



SBC NEWSLETTER

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79TH SBC (I) ANNUAL MEETING

DECEMBER 13-15, 2010

J N Tata Auditorium, IISc. Campus, Bangalore - 560 012

Regulation of biochemical and cellular processes in diverse systems

For further information please contact:

Drs. Parag P Sadhale & Dipankar Nandi

Convenors

Society of Biological Chemists, India, Indian Institute of science, Bangalore 560012

Ph: 080- 23601412; Email: sbcihq@gmail.com

Web: <http://sbconference2010.org>

The meeting will consist of plenary lectures, award winning lectures, invited lectures in various symposia by leading scientists from all over the country and the talks will represent cutting edge biological research.

The tentative topics for the main scientific session are:

1. PROTEINS: STRUCTURE AND FUNCTION
2. PATHOGENS and DISEASE
3. REGULATION OF TRANSCRIPTION
4. ENZYMES AND METABOLIC PATHWAYS
5. SYSTEMS BIOLOGY
6. DNA AND CHROMOSOME DYNAMICS
7. CELLULAR RESPONSES and SIGNALING
8. MOLECULAR MICROBIOLOGY
9. THE IMMUNE RESPONSE
10. DNA REPAIR
11. CELLULAR DIFFERENTIATION
12. REGULATION OF CELL CYCLE and GROWTH
13. RNA BIOLOGY
14. BACTERIAL PATHOGENS
15. ECOLOGY
16. MECHANISMS OF CELL DEATH
17. NEUROBIOLOGY

The topics for poster presentation are:

1. METABOLISM & NUTRITION
2. CLINICAL & ANALYTICAL BIOCHEMISTRY
3. LIPIDS, MEMBRANES & BIOENERGETICS
4. ENZYMES & OTHER BIOCATALYSTS
5. STRUCTURAL BIOLOGY
6. GENE EXPRESSION
7. GENOMICS AND GENOME DIVERSITY
8. MICROBIOLOGY & INFECTIOUS DISEASES
9. ALLERGY & IMMUNOLOGY
10. PLANT PHYSIOLOGY & BIOCHEMISTRY
11. DEVELOPMENT BIOLOGY
12. REPRODUCTIVE BIOLOGY & ENDOCRINOLOGY
13. NEUROBIOLOGY & NEUROCHEMISTRY
14. BIOTECHNOLOGY & BIOENGINEERING
15. GENETIC DISORDERS & CANCER BIOLOGY
16. CELL BIOLOGY & CELL SIGNALING
17. PHARMACOLOGY & PHARMACOKINETICS
18. BIOINFORMATICS
19. VIROLOGY
20. ENVIRONMENTAL BIOLOGY
21. AGRICULTURAL BIOCHEMISTRY

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IMPORTANT DEADLINES

Last date for payment of
Registration fee
Oct 01, 2010

Last date for
submission of Abstracts
Oct 01, 2010

Information of
Abstracts acceptance
Oct 15, 2010

ABSTRACTS

A student needs to be a SBC(I) member in order to present the work. Abstracts reporting studies on all topics given above (poster presentation) are welcome.

The last date for receiving abstracts is October 01, 2010 and acceptance of selected abstracts will be communicated by October 15, 2010 by email.

Participants are invited to submit abstracts in a format given in the conference web site:
<http://sbciconference2010.org>.

ACCOMMODATION

The participants are requested to make their own arrangements for accommodation. The details of nearby hotels in Bangalore are given below. Students / participants are requested to contact and confirm the reservation with the hotel directly.

List of Hotels	~Rate / day	Tel / email
Hotel Adarsh Lodge, Gandhi Nagar	Rs.300/-	22267089
Hotel Janardhan, Gandhi Nagar	Rs.300/-	22263645/22266745
Hotel Panchavati Palace, Anand Rao Circle	Rs.300/-	41495904
Hotel Regency Deluxe lodge, Gandhinagar	Rs.300/-	22205475/22255146 22255147/22255149 22203264
Saraswathi Lodge, Anand Rao Circle	Rs.300/-	2226 4326
Hotel Sangeeth, Anand Rao Circle	Rs.300/-	22264142/143/144/149
Hotel Sheetal, Anand Rao Circle	Rs.300/-	41139560
Sri Hariram Hotel, K.G. Road	Rs.300/-	22267990
Suprabhatha, Anand Rao Circle	Rs.300/-	22873140/141/142/143

The organizers will arrange limited accommodation on a first come basis upon acceptance of the abstract. Kindly send an email to sbcihq@gmail.com regarding accommodation. Confirmation of this type of accommodation for students will be intimated by email and will be available in the conference web site <http://sbciconference2010.org>.

REGISTRATION

Registration Fee - CATEGORY	Before 1/10/2010 (Rs.)	After 1/10/2010 (Rs.)
Student & Life Members	Rs.2000	Rs.3000
Student Non Members	Rs.2500	Rs.3500
Others	Rs.3500	Rs.4500
Industries	Rs.5000	Rs.6000
Accompanying persons (without conference material)	Rs.2000	Rs.3000

Registration fee includes conference material, lunch and dinner on conference days.; Students registration form must be signed by his/her research guide.; Society of Biological Chemists encourages all students to become members.; Send your registration fee by Bank Demand Draft in the favour of SBC2010 to Hon Secretary, Society of Biological Chemists, India, Indian Institute of Science, Bangalore 560012.

REGISTRATION FORM

79th Annual Meeting of the Society of Biological Chemists, (India)
Indian Institute of Science, Bangalore, December 13-15,2010

Please complete this form and submit it **on-line** not later than **October 1, 2010**

Name

Designation

Affiliation

Department

Address

City Post/PinCode

Telephone & Fax

Email

Age: Sex: Male Female

Member of Society: Yes No Category: Life ordinary student

Have you submitted an abstract: Yes No

If yes, Title of Abstract

(for further details on Abstract submission, please see <http://sbciconference2010.org>)

I intend to make a poster presentation/ only attend the meeting (strike whichever is not applicable)

Would like to share accommodation, if any

Mode of arrival: Air Train Bus Date and Time of Arrival

Mode of Departure Air Train Bus Date and Time of Departure

Payment : Bank draft should be drawn in favour of "SBC(I)-2010, payable at Bangalore

Amount Draft No. Dated

Name of the Bank

NOMINATIONS FOR 2010 SBC(I) AWARD

This year **P S Sarma Memorial Award, Shadaksharaswamy Endowment Award, P A Kurup Award, D P Burma Memorial Lecture Award, A Krishnamurthy Award** (best paper published in Indian Journal), will be given at the annual meeting held at Bangalore. Please send the nominations including the date of birth, member status, brief resume of scientific contributions and a list of publications and few selected reprints in five copies of the nominee to:

Dr V S Chauhan

President SBC(I), Director ICGEB
Aruna Asaf Ali Marg, New Delhi 110 067
E-mail: virander@icgeb.res.in

Complete application should reach Dr. V S Chauhan on or before November 1st 2010.

CRITERIA FOR 2010 AWARDS

P. S. SARMA MEMORIAL AWARD

Year of Commencement : 1973
Frequency : Once in three years
Value : Rs.10,000/- with a citation

Eligibility

1. The award is for the best work done in the field of Biochemistry and Allied Sciences in India.
2. The recipient of the award should not have completed 50 years before January 1st in the year for which the award is announced.
3. A lecture will be scheduled at the Annual Meeting of SBC(I) and presentation will be made at that time.

PROF. M. SHADAKSHARA SWAMY ENDOWMENT LECTURE AWARD

Year of Commencement : 1982
Sponsor : Prof. M. Shadaksharaswamy Felicitation Committee.
Frequency : Once in three years
Value : Rs.10,000/- with a citation

Eligibility

1. Eminent teachers in Biological Chemistry & Allied Sciences at the postgraduate level in Indian Universities, deemed Universities and Institutions of higher learning for their contributions in teaching and research.
2. No age limit.
3. The recipient of the award should give a lecture at the time of annual meeting of SBC(I) on a topic of his/her choice.

PROF. P. A. KURUP ENDOWMENT AWARD

Year of Commencement: 1991
Frequency : Once in three years
Value : Rs.10,000/- with a citation

Eligibility

1. The recipient of the award should be below 60 yrs of age on January 1st of the year of the award.
2. The award is given for Biomedical Research.
3. The awardee should give a lecture during the Annual Meeting.

Dr. D.P. BURMA MEMORIAL LECTURE AWARD IN BIOLOGICAL SCIENCES

Year of Commencement: 2007

Frequency : Once in three years

Value : Rs.20,000/- with a citation

Eligibility

1. The award will be given to an eminent scientist for outstanding life time contribution in the field of Biological Sciences.
2. The awardee should give a lecture at the Annual Meeting of SBC (I).

A. KRISHNAMURTHY AWARD

Year of Commencement: 1976

Frequency : Annually

Value : Rs.2,000/- with a citation

Eligibility

1. The recipient of the award should be below 30 years of age on January 1, of the year of the award.
2. The paper should be in the area of Biological Chemistry and Allied Sciences and the work should have been carried out in India.
3. The paper published in any Indian Scientific Journal in the previous year will be considered for the award.
4. In the case of multiple authorship, the senior author can nominate one of the authors or could be shared by all the eligible authors.

BEST POSTER AWARDS

The list of recipients for Best Poster Awards at the 78th Annual General Body Meeting held at Pune during October 30-November 1, 2009 is as follows:

Name	Topic
Manoj Bhosale, IISc.	Roles of <i>Salmonella enterica</i> serovar Typhimurium encoded Peptidase N during stress and infection.
Gregor P J, IISc.	Anti-bacterial and anti-cancer activity of copper nano particles.
Sheetal S Salvi, IISc.	Mutations in <i>Drosophila</i> myosin rod cause muscle defects similar to human myopathies.
Barathi S, Vision Res.Foundation	Amino acids glycine and glutamic acid functions inhibitors by down regulating VEGF expression.
Sushma Padmaja, IIT-Madras	Organization of Scaffold/Matrix attachment regions in <i>Giardia lamblia</i> .
Anuj Pathak, IMTECH	Domain structure of virulence associated response regulator PhoP of <i>Mycobacterium tuberculosis</i> .
Sreejith R K, Univ of Pune	Conformational characterization of human eukaryotic translation initiation factor 2a : a single tryptophan protein.
Shweta Jha, NBRI	Expression, subcellular localization and glycosylation of functionally active recombinant human α 1 – proteinase inhibitor in transgenic tomato plants.
Abani Kanta Naik, IISc.	Cytosines determine RAG induced breaks on altered DNA structures: implications for genomic stability.
Sohrab Z. Khan, NCCS	Host cellular factors in HIV-1 pathogenesis: interactions and interplay.

I am still mightier than you!

I am everywhere with no neuronal cells
Catch me if you can with all your neuronal cells
With New Generation antibiotics and New generation Vaccines
Come and kill me if you can with all your new tactics
I will design smarter ways to make your efforts go waste
And the Tug-of-War will continue in a zillion ways
If you want to kill me, understand me first 3,000 vs 30,000 is not that much!
I am slim, I am trim, I grow fast and You grow Slow
How will you ever overtake me in this show?
I will hide inside your body in a "Customized Shell"
I will hijack everything and make you unwell
Your antibiotics can never reach me
And you will be never able to breach me
In this Game of "Hide and Seek"! Who will hide and Whom to seek
I am the "Tortoise" and you are the "Hare"
I am a tiny person, understand me somewhere!
My meal is simple and yours is complex
My life is poor and yours is lavish
Still I Win and you Loose
My strategies are powerful and yours are weak!
Still I exist in your feast!
And this "Party" continues unleashed!!!
Dipshikha Chakravorty

(Dipshikha Chakravorty is a faculty in Department of Microbiology and Cell Biology, Indian Institute of Science, Bangalore. She is fascinated by the strategies the bacteria uses to defend the host. She and her team are involved in elucidating the evasion strategies by bacterial pathogens. In this poem, "I" refers to a pathogenic bacteria and "You" refers to human. The "bug" refers to the bacterial pathogen which grows intra-cellularly inside the cells like *Mycobacteria*, *Salmonella*, *Chlamydiae*, *Coxiella*. These pathogens have intelligent strategies to overcome the host defense mechanism.

Epigenetics and Transcriptional Regulation: Implications in Cancer Manifestation

Yogesh Ostwal*, Parijat Senapati* and Tapas K Kundu

Transcription and Disease Laboratory, Molecular Biology and Genetics Unit, Jawaharlal Nehru Centre for Advanced Scientific Research, Bangalore, India.

*contributed equally to the article

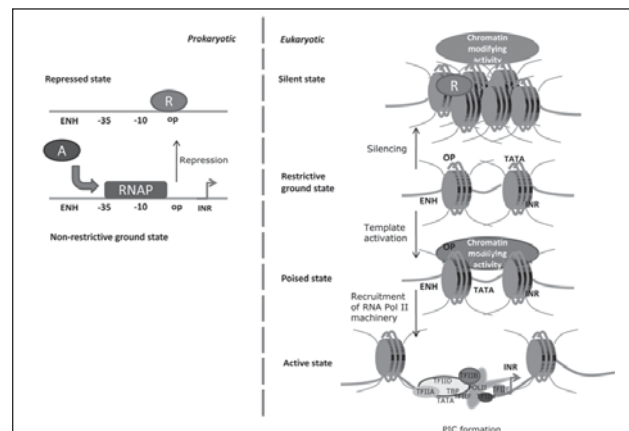
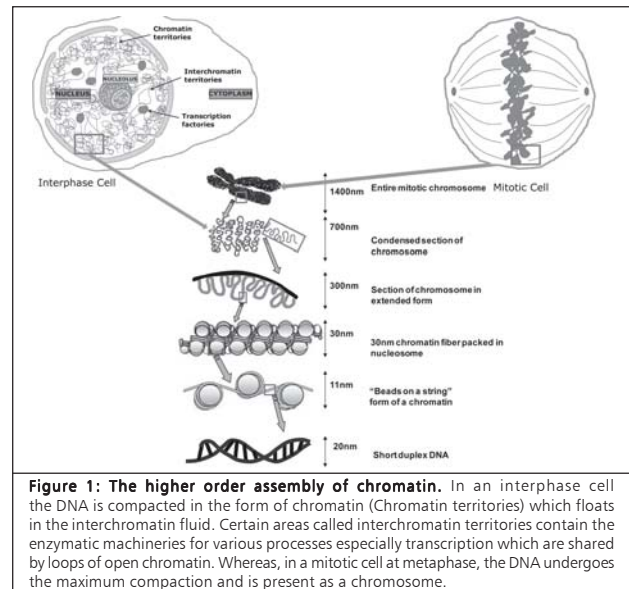
Introduction

The eukaryotic genome is organized into a highly compact and dynamic nucleoprotein structure composed of DNA, proteins and RNA. Apart from the nucleus, DNA is also present in the mitochondria and plastids (in plants) which is also organized into a nucleoprotein structure but is not chromatinised in a regular manner as that of the nucleus. In the nucleus, nucleolus also presumably possesses the chromatin structure. In humans and other higher eukaryotes, most of the functional genes are present in the nuclear DNA. Over the period, the definition of genes has changed a lot and as per the present concept, any fragment of DNA that codes for RNA may be referred to as a 'gene'. All the RNAs transcribed from DNA need not be translated into a protein. Several RNAs are known which are not translated into proteins but nevertheless play an important role in many biological processes. However, in a loose sense the entire complement of DNA molecules present in a cell is referred to as the genetic material. To sustain life, this genetic material needs to be replicated and also repaired to overcome the constant assault from the environment. Therefore, the compact nucleoprotein structure needs to be dynamic, meaning that based on the requirement, it should fold and unfold.

Chromatin organization and transcription

In a tiny nucleus of few microns, almost two meters long human DNA is packaged in an orderly manner. During metaphase, the entire human DNA is compacted in the form of 23 pairs of chromosomes. However, in interphase, each homologous chromosome occupies a unique three dimensional niche and floats in the highly dense interchromatin territory (Figure 1). The primary thread of chromatin is a beads on a string structure composed repeating units called nucleosome. Each nucleosome is composed of a histone octamer consisting of two molecules each of the histones H2A, H2B, H3 and H4 around which approximately two superhelical turns of DNA are wrapped. The N-terminal tails of these histones are highly unstructured, projecting out of the nucleosome. One molecule of histone H1 associates at the point where the DNA enters and exits the nucleosome core particle thus sealing the two turns of DNA. These histones especially their N-terminal tails undergo vast array of post-translational modifications thereby influencing chromatin structure. Apart from canonical histones, there exist several histone variants that affect chromatin structure. Also, there are several ATP-dependent remodeling complexes and histone chaperones which are known to influence chromatin structure.

All DNA-templated processes such as transcription, replication and repair require that the chromatin structure be accessible to the enzymatic machineries. Since, prokaryotic DNA is not present in the form of chromatin, there exist fundamental differences in all these processes. In the case of transcription, these differences are well explained by the concept of transcriptional ground state. The transcriptional ground state implies the inherent activity of promoters (and hence core transcription machineries) in the absence of specific regulatory sequences (and hence activators and repressors). In prokaryotes, there is no inherent restriction on the ability of RNA polymerase to gain access to the DNA template and initiate transcription in vivo. Thus, for prokaryotic



organisms, the ground state for transcription is non restrictive (**Figure 2**). Whereas, in the case of eukaryotes, the transcriptional ground state is restrictive due to the chromatin structure, meaning that the core transcriptional machineries cannot get access to the promoters. Hence, prokaryotic genes can be either in the transcriptional ground state or repressed state due to the binding of a repressor. Whereas eukaryotic chromatin can have multiple alternative states, viz; the silent state where repressors are bound and chromatin (gene) is compact, the transcriptional ground state where there is absence of a repressor or an activator, a poised state where the chromatin modifying enzymes have displaced the histones and basal transcriptional machineries can now be recruited to give the active state of chromatin.

These different states of chromatin are governed by the DNA methylation status, canonical histones or histone variants present in the promoter proximal nucleosomes and the post-translational modification status of the histones which function as marks for recruitment of activators or repressors, and further modulate gene expression. There are many chromatin modifying enzymes which help in altering the states of chromatin, mostly they are histone modifying enzymes which post-translationally modify the histones, ATP-dependent chromatin remodelers which can remodel or change the position of histones along the DNA and histone chaperones which help in assembly or disassembly of histones during various DNA-templated phenomena and overall histone metabolism (**Figure 3**). Histone modifications are exclusively associated with particular chromatin states and form genome-wide patterns that reflect global states of chromatin. These modifications include reversible acetylation, methylation (of lysine and arginine), and phosphorylation. Acetylation is generally considered as a mark of transcriptionally active state of the chromatin. In a broad sense, chromatin can be divided into euchromatin and heterochromatin which roughly align with active and inactive states of chromatin. One of the important histone modifications that is exclusively associated with active state is H3K4me3 (trimethylation of histone H3 at lysine 4 residue). It increases acetylation turnover of the associated genes making them poised for transcription activation and is conserved from yeast to humans. On the other hand, H3K9me3 (trimethylation of histone H3 at lysine 9 residue) in *Drosophila* and mammals is exclusively associated with inactive states. It serves as a platform for the binding of Heterochromatin protein 1 (HP1) and initiates and maintains heterochromatinization (**Figure 4**).

Epigenetics and gene expression

From the above discussion, it is clear that the DNA sequence present is not the only determinant of gene expression. All the cell types in an organism contain the same DNA sequence, yet they show enormous variability in their phenotypes. Several mechanisms have been discovered that regulate distinct gene expression patterns and all such mechanisms come under the purview of Epigenetics. The term 'Epigenetics' was coined by a developmental biologist Conrad Waddington, in early 1950s. It now refers to "potentially heritable changes in gene expression without any change in DNA sequence". These epigenetic mechanisms include DNA methylation, post-translational modifications of histones, chromatin remodeling, and replacement of canonical histones by histone variants etc. giving rise to stable states of gene expression that are inherited from one cell generation to the next. DNA methylation is the earliest proposed epigenetic mark which was thought to play a critical role in gene silencing. The sites of DNA methylation are palindromic, CpG rich. There are two sets of enzymes that catalyze DNA methylation and methylate unmodified DNA and hemi-methylated DNA respectively. Later on, it was discovered that CpG rich sequences are present upstream of most of the transcription start sites as CpG islands and have evolved in warm-blooded animals as an important regulatory epigenetic mechanism. Methylation of DNA at the promoter is closely linked to histone modifications and thereby the entire system of transcription regulation is highly co-ordinated.

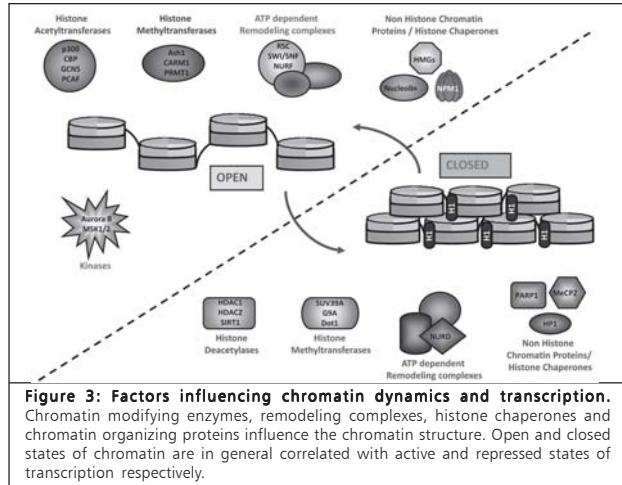


Figure 3: Factors influencing chromatin dynamics and transcription. Chromatin modifying enzymes, remodeling complexes, histone chaperones and chromatin organizing proteins influence the chromatin structure. Open and closed states of chromatin are in general correlated with active and repressed states of transcription respectively.

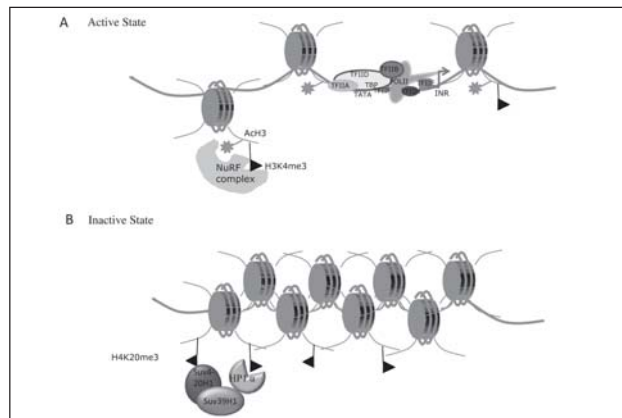


Figure 4: Promoter-specific chromatin states of the genome determine the gene expression pattern of a particular cell type. (A) Active chromatin state: Histone acetylation and H3K4me3 is associated with transcriptionally active genes. Specific modules such as bromodomain and PHD finger in the NuRF complex recognize these modifications respectively. (B) Repressed chromatin state: H3K9me3 and H4K20me3 is associated with transcriptionally repressed genes. Chromodomain of Heterochromatin protein 1 alpha (HP1 α) binds H3K9me3 and recruits Suv39H1 and Suv4-20H1 enzymes which in turn mediate H3K9me3 and H4K20me3 respectively causing propagation of heterochromatin.

Epigenetics and cancer

Now that the genetic basis of most of the human diseases is known, the challenge is to understand the genotype-phenotype relations and how environment affects the phenotype on the common genetic backgrounds. Certainly, epigenetic alterations are the earliest alterations in most of the disease progression states, and function as a platform for other alterations to occur. In fact, there are many developmental and neurological disorders which occur due to altered genomic imprinting patterns.

Cancer is an evolving state of genetic changes of gene expression patterns in the context of cell cycle regulation as a whole. However, most of the cancers are not heritable. There are six hallmarks of cancer which are: unlimited replication potential, evasion of apoptosis, evasion of growth inhibitory signals, angiogenesis, metastasis and growth signal autonomy (Figure 5). In order to achieve these hallmarks, many genes are upregulated or downregulated. Few of these genes are mentioned in the Figure 5. The two basic mechanisms that can lead to cancer are inappropriate activation of proto-oncogenes and silencing of tumor suppressor genes, both contributing positively to the growth of cancer (Figure 6). Apart from genetic mutations in these two classes of genes, many epigenetic alterations have been discovered which drive either of these two mechanisms. Global hypomethylation and local hypermethylation at the promoters (CpG islands) of tumor suppressor genes is a common characteristic of all the human cancer types. Changes in histone modification patterns have also been correlated with various types of cancers as well as different stages of a particular type of cancer. Apart from this, there are several evidences to suggest that various enzymes that modify histones show altered activity in different cancers (Figure 6). For example, missense mutations of the histone acetyltransferase p300 or mutated fusion proteins with MLL drive the unregulated expression of Ras proto-oncogene leading to cancer (Figure 6).

Several studies have reported an enhanced expression of class I HDAC isoforms in solid human tumors. In contrast, the expression of class II HDACs has been found to be reduced in tumors. There are increasing evidences which show that apart from HDACs, acetyltransferase activity of certain histone acetyltransferases (HATs) may have wider roles in cancer manifestation as mentioned above. There are a few instances of altered activity of histone-modifying enzymes resulting in aberrant expression of genes that might have a function in tumorigenesis. Recently, it was reported that changes in global levels of individual histone modifications are associated with prostate cancer, where it was found that H3K18 acetylation and H4K12 acetylation positively correlated with the increasing grade of prostate tumors. Recently, we have discovered that p300 mediated hyperacetylation of histones and histone chaperone leads to oral cancer manifestation presumably through the activation of several genes essential for the establishment of the malignancy. These reports suggest that the aberrant activity and expression of such covalent modifying enzymes can result in cancerous malignancy by skewing the normal epigenetic marks of the chromatin. Apart from acetylation, aberrant methylation of histones is widespread during the process of oncogenic transformation. Mice deficient for the lysine methyltransferase Suv39H1/KMT1A are prone to cancer. In addition to the deregulation in the activities of chromatin modifying enzymes aberrant expression and activity of chromatin remodelers can also contribute to the manifestation of cancer by altering gene expression patterns.

Implications and Future perspectives

All these studies suggest that epigenetic mechanisms contribute equally as genetic alterations in the initiation and progression of cancer. They are also responsible for alteration of gene expression patterns which may lead to pathophysiological conditions. Thus, understanding the epigenetic changes associated with changes from normal

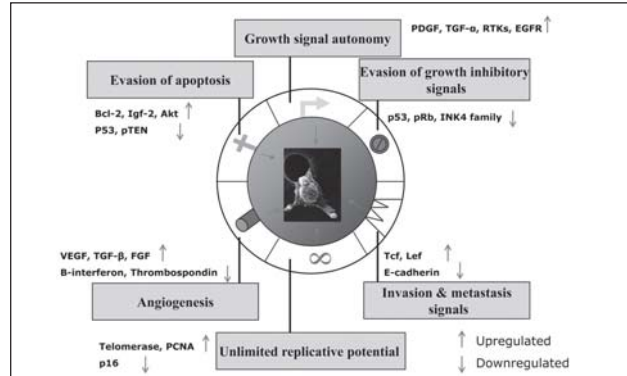


Figure 5: The six hallmarks of cancer. The regulation of several genes is altered in cancer which lead to the six hallmarks as mentioned above. Red arrows indicate upregulated genes whereas green arrows indicate downregulated genes (adapted from Doglous H, 2000).

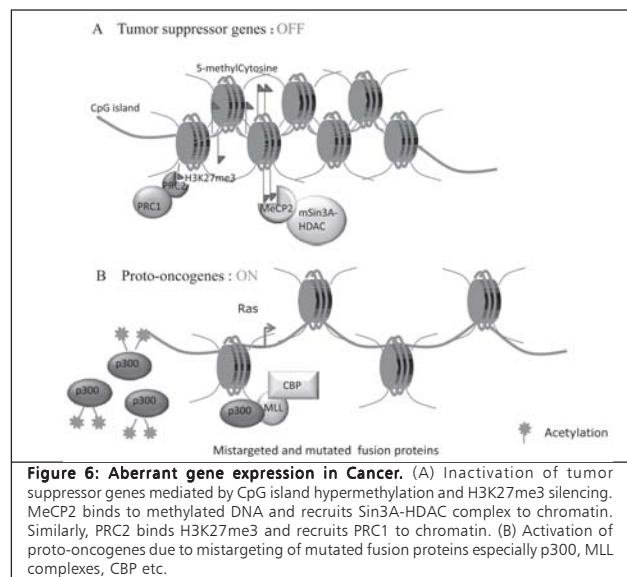


Figure 6: Aberrant gene expression in Cancer. (A) Inactivation of tumor suppressor genes mediated by CpG island hypermethylation and H3K27me3 silencing. MeCP2 binds to methylated DNA and recruits Sin3A-HDAC complex to chromatin. Similarly, PRC2 binds H3K27me3 and recruits PRC1 to chromatin. (B) Activation of proto-oncogenes due to mistargeting of mutated fusion proteins especially p300, MLL complexes, CBP etc.

to diseased phenotype and targeting them specifically will certainly help treat such diseases in addition to the conventional therapy. Chromatin modifying enzymes thus are the new targets for the new generation anti-neoplastic therapeutics especially for better combinatorial therapy. Several small molecule modulators have been discovered and are being tested. These molecules will also be highly useful to understand the global role of epigenetic modifications in transcription regulation.

Further reading

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5. Struhl K. Fundamentally different logic of gene regulation in eukaryotes and prokaryotes. Cell. 1999, 98:1-4.
6. Swaminathan V, Reddy BA, Ruthrotha Selvi B, Sukanya MS, Kundu TK. Small molecule modulators in epigenetics: implications in gene expression and therapeutics. Subcell Biochem. 2007, 41:397-428.



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2009 ANNUAL AWARDS

The Society announced the Annual Awards for 2009 at its Annual General Body Meeting held at Pune during October 30-November 1, 2009. A brief description of the research interest is given below:

P B RAMA RAO MEMORIAL AWARD

Dr. Y.P. Venkatesh's Lab

The research program pursued in Dr. Y. P. Venkatesh's laboratory at the Central Food Technological Research Institute (CFTRI, Mysore) may be broadly defined under the field of 'Food allergy and Immunology' involving three aspects: (1) Molecular immunology of food allergens, (2) Interaction of dietary lectins with mast cells and basophils, and (3) Immunomodulatory agents from plant sources.



Molecular immunology of food allergens: Food allergy affects 2-4% of adults. Due to the diverse nature of the ethnic groups in the Indian sub-continent, a variety of foods are consumed in different regions. A comprehensive study of a rare case of anaphylaxis to the ingestion of pomegranate and mushroom led to the identification of mannitol as a haptenic allergen. Mannitol being non-immunogenic, a hypothesis (wherein the Schiff base conjugate of mannose with protein acts as a *sensitizer*) was proposed to explain the mechanism of sensitization and hypersensitivity to mannitol, followed by validation using allergy diagnostic tests. Further, the validity of this hypothesis for immunogenicity has been tested *in vivo* by producing specific antibodies to mannitol in rabbits, using reductively aminated mannose-albumin conjugate as an immunogen. Similarly, antibodies specific to erythritol and xylitol have been prepared, characterized, and utilized for the development of sensitive immunoassays for specific sugar alcohols in natural/processed foods.

Based on allergy diagnostic tests in atopic subjects (susceptible to allergic diseases) presenting oral allergy syndrome to sapodilla (sapota fruit), the major sapodilla allergen has been identified as an arginine-rich basic protein (21.5 kD thaumatin-like protein or TLP) belonging to the pathogenesis-related (PR-5) family of plant defense proteins. Efforts are focused on delineating the allergenic nature of sapodilla TLP by way of cloning the specific gene.

A cross-sectional study on the prevalence of food allergy to eggplant (brinjal, a native of India) shows that the incidence is 0.8% having a female predominance. About 9 allergens in the 26-71 kD range have been detected (predominantly localized in the peel), of which two glycoproteins (64, 71 kD) have been identified as polyphenol oxidase (PPO). The gene for eggplant PPO has been cloned from green long eggplant cv. Arka Shirish (GenBank Accession GQ246219.2). The deduced amino acid sequence consists of a chloroplast transit peptide (90 aa), and a mature non-glycosylated polypeptide (510 aa; 58.3 kD). Efforts are underway for *Agrobacterium*-mediated transformation of eggplant for PPO silencing using inverse repeat constructs taken from unique sequences of the PPO gene.

Interaction of dietary lectins with mast cells and basophils: Some dietary lectins are known to exert a variety of effects on the cells of the immune system. Studies with horse gram lectin on atopic subjects led to the identification of the lectin as an allergen (Dol b Agglutinin). Certain dietary lectins (potato, banana, and garlic) non-specifically activate and degranulate mast cells/basophils by cross-linking the core GlcNAc or mannose residues of IgE glycans. Higher intake of such foods may increase the clinical symptoms of allergy, as a result of non-allergic food hypersensitivity reactions in atopic subjects whose serum total IgE levels are higher than normal.

Immunomodulatory agents from plant sources: The immunomodulatory proteins present in raw garlic and aged garlic extract have been identified as the major garlic lectins ASA I and ASA II. They display mitogenic activity towards murine lymphocytes, and activation of macrophages. Garlic is also rich in fructans which display immunomodulatory activity. Immunological studies with purified garlic lectins in BALB/c mice (systemic, intranasal, and oral administration without an adjuvant) have shown that they are highly immunogenic in nature. This response seems to explain the earlier reports of the presence of natural IgG antibodies to dietary proteins in humans, since small amounts of garlic are consumed almost universally. Both ASA I and ASA II were found to exhibit adjuvant activity for serum IgG response to a test antigen in BALB/c mice upon systemic or mucosal administration; ASA I is more potent compared to ASA II. Based on the safety of garlic as a spice and the non-toxic/stable nature of garlic lectins, these studies indicate the potential utility of garlic lectins as an adjuvant in vaccines.

C R KRISHNAMURTHY AWARD

Pawan Malhotra

International Center for Genetic Engineering and Biotechnology, New Delhi-110067

Plasmodium falciparum is a wide spread protozoan parasite responsible for over a million deaths annually. The invasion process in *Plasmodium falciparum* is unique, complex and involves multiple, highly specific and sequential interactions. Once the invasion has taken place, the success of the parasite in human host depends on a series of adaptations that enables it evade destruction by the host immune system. Characterization of key genes/proteins involved in invasion as well as in host-parasite interplay should lead to better understanding of these biological processes as well as to develop vaccine and identify new drug targets. In this direction, detailed analysis of extracellular secretory antigens at asexual blood stages of *P. falciparum* was carried-out. To do so, two complementary proteomic approaches, two dimensional gel electrophoresis/MS and LC-MS/MS were tried and the study revealed a cohort of 27 novel proteins with possible role(s) in immune evasion. Of these antigens, preliminary functional characterization data for two novel (Sel1 repeat-containing) parasite proteins, PfSel1 and PfSel2 revealed that they down-regulate expression of cell surface Notch signalling molecules in host cells. Cell binding assays as well as ELISA for cytokine release are being performed to understand the role(s) of PfSel1 and – Sel 2 in innate immunity. Currently work is in progress to express other extracellular antigens and look at their immune-modulation potential. In this direction, we have focussed on characterization of a kinase (PF11-0220), a phosphatase (PF11-0139), PfDegP (a serine protease) and PflCCL domain containing protein. We believe that characterization of some of these extracellular antigens may lead us to develop inhibitors that can block these parasite specific immune-modulators so that parasite cannot survive in the hostile host.



Another objective of the group has been to identify protein complexes [ligand complex(s)] on the surface of merozoites, the invasive form of malaria parasites at asexual blood stages of *P. falciparum*. About 252 proteins have been shown to have expression profiles similar to important malaria surface antigens. Many of these proteins individually have been tested for their vaccine potential. However, an effective blood stage is still a dream. Recently, using a set of approaches, like bacterial two hybrid analysis, *in vitro* interaction study and immune-precipitation analysis, we have identified a molecular interaction between a major *Plasmodium* surface protein, Merozoite surface protein-1 and a rhoptry secreted protein, PfrhopH3 that is a part of a high molecular weight rhoptry protein complex, referred as PfrhopH complex. Mass spectrometry based proteomic analysis of anti-PfMSP-1 and anti-PfrhopH3 immuno-precipitates revealed stable association of eight parasite surface proteins, PfMSP-3, PfMSP-7, PfrhopH1, PfrhopH3, PfrAP-1, PfrAP-2 and two putative Pfrhoptry associated proteins with PfMSP-1. Immuno-staining of merozoites with antibodies raised against each component of the complex showed their presence on the merozoite surface. Presently work is on to functionally characterize the PfMSP-1 associated complex and test the vaccine potential of isolated complex. If successful, we plan to express each protein and then study their vaccine potential. In addition, we have also isolated a hemozoin associated protein complex from *P. falciparum* and work is on to know the determinants of this complex.

AMAR BHADURI MEMORIAL LECTURE AWARD

Dr. Snehasikta Swarnakar

Indian Institute of Chemical Biology, Kolkata

Gastric inflammation is the physiologic response to injury caused by wounding, chemical irritation, or infection. Gastric inflammation predisposes the stomach to both ulceration and in more advanced cases to adenocarcinoma. Gastric ulceration affecting ~15% population is one of the major socio-economic burden to Asian countries. It is induced by various factors including alcohol, non-steroidal anti-inflammatory drugs (NSAIDs), stress and infection by *Helicobacter pylori*. In recent years there is a paradigm shift in understanding the mechanism of gastric ulceration because acid secretion is no more considered as major factor for gastric ulceration. An imbalance between damaging factors and the gastroprotective factors is responsible for gastric damage. The endogenous damaging factors mainly include HCl, pepsin, refluxed bile, leukotrienes and reactive oxygen species (ROS). Damage of lipid, protein and DNA by ROS has been well studied in gastric ulcer and, recent studies indicate that apoptosis plays a significant role in gastric ulceration. It is rather a multifaceted process including acid-secretion, ROS production, prostaglandin inhibition and extracellular matrix (ECM) degradation. The latter is governed by matrix metalloproteinases (MMPs), a family of zinc dependent endopeptidases that play a critical role in cancer metastasis, rheumatoid arthritis, and embryogenesis. However, the underlying mechanism regarding the pathogenesis of gastric ulcers remains unknown in the context of MMPs regulation. Among the MMP family, gelatinases (MMP-2 and -9) stromelysin-1 (MMP-3) collectively can cleave ECM proteins including elastin, fibronectin, laminin and collagen type I, IV, V, VII, XI. Gene expression of MMP-9, -3 and -2 is promoted by many factors including cytokines and ROS via



activation of transcription factors. Catalytic activity of MMP enzymes are tightly controlled at multiple levels, including gene expression, spatial localization, zymogen activation, and inhibition by tissue inhibitors of metalloproteases (TIMPs). The delicate balance between MMPs and TIMPs is critical to limit pathological outcomes.

Dr Swarnakar's work is focused on new mechanism of gastric ulceration via regulation of MMPs. The breadth of research ranges from the understanding of basic mechanism of gastric ulcers to clinical approach for disease prevention. Her major contribution to the development of scientific material is the association of MMP-9 gene upregulation to the pathogenesis of NSAIDs-induced, ethanol-induced and *Helicobacter pylori* infected gastric ulcers. Her work established that MMP dependent pathway in addition to MMP independent pathway for gastric ulceration. She has also demonstrated the novel mechanism for *cag* pathogenicity island negative *H. pylori* infection that caused elevation of proMMP-9 and -3 activities in mouse gastric tissues. She has revealed through her research a common ancestral pathway for inflammation by both *cag*-ve and +ve *H. pylori* strain that include MMP-9 upregulation. Furthermore, her interest has been on gastric inflammation in relation to redox signalling. The work highlighted the critical role of antioxidants in gastroprotection via regulation of MMP-2 gene. The cross talk between ROS and MMP-2 was found to be a major signalling mechanism of gastric damage, and antioxidants regulate MMP-2 activity during gastroprotection. She has shown that omeprazole, curcumin (a constituent of 'haldi') and melatonin (N-Acetyl-5-hydroxy tryptamine), regulate redox-mediated MMP-2 expression during gastroprotection while famotidine but not the drug omeprazole protected gastric ulcer via MMP-9 dependent pathway. The quality of ulcer healing remains a challenge. Her research work detailed the critical role of melatonin in regulating MMP-3 activity via TIMP-1 during protection and healing of acute as well as chronic gastric ulcers. The action of curcumin and melatonin in arresting the MMP-dependent pathway in healing gastric ulcer may be an alternative approach for better therapeutic that limits recurrence of the disease.

BRANCH ACTIVITIES - 2010

SPEAKER	TOPIC
BANGALORE	HEADQUARTERS
Dr. Ananda Sarkar, USA	Stem Cell regulaton and Organ patterning in higher plants
Dr. Dhan V Kalvakolanu, USA	DAPK, Autophagy and the transcriptional control: Roads to control metastasis
Dr. Ennio Carbone,	NK Cells and tumor progression
Dr. Jayanta Roy Chowdhury, USA	Liver repopulation with transplanted hepatocyte: a novel approach for cell therapy
Dr. Lashitew Gedamu, Canada	Structure and functional analyses of custeine proteases of Leishmania donovani complex
Ms Mary Riley	Flash gel system and precast gels for nucleic acid and protein electrophoresis
Dr. Neva Elizabeth Haites Obe, UK	Advances in medical genetics and stem cell research and their regulation in the UK
Dr. Ram Laxman, Singapore	Enabling genome biology with illumina's solexa sequencing technology
Dr. Renuka Pudi, USA	Prostate Cancer stem cells: role of developmental signaling pathways
Dr. Samit Adhya, Kolkata	How large RNAs can be directed to the defective mitochondria
Dr. Siddappa Negedanahalli, USA	Shiv models and HIV
Dr. Suprana Sanyal, Sweden	The key role of the ribosomal stalk in bacterial translation initiation
Dr. Suresh C Jhanwar, USA	Cancer Genetics I pathology and translational medicine
Dr. Ueli Grossniklaus, Switzerland	Cell- Cell communication during double fertilization : a surprising link to disease resistance

COIMBATORE**Dr. Parvatham: Convenor**

Mr. Anantha Padmanabhan Coimbatore	The global perspectives and career opportunities in clinical research.
Dr. Anantha Subramanian, Coimbatore	Expressed sequence tags (EST) A poor man's genome
Ms M P Bhavani, USA	Role of FGF Receptors on lens development
Dr Gowaskar Bhojan, Coimbatore	PHP& SQL
Dr. Hisato Okuizumi, Japan	Molecular Analysis of Genomie- Wide scanning
Dr Hyung- Kyoon Choi	Applications of NMR and MS based metabolomics in natural product sciences
Dr N Jeyakumar, Coimbatore	Microarray informatics
Dr G P Jeyanthi, Coimbatore	Statistical Tools appropriate for scientific Research
Dr. K Kalaiselvi, Coimbatore	Nutrigenomics – an Indian Scenario
Dr Kamala Nehru, Coimbatore	Fundamentals of Mathematics
Dr. N Manikanda Boopathi, Coimbatore	Applications of Proteomics in Genetic improvement of crop plants, (present positions, problems and prospective)
Dr N Manivannan, Coimbatore	Plant Breeding and Genetic diversity
Dr Manivel ponnuchamy, Gujarat	Breeding of medicinal plants in Inda: current Scenario and Future Opportunities
Dr Moon - Ki Park, Korea	Cosmeceutical Material Bank
Dr. P R Padma, Coimbatore	Silence in gold- is it not costly?
Dr. P R Padma, Coimbatore	Literature Collection: The backbone of your research
Dr J Reeves Wesley, Coimbatore	Scaling, sampling, samples and non parameteric test
Dr. R Selvam, Thiruchirappalli	Advances in Nanomedicine
Dr Tae-Sun Min, Korea	Korean bioindustry & B T R&D policy
Dr. K Umaa, Coimbatore	Drug Design & QSAR- An overview
Dr K Usha, Coimbatore	Report Writing
Mrs Vidhyalakshmi, Coimbatore	Stem Cell therapy
Dr. Yung Base Seo	Introduction to Korean traditional medicine

JORHAT**Dr. B G Unni: Convenor**

Prof. Alexander Aleshin, Moscow	Kalinigrad Earthquake, September 21, 2004
Prof. Alok Srivastava, Chandigarh	Nanotechnology from the prospective of a Nuclear chemist
Ms Amrita Difusa, Guwahati	Application of stem cell Research in the Health Sector
Dr. Davleena Shivkumar, USA	Prediction of Cytochrome P450 mediated Oxidation using Induced Fit Docking
Prof. Debashis Basu, Chandigarh	Pharmacogenetics
Dr. J. N. Goswami, Ahmedabad	Chandrayaan-1
Dr. Kankan Bhattacharyya, Kolkata	Rise of Modern Science In India

Dr. J.R.Kayal , Kolkata	Seismotectonics of NE India: A review".
Dr Madhavi Shastri , USA	Recent Advances in Cheminformatics methods
Prof. Partha Banerjee , USA	Epigenetic Silencing of RASSF1A and its restoration by Methylation.
Prof.Parul Chkrabarty , Kolkata	Biotechnology in 21 st Century-Promises & prospects
Prof. Pinakpani Chakrabarti , Kolkata	Protein folding, threading & Prediction of Structures
Prof. Sameer Bhattacharyya , Kolkata	Why we should concentrate effort on cancer Research
Prof. Sergei Arifiev , Russia	Studies on seismic sources: current state, problems and perspectives"
Dr. Shika Rastogi , New Delhi	Protecting intellectual property: Chemical and Biological inventions
Dr. K. Venkataraman , Chennai	Biodiversity Act 2002
Mr. K C Velappan , Chennai	Non- conventional fuels with reference to Bio-fuels
Prof. J.D.Woollins ,UK	From P-S to P-Se Chemistry: The Development And Applications of "Woollins' Reagent".

KARNAL

Dr. Suman Kapila: Convenor

Dr. Jens Vanselow , Germany	Role of estrogens and regulation of the bovine aromatase gene during bovine reproduction
Dr. Sunil Bhand , Goa	Environmental analysis using Bio-Mems

NAGPUR

Dr. M Patil: Convenor

Dr. Amul Sakharkar , USA	Brain chromatin remodeling, a novel mechanism in alcoholism
Dr. Anup Kale	Lighting up the cell with semiconductor quantum dots: Interfacing nano materials with biology
Dr. Arvind K.N. Nandedkar , USA	Saliva, a specimen of choice for monitoring health & diseases
Dr. Dipanwita Pal , USA	Regulation of protein kinase C ϵ
Dr. Soniya Gavande	Lighting up the cell with semiconductor quantum dots: Exploring roles nano materials in biology
Dr. Sunil Panchal , Australia	Antioxidants and Metabolic syndromes



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