.00
178248.00	Conveyance Advance	127648.00
0.00	DA	6638.00
48332459.45	Equipment [Advance]	270864.00
75300.00	Festival Advance	104175.00
134485.00	Health Insurance	282172.00
0.00	Honorarium [Advance]	0.00
31000.00	Liveries & Blankets [Advance]	130351.00
2524960.00	LTC [Advance]	2685964.00
0.00	Miscellaneous Salary	30843.00
0.00	NPS Subscription	95557.00
22700.00	Office Equipment [Advance]	22700.00
9166.00	Other Research Expenses [Advance]	0.00
5951565.00	Others [Advances]	5652868.00
0.00	Pay of Establishment	53387.00
0.00	Public Provident Fund	0.00
304569.00	Rent [Advance]	304569.00
19751667.00	Research Fellows-Associates	12343905.00
153174.00	Revolving Advance	105466.00
78143.00	Service Tax	0.00
457303.00	TA Abroad [Advance]	0.00
563968.56	TA With in India [Advance]	270836.56
27500.00	Trainee Security Deposit	26500.00
215051.00	Workshop & Conference	639183.00
151473164.51		**51994904.06**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	DEPOSITS	
22381635.00	General Deposits And Advances	16465765.00
735977.00	GDA[Others]	735977.00
23117612.00		**17201742.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	INVESTMENT A/C	
8098273.00	Investments	35098273.00
11300000.00	Other Investments	0.00
19398273.00		**35098273.00**

CENTER FOR DNA FINGERPRINTING AND DIAGNOSTICS
FOR THE YEAR ENDED 31st MARCH 2015

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

Previous Year Amount Rs.	Particulars	Current Year Amount Rs.
	CDFD C.P.F INVESTMENT A/C	
25159583.00	Deposit with Banks	33131298.00
4830500.00	Employee subscription	5466128.00
6858785.00	Less Transfer To Bank A/C	5004050.00
23131298.00		**33593376.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-03: "Transgenesis and Genetic basis of Pathogen Resistance in the Silkworm, Bombyx Mori
P.I:
Receipts and Payments Account from 01/04/2014 to 31/03/2015

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
0.0		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**	**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"
P.I: Dr Seyed E Hasnain
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
244305.00	Opening Balance	244305.00		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	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
2443050.00		**244305.00**	**244305.00**		**244305.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-10: "Role of upstream sequence elements in Hyper activation of transcription from Baculovirus polyhedrin gene promoter"

P.I: Dr M D Bashyam

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	28332.00	Opening Balance	28332.00
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	Transfer of Funds	0.00
0.00		**0.00**	**28332.00**		**28332.00**
28332.00	Excess of Expenditure over Income	28332.00	0.00	Closing Balance	0.00
28332.00		**28332.00**	**28332.00**		**28332.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-13: "Programme to delineate gene functions in the post – genomics era by a systematic two gene knockout method"

P.I: Dr J Gowrishankar

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
6737.00	Opening Balance	6737.00		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	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
6737.00		**6737.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	6737.00	Closing Balance	6737.00
6737.00		**6737.00**	**6737.00**		**6737.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-17: "Studies on inosital-phosphate synthesis – a novel enzyme from Mycobacterium tuberculosis H37RV" – Transfer from IMTECH, Chandigarh
P.I: Dr Sekhar C Mande
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
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**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-18: "Mapping of receptor binding site on the Eythrocyte binding of malaria parasyte"
P.I: Dr Akash Ranjan
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		529111.00		Opening Balance	529111.00	
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**		**529111.00**			**529111.00**	
529111.00		Excess of Expenditure over Income	529111.00		0.00		Closing Balance	0.00	
529111.00			**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:
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		79533.00		Opening Balance	79533.00	
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**		**79533.00**			**79533.00**	
79533.00		Excess of Expenditure over Income	79533.00		0.00		Closing Balance	0.00	
79533.00			**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 01/04/2014 to 31/03/2015

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 01/04/2014 to 31/03/2015

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-30: Transcription termination and anti termination in E-coli
P.I: Dr Ranjan Sen
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
2045696.00	Opening Balance	2045696.00		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	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
2045696.00		**2045696.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	2045696.00	Closing Balance	2045696.00
2045696.00		**2045696.00**	**2045696.00**		**2045696.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-31: Role of K-ras in Lung type II epithelial cells
P.I: Dr Gayatri Ramakrishna
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
746453.00	Opening Balance	746453.00		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	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
746453.00		**746453.00**	**0.00**		**0.00**
0.00	Excess of Expenditure over Income	0.00	746453.00	Closing Balance	746453.00
746453.00		**746453.00**	**746453.00**		**746453.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-33: "Molecular and Epidemiological characterisation of cryptosporidium – An enteric protozoon parasite"
P.I: Dr Radha Rama Devi
Receipts and Payments Account from 01/04/2014 to 31/03/2015

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 protiens from silkmoths"
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2014 to 31/03/2015

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		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	
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**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-35: "Identification, Characterization and Physical mapping of Z-Chromosome linked genes of the silk worm, Bombyxmori"
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-36: "Development of Artificial retina using Bacterio rhodospin and genetically engineered analogues "
P.I: Dr Sekhar C Mande
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
2073896.00		Opening Balance	2073896.00				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		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**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
1873605.00		Opening Balance	1873605.00				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		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**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-42: "Structural and functional studies on Mycobacterium tuberculosis heat shock proteins".
P.I: Dr Sekhar C Mande
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		2237285.00		Opening Balance	2237285.00	
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**		**2237285.00**			**2237285.00**	
2237285.00		Excess of Expenditure over Income	2237285.00		0.00		Closing Balance	0.00	
2237285.00			**2237285.00**		**2237285.00**			**2237285.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-43: "A generalized mechanism of transcription termination in prokaryotes: a quest for mechanism based transcription inhibitors for microbial pathogens".
P.I: Dr Ranjan Sen
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
685906.70		Opening Balance	685906.70						
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	
685906.70			**685906.70**		**0.00**			**0.00**	
0.00		Excess of Expenditure over Income	0.00		685906.70		Closing Balance	685906.70	
685906.70			**685906.70**		**685906.70**			**685906.70**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-45: Specialized chromatin structures as epigenetic imprints to distinguish parental alleles".
P.I: Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
605714.00		Opening Balance	605714.00				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		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	
605714.00			**605714.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure over Income	0.00		605714.00		Closing Balance	605714.00	
605714.00			**605714.00**		**605714.00**			**605714.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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**	

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 01/04/2014 to 31/03/2015

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		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	
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**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-49A: International Atomic Energy Agency (IAEA)
P.I: J Nagaraju
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
308361.00		Opening Balance	804660.00				Opening Balance		0.00
496299.00		Grant In Aid	237292.00			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
804660.00			**1041952.00**			**0.00**			**0.00**
0.00		Excess of Expenditure Over Income	0.00		804660.00		Closing Balance	1041952.00	
804660.00			**1041952.00**		**804660.00**			**1041952.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-51: "Understanding the mechanism of doxorubicin resistance in breast cancer celline MCF-7"
P.I: Dr Sunil Kumar Manna
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
224.00		Opening Balance		224.00					
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
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**

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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**

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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount 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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
482124.00	Opening Balance	482124.00			
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
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 nucleoied protein H-NS"

P.I: Dr Abhijit A Sardesai

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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 Transciption and Nuclear Transport of Viral Genome"

P.I: Dr S Mahalingam

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		837574.00		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**		**837574.00**			**837574.00**	
837574.00		Excess of Expenditure over Income	837574.00		0.00		Closing Balance	0.00	
837574.00			**837574.00**		**837574.00**			**837574.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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 Helicobater pylori"
P.I: Dr Ayesha Alvi
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount 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
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
			0.00	Opening Balance	0.00
19734821.00	**Opening Balance**	20617169.00	179652.00	Salaries- Manpower	179652.00
0.00	Grant in aid	0.00	0.00	Consumables	0.00
1062000.00	Basmati Analysis Charges	1211236.00	0.00	Contingencies	0.00
			0.00	Travel	0.00
			0.00	Overheads	0.00
			0.00	Consultancy & Knowledge fee	0.00
			0.00	Vehicle	0.00
			0.00	Equipment	0.00
20796821.00		21828405.00	179652.00		179652.00
	Excess of expenditure over income		20617169.00	Closing Balance	21648753.00
20796821.00		**21828405.00**	**20796821.00**		**21828405.00**

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

P.I: Dr Sanjeev Khosla

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		681246.00		Opening Balance	681246.00	
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**		**681246.00**			**681246.00**	
681246.00		Excess of Expenditure over Income	681246.00		0.00		Closing Balance	0.00	
681246.00			**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

P.I: Dr M D Bashyam

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		113545.00		Opening Balance	113545.00	
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**		**113545.00**			**113545.00**	
113545.00		Excess of Expenditure over Income	113545.00		0.00		Closing Balance	0.00	
113545.00			**113545.00**		**113545.00**			**113545.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
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**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	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 cpy number alterations
P.I: Dr M D Bashyam
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	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 01/04/2014 to 31/03/2015

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 01/04/2014 to 31/03/2015

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 01/04/2014 to 31/03/2015

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		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	
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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
1304.00		Opening Balance	1304.00				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		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**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-79: Understanding the role of AGE proteins in inducing inflammatory responses and its regulation
P.I: Dr S K Manna
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-80: Referral centre for detection of genetically modified foods employing DNA-based markets
P.I: Dr Madhusudan Reddy
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
143470.00		Opening Balance	143470.00						
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	
143470.00			**143470.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure over Income	0.00		143470.00		Closing Balance	143470.00	
143470.00			**143470.00**		143470.**00**			**143470.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
562620.00		Opening Balance	463453.00				Opening Balance	0.00	
1360000.00		Grant In Aid	1360000.00		300000.00		Salaries - Manpower	300000.00	
0.00			0.00		1000000.00		Consumables	962116.50	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		99167.00		Travel	429371.50	
0.00			0.00		60000.00		Overheads	60000.00	
0.00			0.00		0.00		Equipment	9345.00	
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	
1922620.00			**1823453.00**		**1459167.00**			**1760833.00**	
0.00		Excess of Expenditure Over Income	0.00		463453.00		Closing Balance	62620.00	
1922620.00			**1823453.00**		**1922620.00**			**1823453.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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 1**		**118755.00**			**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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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-88: Introduction of anti-baculoviral property in commercial silkworm strains by expression of multiple RNAi viral targets
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
218818.00	Opening Balance	218818.00		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	218818.00
218818.00		**218818.00**	**0.00**		**218818.00**
0.00	Excess of Expenditure Over Income	0.00	218818.00	Closing Balance	0.00
218818.00		**218818.00**	**218818.00**		**218818.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
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: DMMT3L: epigenetic correlation with cancer
P.I: Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
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**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-92: Swarnajayanti fellowship proj on "Designing transcription anti-terminators: a novel approach for making new inhibitors of gene expression"
P.I: Dr Ranjan Sen
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	268823.00		3090255.00		Opening Balance	0.00	
4000000.00		Grant In Aid	0.00		410922.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	0.00	
0.00			0.00		30000.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		200000.00		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	
4000000.00			**268823.00**		**3731177.00**			**0.00**	
0.00		Excess of Expenditure Over Income	0.00		268823.00		Closing Balance	268823.00	
4000000.00			**268823.00**		**4000000.00**			**268823.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-93/A1 : Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against tuberculosis
P.I.: Dr Shekar
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance		0.00	661454.00		Opening Balance	605745.00	
645000.00		Grant In Aid		0.00	589291.00		Salaries - Manpower	6088.00	
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	
645000.00				**0.00**	**1250745.00**			**611833.00**	
605745.00		Excess of Expenditure Over Income	611833.00		0.00		Closing Balance	0.00	
1250745.00			**611833.00**		**1250745.00**			**611833.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-93/A2 : Virtual Centre of Excellence on multidisciplinary approaches aimed at interventions against Mycobacterium tuberculosis
P.I.: Dr. Sangita Mukhopadhyay
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance		0.00	2446997.00		Opening Balance	2469833.00	
985000.00		Grant In Aid		0.00	817732.00		Salaries - Manpower	495876.00	
0.00				0.00	0.00		Consumables	0.00	
0.00				0.00	0.00		Contingencies	0.00	
0.00				0.00	4859.00		Travel	0.00	
0.00				0.00	0.00		Overheads	0.00	
0.00				0.00	185245.00		Equipment	59352.00	
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	
985000.00				**0.00**	**3454833.00**			**3025061.00**	
2469833.00		Excess of Expenditure Over Income	3025061.00		0.00		Closing Balance	0.00	
3454833.00			**3025061.00**		**3454833.00**			**3025061.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-93B2 (II) : Evaluation of peptides / small molecules targeting ESAT-6:B2M interaction and PPE18-TLR2 interaction as potent anti tuberculosis therapautics

P.I.: Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		1110000.00		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			**1110000.00**		**0.00**			**0.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		1110000.00
	0.00			**1110000.00**		**0.00**			**1110000.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00		146870.00	Opening Balance		276552.00
	0.00	Grant In Aid		0.00		96284.00	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		33398.00	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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	255844.00	Opening Balance	203419.00
0.00	Grant In Aid	0.00	-46800.00	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	-5625.00	Equipment	32623.00
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**	**203419.00**		**236042.00**
203419.00	Excess of Expenditure Over Income	236042.00	0.00	Closing Balance	0.00
203419.00		**236042.00**	**203419.00**		**236042.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-99: Role of inositol Pyrophosphates in eukaryotic cell growth, proliferation and ribosomae biogenesis
P.I: Dr Rashna Bhandari
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	315780.00	Opening Balance	567516.00
0.00	Grant In Aid	0.00	251736.00	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**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-100: Effect of raective 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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount 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-101: Role of inositol pyrophosphates in cell physiology: Investigating the biochemical significance of protein pyrophosphorylation - Senior Fellowship

P.I: Dr Rashna Bhandari

Receipts and Payments Account from 01/04/2014 to 31/03/205

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
4364267.00	Opening Balance	3727878.00		Opening Balance	0.00
6230314.00	Grant In Aid	9098800.00	1440715.00	Salaries - Manpower	1227962.00
0.00		0.00	3409058.00	Consumables	2883561.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	22013.00	Travel	268246.00
0.00		0.00	615900.00	Overheads	526687.00
0.00		0.00	1379017.00	Equipment	887105.00
0.00		0.00	0.00	Books	173316.00
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
10594581.00		**12826678.00**	**6866703.00**		**5966877.00**
0.00	Excess of Expenditure Over Income	0.00	3727878.00	Closing Balance	6859801.00
10594581.00		**12826678.00**	**10594581.00**		**12826678.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-102: "Understanding the role of Mycobacterium tuberculosis heat shockprotein 60 as Th1/Th2 immuno modular"
P.I: Dr Sangita Mukhopadhyay
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		430020.00		Opening Balance	27922.00	
457596.00		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		19043.00		Travel	0.00	
0.00			0.00		19026.00		Overheads	0.00	
0.00			0.00		17429.00		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	
457596.00			**0.00**		**485518.00**			**27922.00**	
27922.00		Excess of Expenditure Over Income	27922.00		0.00		Closing Balance	0.00	
485518.00			**27922.00**		**485518.00**			**27922.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-103: National Bioscience Award - Regulation of mast cell signaling, apoptosis and surface receptors
P.I: Dr Sunil Kumar Manna
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		600000.00		Opening Balance	300000.00	
300000.00		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	
300000.00			**0.00**		**600000.00**			**300000.00**	
300000.00		Excess of Expenditure Over Income	300000.00		0.00		Closing Balance	0.00	
600000.00			**300000.00**		**600000.00**			**300000.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-104: Virtual Centre of Excellence on Epigenetics
P.I: Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		2017875.00		Opening Balance	3307223.00	
0.00		Grant In Aid	2898000.00		354407.00		Salaries - Manpower	403779.00	
0.00			0.00		884941.00		Consumables	220853.00	
0.00			0.00		50000.00		Contingencies	100000.00	
0.00			0.00		0.00		Travel	26653.00	
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			**2898000.00**		**3307223.00**			**4058508.00**	
3307223.00		Excess of Expenditure Over Income	1160508.00		0.00		Closing Balance	0.00	
3307223.00			**4058508.00**		**3307223.00**			**4058508.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-105: Cloning, Characterization and analysis of chromosomal rearrangements in human genetic disorders
P.I: Dr Ashwin B Dalal
Receipts and Payments Account from 01/04/2014 to 31/03/2015

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		17739.00		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-106: Clinical, Biochemical and molecular analysis of treatable lysosomal storage disorders
P.I: Dr Ashwin B Dalal
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		189211.00		Opening Balance	227909.00	
0.00		Grant In Aid	227909.00		38698.00		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			**227909.00**		**227909.00**			**227909.00**	
227909.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	0.00	
227909.00			**227909.00**		**227909.00**			**227909.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
435.00		Opening Balance	15400.00				Opening Balance	0.00	
0.00		Grant In Aid	1854000.00		78000.00		Salaries - Manpower	232709.00	
0.00			0.00		-74153.00		Consumables	600000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		-18812.00		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	
435.00			**1869400.00**		**-14965.00**			**832709.00**	
0.00		Excess of Expenditure Over Income	0.00		15400.00		Closing Balance	1036691.00	
435.00			**1869400.00**		**435.00**			**1869400.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		392965.00		Opening Balance	454643.00	
0.00		Grant In Aid	0.00		42774.00		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		18904.00		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 suing proteomics based approach: A study to identify novel potential oncogenes and tumor suppressors
P.I: Dr M Subba Reddy
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
94426.00		Opening Balance	57690.00				Opening Balance	0.00	
0.00		Grant In Aid	5056000.00		36736.00		Salaries - Manpower	211664.00	
0.00			0.00		0.00		Consumables	1550000.00	
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	690.00	
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	
94426.00			**5113690.00**		**36736.00**			**1762354.00**	
0.00		Excess of Expenditure Over Income	0.00		57690.00		Closing Balance	3351336.00	
94426.00			**5113690.00**		**94426.00**			**5113690.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-110: India-Japan research project title"Identification and analysis of sex determining genes in silkmoths"
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		191391.00		Opening Balance	191391.00	
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**		**191391.00**			**191391.00**	
191391.00		Excess of Expenditure Over Income	191391.00		0.00		Closing Balance	0.00	
191391.00			**191391.00**		**191391.00**			**191391.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-111: Ramalingaswami Fellowship - Refractoriness mechanism in Mosquito: cracking molecular codes at genomic scale
P.I: Dr Shweta Tyagi
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
550416.00		Opening Balance	450416.00				Opening Balance	0.00	
1490000.00		Grant In Aid	1635000.00		1182684.00		Salaries - Manpower	247950.00	
0.00			0.00		200516.00		Consumables	650767.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		0.00		Travel	17022.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		206800.00		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	
2040416.00			**2085416.00**		**1590000.00**			**915739.00**	
0.00		Excess of Expenditure Over Income	0.00		450416.00		Closing Balance	1169677.00	
2040416.00			**2085416.00**		**2040416.00**			**2085416.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-113: Clinical and molecular genetic analysis of squamous cell carcinoma of the tongue
P.I: Dr M D Bashyam
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance		0.00	1036754.00		Opening Balance		0.00
1419047.00		Grant In Aid		0.00	372663.00		Salaries - Manpower		0.00
0.00				0.00	0.00		Consumables		0.00
0.00				0.00	4000.00		Contingencies		0.00
0.00				0.00	0.00		Travel		0.00
0.00				0.00	5630.00		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
1419047.00				**0.00**	**1419047.00**				**0.00**
0.00		Excess of Expenditure Over Income		0.00	0.00		Closing Balance		0.00
1419047.00				**0.00**	**1419047.00**				**0.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-114: Evaluating the Calcineurin-NFAT Pathway and its regulators superoxide dismutase (SOD) AND RCAN1 (regular of Calcineurin) Down Syndrome
P.I: Dr Gayatri Ramakrishna, Dr Ashwin Dalal
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

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

P.I: Dr Gayatri Ramakrishna

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	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**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-119: Analysis of DNA copy number alterations in esophaeal cancer

P.I: Dr M D Bashyam

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance		0.00	1132629.00		Opening Balance	2892.00	
1328000.00		Grant In Aid		0.00	198263.00		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	
1328000.00				**0.00**	**1330892.00**			**2892.00**	
2892.00		Excess of Expenditure Over Income	2892.00		0.00		Closing Balance	0.00	
1330892.00			**2892.00**		**1330892.00**			**2892.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		600218.00		Opening Balance	1474723.00	
0.00		Grant In Aid	828000.00		205400.00		Salaries - Manpower	92761.00	
0.00			0.00		619105.00		Consumables	0.00	
0.00			0.00		50000.00		Contingencies	30000.00	
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			**828000.00**		**1474723.00**			**1597484.00**	
1474723.00		Excess of Expenditure Over Income	769484.00		0.00		Closing Balance	0.00	
1474723.00			**1597484.00**		**1474723.00**			**1597484.00**	

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 01/04/2014 to 31/03/2015

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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
13089682.00	Opening Balance	4377125.00		Opening Balance	0.00
4986110.00	Grant In Aid	1213195.00	1207355.00	Salaries - Manpower	939806.00
0.00		0.00	1839406.00	Consumables	2798321.00
0.00		0.00	0.00	Contingencies	30454.00
0.00		0.00	22013.00	Travel	24747.00
0.00		0.00	1199850.00	Overheads	472875.00
0.00		0.00	9430043.00	Equipment	935425.00
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
18075792.00		**5590320.00**	**13698667.00**		**5201628.00**
0.00	Excess of Expenditure Over Income	0.00	4377125.00	Closing Balance	388692.00
18075792.00		**5590320.00**	**18075792.00**		**5590320.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1151969.00	Opening Balance	513310.00		Opening Balance	0.00
1203108.00	Grant In Aid	2449811.00	438409.00	Salaries - Manpower	339509.00
0.00		0.00	1016274.00	Consumables	480492.00
0.00		0.00	100000.00	Contingencies	100000.00
0.00		0.00	199743.00	Travel	159294.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	87341.00	Equipment	481691.00
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
2355077.00		**2963121.00**	**1841767.00**		**1560986.00**
0.00	Excess of Expenditure Over Income	0.00	513310.00	Closing Balance	1402135.00
2355077.00		**2963121.00**	**2355077.00**		**2963121.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	549916.00	Opening Balance	549916.00
0.00	Grant In Aid	0.00	0.00	Salaries - Manpower	109200.00
0.00		0.00	0.00	Consumables	89295.00
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**	**549916.00**		**748411.00**
549916.00	Excess of Expenditure Over Income	748411.00	0.00	Closing Balance	0.00
549916.00		**748411.00**	**549916.00**		**748411.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-125: Mechanistic studies on the role of protein kinase Snfilk in cell cycle and cancer
P.I: Dr M Subba Reddy
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	172619.00	480981.00	Opening Balance	0.00
1374000.00	Grant In Aid	0.00	220400.00	Salaries - Manpower	-10800.00
0.00		0.00	500000.00	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	183419.00
1374000.00		**172619.00**	**1201381.00**		**172619.00**
0.00	Excess of Expenditure Over Income	0.00	172619.00	Closing Balance	0.00
1374000.00		**172619.00**	**1374000.00**		**172619.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-126: Rho-dependent transcription termination machinery: mechanism of action
P.I: Dr Ranjan Sen
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	35390.00	685428.00	Opening Balance	0.00
1780400.00	Grant In Aid	1433700.00	298393.00	Salaries - Manpower	302538.00
0.00		0.00	704577.00	Consumables	513978.00
0.00		0.00	40000.00	Contingencies	0.00
0.00		0.00	0.00	Travel	20372.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	16612.00	Equipment	189678.00
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
1780400.00		**1469090.00**	**1745010.00**		**1026566.00**
0.00	Excess of Expenditure Over Income	0.00	35390.00	Closing Balance	442524.00
1780400.00		**1469090.00**	**1780400.00**		**1469090.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-127: Systematic studies on the functional network of phosphatases in cell life and death
P.I: Dr M Subba Reddy
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
4162538.00	Opening Balance	283993.00		Opening Balance	0.00
6910824.00	Grant In Aid	4990612.00	620441.00	Salaries - Manpower	776984.00
0.00		0.00	5827231.00	Consumables	3758682.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	3700.00	Travel	63992.00
0.00		0.00	953874.00	Overheads	495162.00
0.00		0.00	3087370.00	Equipment	351961.00
0.00		0.00	296753.00	Books	122340.00
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
11073362.00		**5274605.00**	**10789369.00**		**5569121.00**
0.00	Excess of Expenditure Over Income	294516.00	283993.00	Closing Balance	0.00
11073362.00		**5569121.00**	**11073362.00**		**5569121.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
537771.00		Opening Balance	0.00				Opening Balance	608942.00	
0.00		Grant In Aid	807800.00		185732.00		Salaries - Manpower	185407.00	
0.00			0.00		900000.00		Consumables	0.00	
0.00			0.00		30000.00		Contingencies	0.00	
0.00			0.00		15947.00		Travel	9626.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		15034.00		Equipment	80933.00	
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	
537771.00			**807800.00**		**1146713.00**			**884908.00**	
608942.00		Excess of Expenditure Over Income	77108.00		0.00		Closing Balance	0.00	
1146713.00			**884908.00**		**1146713.00**			**884908.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-130: Comparative genetic analysis of sex chromosomes and sex determining genes in silkmoths
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
465973.00		Opening Balance	2865531.00				Opening Balance	0.00	
4300000.00		Grant In Aid	0.00		699263.00		Salaries - Manpower	783258.00	
0.00			0.00		1000000.00		Consumables	4450000.00	
0.00			0.00		100000.00		Contingencies	50000.00	
0.00			0.00		19679.00		Travel	132323.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		81500.00		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	
4765973.00			**2865531.00**		**1900442.00**			**5415581.00**	
0.00		Excess of Expenditure Over Income	2550050.00		2865531.00		Closing Balance	0.00	
4765973.00			**5415581.00**		**4765973.00**			**5415581.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		768669.00		Opening Balance	1245339.00	
1768900.00		Grant In Aid	1902500.00		311665.00		Salaries - Manpower	212529.00	
0.00			0.00		1861029.00		Consumables	0.00	
0.00			0.00		50000.00		Contingencies	46000.00	
0.00			0.00		22876.00		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	
1768900.00			**1902500.00**		**3014239.00**			**1503868.00**	
1245339.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	398632.00	
3014239.00			**1902500.00**		**3014239.00**			**1902500.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-132: Characterization of tumor suppressor function of ARIDIB, a component of the human SWI/SNF chromatin remodelling complex
P.I: Dr M D Bashyam, Dr Rohit Joshi
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		1228480.00		Opening Balance	2166471.00	
0.00		Grant In Aid	3046200.00		400113.00		Salaries - Manpower	429347.00	
0.00			0.00		484566.00		Consumables	1068571.00	
0.00			0.00		20000.00		Contingencies	0.00	
0.00			0.00		33312.00		Travel	21814.00	
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			**3046200.00**		**2166471.00**			**3686203.00**	
2166471.00		Excess of Expenditure Over Income	640003.00		0.00		Closing Balance	0.00	
2166471.00			**3686203.00**		**2166471.00**			**3686203.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-133: Investigating the role of Hox gene deformed in central nervous system patterning in Drosophila melanogaster

P.I: Dr Rohit Joshi

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
969489.00	Opening Balance	534614.00		Opening Balance	0.00
981000.00	Grant In Aid	867000.00	326452.00	Salaries - Manpower	287200.00
0.00		0.00	600000.00	Consumables	567124.00
0.00		0.00	0.00	Contingencies	8000.00
0.00		0.00	0.00	Travel	24876.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	489423.00	Equipment	54297.00
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
1950489.00		**1401614.00**	**1415875.00**		**941497.00**
0.00	Excess of Expenditure Over Income	0.00	534614.00	Closing Balance	460117.00
1950489.00		**1401614.00**	**1950489.00**		**1401614.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-134: Exploration of wild silk moth biodiversity in Manipur and their genetic characterization using molecular markers

P.I: Dr K P Arun Kumar

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	0.00	141437.00	Opening Balance	156437.00
425000.00	Grant In Aid	235000.00	0.00	Salaries - Manpower	0.00
0.00		0.00	400000.00	Consumables	119000.00
0.00		0.00	25000.00	Contingencies	30000.00
0.00		0.00	15000.00	Travel	6624.00
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
425000.00		**235000.00**	**581437.00**		**312061.00**
156437.00	Excess of Expenditure Over Income	77061.00	0.00	Closing Balance	0.00
581437.00		**312061.00**	**581437.00**		**312061.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-135: Sys TB: A Network Program for Resolving the Intracellular Dynamics of Host Phthogen Interaction in TB Infection
P.I: Dr. Sanjeev Kholsa
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
5376566.00	Opening Balance	0.00		Opening Balance	298323.00
2057700.00	Grant In Aid	2371000.00	274929.00	Salaries - Manpower	343200.00
0.00		0.00	1885265.00	Consumables	2000000.00
0.00		0.00	50000.00	Contingencies	50000.00
0.00		0.00	22395.00	Travel	36745.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	5500000.00	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
7434266.00		**2371000.00**	**7732589.00**		**2728268.00**
298323.00	Excess of Expenditure Over Income	357268.00	0.00	Closing Balance	0.00
7732589.00		**2728268.00**	**7732589.00**		**2728268.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-136: Raf Kinase - a key target for modem-day theraphy against tumors
P.I: Dr Sunil Kumar Manna
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
77980.00	Opening Balance	13618.00		Opening Balance	0.00
759000.00	Grant In Aid	570000.00	187200.00	Salaries - Manpower	187200.00
0.00		0.00	606162.00	Consumables	626858.00
0.00		0.00	0.00	Contingencies	30000.00
0.00		0.00	0.00	Travel	31894.00
0.00		0.00	30000.00	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
836980.00		**583618.00**	**823362.00**		**875952.00**
0.00	Excess of Expenditure Over Income	292334.00	13618.00	Closing Balance	0.00
836980.00		**875952.00**	**836980.00**		**875952.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-137: Signaling pathways involved in down regulation of proinflammatory responses by PPE18 protein of Mycobacterium tuberculosis: Implication of PPE18 as therapeutics

P.I: Dr Sangita Mukhopadhyay

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
685020.00	Opening Balance	44141.00		Opening Balance	0.00
473256.00	Grant In Aid	2500000.00	79733.00	Salaries - Manpower	224180.00
0.00		0.00	750000.00	Consumables	696860.00
0.00		0.00	53423.00	Contingencies	34577.00
0.00		0.00	0.00	Travel	44797.00
0.00		0.00	100000.00	Overheads	100000.00
0.00		0.00	130979.00	Equipment	684253.00
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
1158276.00		**2544141.00**	**1114135.00**		**1784667.00**
0.00	Excess of Expenditure Over Income	0.00	44141.00	Closing Balance	759474.00
1158276.00		**2544141.00**	**1158276.00**		**2544141.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-138: Co-evaluation of Dnmt3l and Genomic imprinting

P.I: Dr Sanjeev Khosla

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
903944.00	Opening Balance	0.00		Opening Balance	638079.00
0.00	Grant In Aid	0.00	151505.00	Salaries - Manpower	186160.00
0.00		0.00	800000.00	Consumables	500000.00
0.00		0.00	25000.00	Contingencies	25000.00
0.00		0.00	0.00	Travel	3999.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	565518.00	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
903944.00		**0.00**	**1542023.00**		**1353238.00**
638079.00	Excess of Expenditure Over Income	1353238.00	0.00	Closing Balance	0.00
1542023.00		**1353238.00**	**1542023.00**		**1353238.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-139: Evaluating the role of Sirtuins and epigenetic changes during cellular senescense in context of p53 status
P.I: Dr Gayatri Ramakrishna, Dr Sanjeev Khosla
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1223583.00	Opening Balance	20000.00		Opening Balance	0.00
520000.00	Grant In Aid	520000.00	109200.00	Salaries - Manpower	0.00
0.00		0.00	520173.00	Consumables	500000.00
0.00		0.00	20000.00	Contingencies	20000.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	500000.00	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	574210.00	Transfer of Funds	0.00
1743583.00		**540000.00**	**1723583.00**		**520000.00**
0.00	Excess of Expenditure Over Income	0.00	20000.00	Closing Balance	20000.00
1743583.00		**540000.00**	**1743583.00**		**540000.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
556091.00	Opening Balance	146091.00		Opening Balance	0.00
394000.00	Grant In Aid	835000.00	204000.00	Salaries - Manpower	284427.00
0.00		0.00	600000.00	Consumables	583701.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	16299.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	500000.00
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
950091.00		**981091.00**	**804000.00**		**1384427.00**
0.00	Excess of Expenditure Over Income	403336.00	146091.00	Closing Balance	0.00
950091.00		**1384427.00**	**950091.00**		**1384427.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1463.00	Opening Balance	0.00		Opening Balance	223537.00
300000.00	Grant In Aid	600000.00	0.00	Salaries - Manpower	0.00
0.00		0.00	425000.00	Consumables	431463.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	100000.00	Overheads	70000.00
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
301463.00		**600000.00**	**525000.00**		**725000.00**
223537.00	Excess of Expenditure Over Income	125000.00	0.00	Closing Balance	0.00
525000.00		**725000.00**	**525000.00**		**725000.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
360148.00	Opening Balance	0.00		Opening Balance	401878.00
211000.00	Grant In Aid	935920.00	173445.00	Salaries - Manpower	187200.00
0.00		0.00	600000.00	Consumables	600000.00
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	199581.00	Equipment	27438.00
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
571148.00		**935920.00**	**973026.00**		**1216516.00**
401878.00	Excess of Expenditure Over Income	280596.00	0.00	Closing Balance	0.00
973026.00		**1216516.00**	**973026.00**		**1216516.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-143: Microarray based characterisation of squamous cell carcinoma of the tongue occuring in non smokers
P.I: Dr M D Bashyam
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
146284.00		Opening Balance		0.00			Opening Balance		751303.00
0.00		Grant In Aid		1144199.00		247587.00	Salaries - Manpower		231400.00
0.00				0.00		650000.00	Consumables		696000.00
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
146284.00			**1144199.00**		**897587.00**			**1678703.00**	
751303.00		Excess of Expenditure Over Income		534504.00		0.00	Closing Balance		0.00
897587.00			**1678703.00**		**897587.00**			**1678703.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-144 : Tri-National Training Program for Psychiatric Genetics
P.I: Dr Ashwin B Dalal
Receipts and Payments Account from 01/04/2013 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance		0.00			Opening Balance		0.00
0.00		Grant In Aid		424130.00		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			**424130.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure Over Income		0.00		0.00	Closing Balance		424130.00
0.00			**424130.00**		**0.00**			**424130.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-145: H3K4 HMT family regulatescell cycle progression
P.I: Dr Shweta Tyagi
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
2208206.00	Opening Balance	0.00		Opening Balance	1064782.00
0.00	Grant In Aid	1870600.00	187200.00	Salaries - Manpower	171600.00
0.00		0.00	1500000.00	Consumables	1400000.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	39509.00	Travel	24740.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	1546279.00	Equipment	321721.00
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
2208206.00		**1870600.00**	**3272988.00**		**2982843.00**
1064782.00	Excess of Expenditure Over Income	1112243.00	0.00	Closing Balance	0.00
3272988.00		**2982843.00**	**3272988.00**		**2982843.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-146: Role of MLL in ribosomal RNA transcription
P.I: Dr Shweta Tyagi
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
812209.00	Opening Balance	763439.00		Opening Balance	0.00
872000.00	Grant In Aid	809000.00	244262.00	Salaries - Manpower	224800.00
0.00		0.00	350000.00	Consumables	582862.00
0.00		0.00	0.00	Contingencies	0.00
0.00		0.00	0.00	Travel	17138.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	326508.00	Equipment	313781.00
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
1684209.00		**1572439.00**	**920770.00**		**1138581.00**
0.00	Excess of Expenditure Over Income	0.00	763439.00	Closing Balance	433858.00
1684209.00		**1572439.00**	**1684209.00**		**1572439.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
315642.00	Opening Balance	41311.00		Opening Balance	0.00
500000.00	Grant In Aid	0.00	187200.00	Salaries - Manpower	187200.00
0.00		0.00	400000.00	Consumables	400000.00
0.00		0.00	50000.00	Contingencies	50000.00
0.00		0.00	50831.00	Travel	31950.00
0.00		0.00	86300.00	Overheads	50000.00
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
815642.00		**41311.00**	**774331.00**		**719150.00**
0.00	Excess of Expenditure Over Income	677839.00	41311.00	Closing Balance	0.00
815642.00		**719150.00**	**815642.00**		**719150.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1770286.00	Opening Balance	270865.00		Opening Balance	0.00
1059500.00	Grant In Aid	0.00	187200.00	Salaries - Manpower	187200.00
0.00		0.00	1700000.00	Consumables	900000.00
0.00		0.00	150000.00	Contingencies	200000.00
0.00		0.00	53001.00	Travel	0.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	468720.00	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
2829786.00		**270865.00**	**2558921.00**		**1287200.00**
0.00	Excess of Expenditure Over Income	1016335.00	270865.00	Closing Balance	0.00
2829786.00		**1287200.00**	**2829786.00**		**1287200.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-150: Genetic and genomic basis of the evolution of bombycid and sturniid silkmoths
P.I: Dr J Nagaraju
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
164706.00	Opening Balance	0.00		Opening Balance	28096.00
0.00	Grant In Aid	153846.00	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	192802.00	Travel	125750.00
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
164706.00		**153846.00**	**192802.00**		**153846.00**
28096.00	Excess of Expenditure Over Income	0.00	0.00	Closing Balance	0.00
192802.00		**153846.00**	**192802.00**		**153846.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-151: Human Exome Sequencing to Identify Novel Genes for Medelian Disorders
P.I: Dr Ashwin B Dalal
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
1993200.00	Opening Balance	594981.00		Opening Balance	0.00
0.00	Grant In Aid	0.00	74729.00	Salaries - Manpower	343200.00
0.00		0.00	1200000.00	Consumables	800000.00
0.00		0.00	100000.00	Contingencies	25000.00
0.00		0.00	23490.00	Travel	28147.00
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
1993200.00		**594981.00**	**1398219.00**		**1196347.00**
0.00	Excess of Expenditure Over Income	601366.00	594981.00	Closing Balance	0.00
1993200.00		**1196347.00**	**1993200.00**		**1196347.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-152 : Global transcriptomics of sex specific spilicing
P.I: Dr K P Arun Kumar
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	1114145.00		Opening Balance	0.00
2872300.00	Grant In Aid	2562571.00	284155.00	Salaries - Manpower	343200.00
0.00		0.00	1474000.00	Consumables	3026000.00
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	278416.00
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
2872300.00		**3676716.00**	**1758155.00**		**3647616.00**
0.00	Excess of Expenditure Over Income	0.00	1114145.00	Closing Balance	29100.00
2872300.00		**3676716.00**	**2872300.00**		**3676716.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-153: An attractive and promising stragegy for early cancer diagnosis through the assembly of the human cancer volatome"
P.I: Dr H A Nagarajaram
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
3000000.00	Opening Balance	3613562.00		Opening Balance	0.00
937000.00	Grant In Aid	621000.00	58877.00	Salaries - Manpower	374400.00
0.00		0.00	70000.00	Consumables	70000.00
0.00		0.00	80000.00	Contingencies	80000.00
0.00		0.00	114561.00	Travel	68610.00
0.00		0.00	0.00	Overheads	0.00
0.00		0.00	0.00	Equipment	3000000.00
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
3937000.00		**4234562.00**	**323438.00**		**3593010.00**
0.00	Excess of Expenditure Over Income	0.00	3613562.00	Closing Balance	641552.00
3937000.00		**4234562.00**	**3937000.00**		**4234562.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-154 : Rational design, synthetic strategies for developing organometallic anticancer compounds based on organotin and organoiron
P.I: Dr Sunil Kumar Manna
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	87432.00		Opening Balance	0.00
1030000.00	Grant In Aid	943000.00	192568.00	Salaries - Manpower	249600.00
0.00		0.00	700000.00	Consumables	700000.00
0.00		0.00	0.00	Contingencies	50000.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	50000.00	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
1030000.00		**1030432.00**	**942568.00**		**999600.00**
0.00	Excess of Expenditure Over Income	0.00	87432.00	Closing Balance	30832.00
1030000.00		**1030432.00**	**1030000.00**		**1030432.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-155: Studies on thecellular roles of calcium signalling proteins in Neurospora crassa
P.I: Dr D P Kasbekar
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
335194.00	Opening Balance	335194.00		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 diesease control

PI : Dr Subhadeep Chatterjee

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	926632.00				Opening Balance	0.00	
2104400.00		Grant In Aid	1076500.00		197768.00		Salaries - Manpower	363601.00	
0.00			0.00		950000.00		Consumables	1600000.00	
0.00			0.00		30000.00		Contingencies	50000.00	
0.00			0.00		0.00		Travel	32201.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	132495.00	
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	
2104400.00			**2003132.00**		**1177768.00**			**2178297.00**	
0.00		Excess of Expenditure Over Income	175165.00		926632.00		Closing Balance	0.00	
2104400.00			**2178297.00**		**2104400.00**			**2178297.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	944665.00				Opening Balance	0.00	
2760800.00		Grant In Aid	1317000.00		165309.00		Salaries - Manpower	195880.00	
0.00			0.00		1200000.00		Consumables	1200000.00	
0.00			0.00		50000.00		Contingencies	50000.00	
0.00			0.00		19974.00		Travel	0.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		380852.00		Equipment	611413.00	
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	
2760800.00			**2261665.00**		**1816135.00**			**2057293.00**	
0.00		Excess of Expenditure Over Income	0.00		944665.00		Closing Balance	204372.00	
2760800.00			**2261665.00**		**2760800.00**			**2261665.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	621787.00				Opening Balance	0.00	
1933141.00		Grant In Aid	0.00		233567.00		Salaries - Manpower	342277.00	
0.00			0.00		1000000.00		Consumables	1300000.00	
0.00			0.00		70000.00		Contingencies	50000.00	
0.00			0.00		7787.00		Travel	9227.00	
0.00			0.00		0.00		Overheads	0.00	
0.00			0.00		0.00		Equipment	299941.00	
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	
1933141.00			**621787.00**		**1311354.00**			**2001445.00**	
0.00		Excess of Expenditure Over Income	1379658.00		621787.00		Closing Balance	0.00	
1933141.00			**2001445.00**		**1933141.00**			**2001445.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	300000.00				Opening Balance	0.00	
300000.00		Grant In Aid	0.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	300000.00	
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	
300000.00			**300000.00**		**0.00**			**300000.00**	
0.00		Excess of Expenditure Over Income	0.00		300000.00		Closing Balance	0.00	
300000.00			**300000.00**		**300000.00**			**300000.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-160 : Understanding the role of novel adhesins of Xanthomonas oryzae PV orzae in Virulence and colonization in Rice

PI : Dr Subhadeep Chatterjee

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	363884.00				Opening Balance	0.00	
382000.00		Grant In Aid	531649.00		18116.00		Salaries - Manpower	187200.00	
0.00			0.00		0.00		Consumables	500000.00	
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	
382000.00			**895533.00**		**18116.00**			**687200.00**	
0.00		Excess of Expenditure Over Income	0.00		363884.00		Closing Balance	208333.00	
382000.00			**895533.00**		**382000.00**			**895533.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-161 : Analysis of co-regulation between DNA replication activity and amino acid homeostatis by transcription factor IciA/ArgP in Eschericia coli

PI : Dr J Gowrishankar

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	350000.00				Opening Balance	0.00	
350000.00		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	10000.00	
0.00			0.00		0.00		Travel	255344.00	
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	
350000.00			**350000.00**		**0.00**			**265344.00**	
0.00		Excess of Expenditure Over Income	0.00		350000.00		Closing Balance	84656.00	
350000.00			**350000.00**		**350000.00**			**350000.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	235671.00		Opening Balance	0.00
799600.00	Grant In Aid	0.00	70955.00	Salaries - Manpower	130928.00
0.00		0.00	477974.00	Consumables	400000.00
0.00		0.00	15000.00	Contingencies	0.00
0.00		0.00	0.00	Travel	21207.00
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
799600.00		**235671.00**	**563929.00**		**552135.00**
0.00	Excess of Expenditure Over Income	316464.00	235671.00	Closing Balance	0.00
799600.00		**552135.00**	**799600.00**		**552135.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount Rs	Receipts	Current Year Amount Rs.	Previous Year. Amount Rs	Payments	Current Year Amount Rs
0.00	Opening Balance	2006048.00		Opening Balance	0.00
2006048.00	Grant In Aid	0.00	0.00	Salaries - Manpower	53577.00
0.00		0.00	0.00	Consumables	800000.00
0.00		0.00	0.00	Contingencies	40000.00
0.00		0.00	0.00	Travel	0.00
0.00		0.00	0.00	Overheads	60000.00
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
2006048.00		**2006048.00**	**0.00**		**953577.00**
0.00	Excess of Expenditure Over Income	0.00	2006048.00	Closing Balance	1052471.00
2006048.00		**2006048.00**	**2006048.00**		**2006048.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00				Opening Balance	26671.00	
0.00		Grant In Aid	188000.00		26671.00		Salaries - Manpower	156000.00	
0.00			0.00		0.00		Consumables	30000.00	
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			**188000.00**		**26671.00**			**212671.00**	
26671.00		Excess of Expenditure Over Income	24671.00		0.00		Closing Balance	0.00	
26671.00			**212671.00**		**26671.00**			**212671.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-165 : Identification and functional characterization of immune response genes in silkmoths
PI : Dr V V Satyavathi
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	1569682.00				Opening Balance	0.00	
1569682.00		Grant In Aid	0.00		0.00		Salaries - Manpower	122400.00	
0.00			0.00		0.00		Consumables	1000000.00	
0.00			0.00		0.00		Contingencies	50000.00	
0.00			0.00		0.00		Travel	17147.00	
0.00			0.00		0.00		Overheads	50000.00	
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	
1569682.00			**1569682.00**		**0.00**			**1239547.00**	
0.00		Excess of Expenditure Over Income	0.00		1569682.00		Closing Balance	330135.00	
1569682.00			**1569682.00**		**1569682.00**			**1569682.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00				Opening Balance	0.00	
0.00		Grant In Aid	4383200.00		0.00		Salaries - Manpower	196003.00	
0.00			0.00		0.00		Consumables	2000000.00	
0.00			0.00		0.00		Contingencies	20000.00	
0.00			0.00		0.00		Travel	1559.00	
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			**4383200.00**		**0.00**			**2217562.00**	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	2165638.00	
0.00			**4383200.00**		**0.00**			**4383200.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00				Opening Balance	0.00	
0.00		Grant In Aid	1700000.00		0.00		Salaries - Manpower	64916.00	
0.00			0.00		0.00		Consumables	862000.00	
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	100000.00	
0.00			0.00		0.00		Equipment	39304.00	
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			**1700000.00**		**0.00**			**1066220.00**	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	633780.00	
0.00			**1700000.00**		**0.00**			**1700000.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		1400000.00		0.00	Salaries - Manpower		29187.00
	0.00			0.00		0.00	Consumables		450000.00
	0.00			0.00		0.00	Contingencies		0.00
	0.00			0.00		0.00	Travel		740.00
	0.00			0.00		0.00	Overheads		100000.00
	0.00			0.00		0.00	Equipment		31450.00
	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			**1400000.00**		**0.00**			**611377.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		788623.00
	0.00			**1400000.00**		**0.00**			**1400000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-169 : Implementation of 3 year DNB Program in Medical Genetics by Department of Biotechnology in colloboration with National Board of Examination ag SGHR, NIBMG&CDFD
PI : Dr J Gowrishankar
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		1890000.00		0.00	Salaries - Manpower		0.00
	0.00			0.00		0.00	Consumables		81892.00
	0.00			0.00		0.00	Contingencies		50000.00
	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			**1890000.00**		**0.00**			**131892.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		1758108.00
	0.00			**1890000.00**		**0.00**			**1890000.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00				Opening Balance	0.00	
0.00		Grant In Aid	820000.00		0.00		Salaries - Manpower	142551.00	
0.00			0.00		0.00		Consumables	300000.00	
0.00			0.00		0.00		Contingencies	50000.00	
0.00			0.00		0.00		Travel	0.00	
0.00			0.00		0.00		Overheads	50000.00	
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			**820000.00**		**0.00**			**542551.00**	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	277449.00	
0.00			**820000.00**		**0.00**			**820000.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00				Opening Balance	0.00	
0.00		Grant In Aid	2415730.00		0.00		Salaries - Manpower	0.00	
0.00			0.00		0.00		Consumables	600000.00	
0.00			0.00		0.00		Contingencies	25000.00	
0.00			0.00		0.00		Travel	36283.00	
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			**2415730.00**		**0.00**			**661283.00**	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	1754447.00	
0.00			**2415730.00**		**0.00**			**2415730.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-172 : Molecular Characterization of early onset sporadic rectal cancer

PI : Dr M D Bashyam

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		2100000.00		0.00	Salaries - Manpower		38253.00
	0.00			0.00		0.00	Consumables		600000.00
	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			**2100000.00**		**0.00**			**638253.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		1461747.00
	0.00			**2100000.00**		**0.00**			**2100000.00**

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

PI : Dr Ashwin B Dalal

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		699782.00		0.00	Salaries - Manpower		29900.00
	0.00			0.00		0.00	Consumables		85000.00
	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			**699782.00**		**0.00**			**114900.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		584882.00
	0.00			**699782.00**		**0.00**			**699782.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

P-174 : Is non-canonical Wnt signalling a major player in early-onset sporadic rectal cancer

PI : Dr M D Bashyam

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Previous Year Rs	Receipts	Current Year Amount	Current Year Rs.	Previous Year. Amount	Previous Year. Rs	Payments	Current Year Amount	Current Year Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		500000.00		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			**500000.00**		**0.00**			**0.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		500000.00
	0.00			**500000.00**		**0.00**			**500000.00**

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"

PI : Dr Ashwin B Dalal

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Previous Year Rs	Receipts	Current Year Amount	Current Year Rs.	Previous Year. Amount	Previous Year. Rs	Payments	Current Year Amount	Current Year Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		0.00		0.00	Salaries - Manpower		9714.00
	0.00			0.00		0.00	Consumables		500000.00
	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**			**509714.00**
	0.00	Excess of Expenditure Over Income		509714.00		0.00	Closing Balance		0.00
	0.00			**509714.00**		**0.00**			**509714.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
P-176 : International Atomic Energy Agency

PI : Dr K P Arun Kumar
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00				Opening Balance	0.00	
0.00		Grant In Aid	200103.00		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			**200103.00**		**0.00**			**0.00**	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	200103.00	
0.00			**200103.00**		**0.00**			**200103.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE1/CORE : COE for Genetics and Genomics of silkmoths

PI : Dr. J. Nagaraju
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		7745497.00		Opening Balance	12372212.00	
3814000.00		Grant In Aid	9102000.00		7103559.00		Salaries - Manpower	7357519.00	
0.00			0.00		1200000.00		Consumables	1200000.00	
0.00			0.00		0.00		Contingencies	0.00	
0.00			0.00		137156.00		Travel	143020.00	
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	
3814000.00			**9102000.00**		**16186212.00**			**21072751.00**	
12372212.00		Excess of Expenditure Over Income	11970751.00		0.00		Closing Balance	0.00	
16186212.00			**21072751.00**		**16186212.00**			**21072751.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE1/P-I : Comparative and function genomics of silkmoths.

PI : Dr. J. Nagaraju
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		512437.00		Opening Balance	449637.00	
750000.00		Grant In Aid	732000.00		187200.00		Salaries - Manpower	137866.00	
0.00			0.00		500000.00		Consumables	500000.00	
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	
750000.00			**732000.00**		**1199637.00**			**1087503.00**	
449637.00		Excess of Expenditure Over Income	355503.00		0.00		Closing Balance	0.00	
1199637.00			**1087503.00**		**1199637.00**			**1087503.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		621001.00		Opening Balance	387740.00	
643000.00		Grant In Aid	459000.00		109739.00		Salaries - Manpower	191226.00	
0.00			0.00		300000.00		Consumables	300000.00	
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			**459000.00**		**1030740.00**			**878966.00**	
387740.00		Excess of Expenditure Over Income	419966.00		0.00		Closing Balance	0.00	
1030740.00			**878966.00**		**1030740.00**			**878966.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		550072.00		Opening Balance	505830.00	
1009000.00		Grant In Aid	1090000.00		614758.00		Salaries - Manpower	709200.00	
0.00			0.00		350000.00		Consumables	350000.00	
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	
1009000.00			**1090000.00**		**1514830.00**			**1565030.00**	
505830.00		Excess of Expenditure Over Income	475030.00		0.00		Closing Balance	0.00	
1514830.00			**1565030.00**		**1514830.00**			**1565030.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE-I/P-IV : Identification and characterization ofimmune response genes of silkmoths.

PI : Dr. J. Nagaraju

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		216524.00		Opening Balance	153724.00	
450000.00		Grant In Aid	463000.00		187200.00		Salaries - Manpower	130839.00	
0.00			0.00		200000.00		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	
450000.00			**463000.00**		**603724.00**			**484563.00**	
153724.00		Excess of Expenditure Over Income	21563.00		0.00		Closing Balance	0.00	
603724.00			**484563.00**		**603724.00**			**484563.00**	

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		10800722.00		Opening Balance	19234494.00	
0.00		Grant In Aid	0.00		7816390.00		Salaries - Manpower	4606321.00	
0.00			0.00		400000.00		Consumables	0.00	
0.00			0.00		158925.00		Contingencies	0.00	
0.00			0.00		58457.00		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**		**19234494.00**			**23840815.00**	
19234494.00		Excess of Expenditure Over Income	23840815.00		0.00		Closing Balance	0.00	
19234494.00			**23840815.00**		**19234494.00**			**23840815.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE2/P-1 : Addressing functiional properties of E. coli through genome-wide protein-protein linkage analysis

PI : Dr. J Gowrishankar
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		684083.00		Opening Balance	684083.00	
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**		**684083.00**			**684083.00**	
684083.00		Excess of Expenditure Over Income	684083.00		0.00		Closing Balance	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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		545571.00		Opening Balance	1097981.00	
0.00		Grant In Aid	0.00		230310.00		Salaries - Manpower	343200.00	
0.00			0.00		215000.00		Consumables	0.00	
0.00			0.00		100000.00		Contingencies	0.00	
0.00			0.00		7100.00		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**		**1097981.00**			**1441181.00**	
1097981.00		Excess of Expenditure Over Income	1441181.00		0.00		Closing Balance	0.00	
1097981.00			**1441181.00**		**1097981.00**			**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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		473063.00		Opening Balance	1085152.00	
0.00		Grant In Aid	0.00		358800.00		Salaries - Manpower	269100.00	
0.00			0.00		200000.00		Consumables	0.00	
0.00			0.00		50000.00		Contingencies	0.00	
0.00			0.00		3289.00		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**		**1085152.00**			**1354252.00**	
1085152.00		Excess of Expenditure Over Income	1354252.00		0.00		Closing Balance	0.00	
1085152.00			**1354252.00**		**1085152.00**			**1354252.00**	

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2/P-B : Molecular genetic approaches to dissect the physiology of osmoadptation in Escherichia coli

PI : Dr. J. Gowrishankar, Dr. Abhijit A Sardesasi

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00		394420.00		Opening Balance	1006509.00	
0.00		Grant In Aid	0.00		358800.00		Salaries - Manpower	269100.00	
0.00			0.00		200000.00		Consumables	0.00	
0.00			0.00		50000.00		Contingencies	0.00	
0.00			0.00		3289.00		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**		**1006509.00**			**1275609.00**	
1006509.00		Excess of Expenditure Over Income	1275609.00		0.00		Closing Balance	0.00	
1006509.00			**1275609.00**		**1006509.00**			**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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
79678.00		Opening Balance	0.00				Opening Balance	473354.00	
0.00		Grant In Aid	0.00		83032.00		Salaries - Manpower	0.00	
0.00			0.00		400000.00		Consumables	0.00	
0.00			0.00		70000.00		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	
79678.00			**0.00**		**553032.00**			**473354.00**	
473354.00		Excess of Expenditure Over Income	473354.00		0.00		Closing Balance	0.00	
553032.00			**473354.00**		**553032.00**			**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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		2186000.00		0.00	Salaries - Manpower		114735.00
	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			**2186000.00**		**0.00**			**114735.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		2071265.00
	0.00			**2186000.00**		**0.00**			**2186000.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		1093000.00		0.00	Salaries - Manpower		89700.00
	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			**1093000.00**		**0.00**			**289700.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		803300.00
	0.00			**1093000.00**		**0.00**			**1093000.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		500000.00		0.00	Salaries - Manpower		0.00
	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			**500000.00**		**0.00**			**200000.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		300000.00
	0.00			**500000.00**		**0.00**			**500000.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		1093000.00		0.00	Salaries - Manpower		89700.00
	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			**1093000.00**		**0.00**			**289700.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		803300.00
	0.00			**1093000.00**		**0.00**			**1093000.00**

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 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		500000.00		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			**500000.00**		**0.00**			**0.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		500000.00
	0.00			**500000.00**		**0.00**			**500000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD

COE2-II/P-E : Understanding (p) ppGpp-mediated functions in E.Coliby deciphering the physiology of strain lacking (p)ppGpp OR altered in its metabolism

PI : Dr J Gowrishankar

Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
	0.00	Opening Balance		0.00			Opening Balance		0.00
	0.00	Grant In Aid		1093000.00		0.00	Salaries - Manpower		16774.00
	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			**1093000.00**		**0.00**			**16774.00**
	0.00	Excess of Expenditure Over Income		0.00		0.00	Closing Balance		1076226.00
	0.00			**1093000.00**		**0.00**			**1093000.00**

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS, HYDERABAD
COE2-II-Core : DBT Centre of Excellence for Microbiology - Phase II

PI : Dr J Gowrishankar
Receipts and Payments Account from 01/04/2014 to 31/03/2015

Previous Year Amount	Rs	Receipts	Current Year Amount	Rs.	Previous Year. Amount	Rs	Payments	Current Year Amount	Rs
0.00		Opening Balance	0.00				Opening Balance	0.00	
0.00		Grant In Aid	11236000.00		0.00		Salaries - Manpower	956577.00	
0.00			0.00		0.00		Consumables	600000.00	
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	156100.00	
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			**11236000.00**		**0.00**			**1712677.00**	
0.00		Excess of Expenditure Over Income	0.00		0.00		Closing Balance	9523323.00	
0.00			**11236000.00**		**0.00**			**11236000.00**	

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Photo Gallery

CODIS Installation and training in CODIS Software (Hon'ble Consul General of USA Mr Michael Mullins visited CDFD)

Visit from Madhya Pradesh Council of Science & Technology, Bhopal under 8th Vigyan Mathan Yatra 2014-15 under M.P. Mission Excellence programme.

Flag hoisting on the occasion of Independence Day 2014.

Hindi Workshop on Unicode Software.

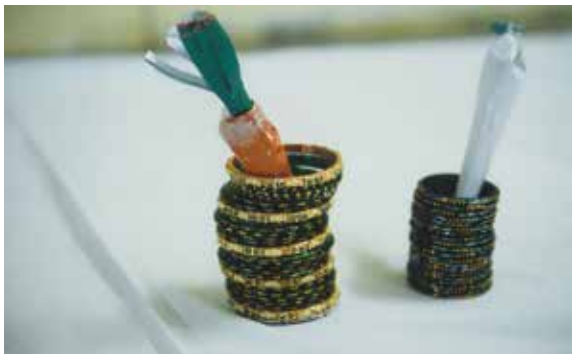
Celebration of Hindi Day, 2014





Glimpses of Foundation Day Celebrations, 2015



Wealth Out of Waste, created by children of CDFD staff

Rangoli and Art Competitions for staff, students and children of CDFD staff