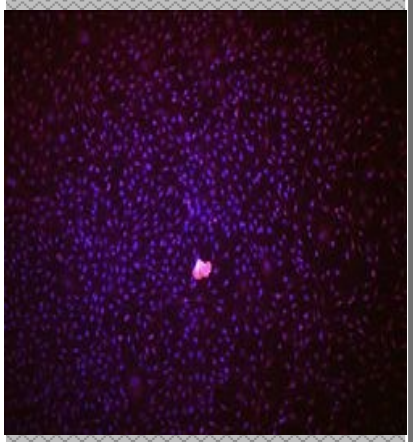

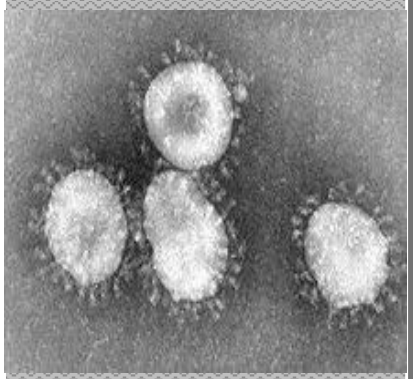

EMGEN Newsletter

Vol. 5, Issue 1

IN THIS ISSUE:

Eastern Mediterranean Health Genomics and Biotechnology Network (EMGEN) was created in 2004 with collaboration of representatives of selected centers of excellence in (health related) molecular biology, biotechnology & genomics in the Eastern Mediterranean region by recommendations and efforts of WHO/ EMRO. Sponsored by Iran Biotechnology Development Council.

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Article

ALTERATIONS OF P53 (GENE MUTATION AND PROTEIN EXPRESSION) IN ORAL EPITHELIAL DYSPLASIA

The paper entitled: "Alterations of p53 (Gene Mutation and Protein Expression) in Oral Epithelial Dysplasia" which is published in the Journal of Oral Pathology (33 (2), 260-265) aims to determine the frequency of p53 gene mutation and protein expression in oral epithelial dysplastic lesions. The study was carried out by Dr. Nighat Ara Bukhari from the Sardar Begum Dental College, Peshawar, Pakistan.

Dr. Nighat Ara Bukhari

Oral cancer is frequently preceded by precancerous lesions, predominantly leukoplakia (white lesion) and erythroplakia (red lesion).

Follow-up research has indicated that carcinomatous transformation usually occurs 2 to 4 years after the start of white plaque, but it may arise during months or even after decades. These lesions show dysplastic changes in the squamous epithelium under microscopic examination.

Oral Epithelial Dysplasia (OED), as defined by Pindborg, is a 'lesion in which architectural disturbance of the epithelium is accompanied by cytological atypia'.

Clinically leukoplakic and erythroplakic lesions show a range of histopathological features under the microscope, varying from non-dysplastic epithelial hyperplasia or hyperkeratosis, eventually leading to epithelial dysplasia.

The aim of the study was to determine the frequency of p53 gene mutation and protein expression in OED lesions. This descriptive study was carried out at the Histopathology Department and Molecular Biology Laboratory of the Armed Forces Institute of Pathology (AFIP), Rawalpindi.

Thirty cases of OED were retrieved from the archives of AFIP. Some fresh/frozen sections were also included. PCR-SSCP analysis was performed to detect mutations in the gene p53. DNA was extracted from the formalin -fixed and paraffin-embedded tissue sections and fresh/frozen sections.

The extracted DNA was amplified by PCR. DNA of all samples was amplified by using forward & reverse primers of Exon 5, Exon 6, Exon 7, Exon 8 and Exon 9 consecutively. These samples were then denatured and immediately chilled on ice.

Gel electrophoresis was performed at 100V for 20 hours at room temperature. A single nucleotide change (gene mutation) discovered as electrophoretic mobility change. The primer couple of Exon 5 of the p53 gene mutation expanded a 167bp fragment. Colon carcinoma was taken as positive control and human DNA (free of p53 mutation) was used as negative control. Gene p53 mutation was present in 6 (20%; CI: 7.7% - 38.6%) cases while in 24 (80%; CI: 62.3% - 90.9%) cases p53 gene mutation was absent.

The immunohistochemical marker of p53 was applied to the same 30 cases. The p53 immunoexpression was seen in 60% (42.3% - 75.4%) of the cases.

Genetic studies applied to the detection of mutations in precancerous lesions, have opened new windows in our understanding of the detrimental effects exerted by the carcinogens on the mucosa of the oral cavity. Several studies worldwide have revealed that the presence of mutation is a definite indicator of progression to malignancy.

A simple and cost effective PCR-SSCP analysis can be used to find out whether mutation is present or absent in the precancerous tissue. If mutation is detected, the dysplastic lesion can be treated and cured in early stages and progression to cancer can be reduced. The early detection of mutations in dysplastic lesions can save mutilating ablative surgeries and may improve the survival rate of patients.

If we could to detect the mutation in the dysplastic lesions, then an early surgical management of these lesions will prevent their progression to cancer and hence avoidance of mutilating ablative surgery. It will improve the prognosis of these patients and thus the survival rate.

Interview

In this issue, we present the following interview with Dr. Ziad W. Jaradat from the Jordan University of Science and Technology. (any views or opinion expressed are solely those of the author and do not necessarily represent those of EMGEN Newsletter).

Dr. Ziad W. Jaradat

1. Please introduce yourself and explain your scientific discipline.

I am Ziad W. Jaradat, an associate professor in Microbiology and Biotechnology. I have obtained a B.Sc. in Public Health/Nutrition from Yarmouk University, Jordan, and an M.Sc. and Ph.D. in Microbiology and Biotechnology from the University of Manitoba, Canada. Upon finishing graduate studies, I have joined Purdue University, West Lafayette, Indiana, as a postdoctoral research associate for almost three years and then worked in a biotechnology firm in San Antonio, Texas for two years. After completing the North American education and training, I decided to go back to academia were I joined the Jordan University of Science and Technology (JUST). At JUST, I taught many biotechnology, microbiology and related courses. I have supervised 18 M.Sc. students as main advisor or co-advisor. Since the beginning of my academic career, I have co-authored around 38 Journal papers, a book and authored 3 book chapters in the Toxicology, Microbiology and the Biotechnology fields. For the past two years I headed the Academic Affairs of Fatima College of Health Sciences and AlA in Campus of the college in the United Arab Emirates. Currently, I am associate editor in chief of the International Journal of Life Sciences and Medical Research and sitting on the editorial board for Advancements in Genetic Engineering, Advances in Microbiology Research, International Journal of Biotechnology and Food Sciences and the Universal Journal of Microbiology.

2. Could you please tell us what your main research area is?

My main research area is molecular and epidemiological typing of *Staphylococcus, Cronobacter* and *Salmonella.* My research also focuses on the antibiotic resistance profiles and other virulence characteristics of these bacteria.

3. Why did you choose this field of research?

I have chosen this field of research as it is exploring bacteria closely associated with human beings and considered dangerous. Therefore, studying it might help in revealing some of its pathogenic characteristics that will be reflected positively on the health and welfare of humanity.

4. Do you apply any biotechnology or genomics tools in your researches and please explain how?

In fact, most of my research comes under the umbrella of biotechnology. Yes, I do use genomics in my research to ascertain the correct identification of the pathogen or to look for the presence of a gene encoding for a certain pathogenic character or a distinguishing maker.

5. What kinds of biotechnology facilities do you have in your laboratory?

My laboratory has some biotechnology facilities including PCR, DNA and protein gel electrophoresis, and gel documentation system. In addition, my students have access to DNA sequencers, nanodrop and other instruments in adjacent laboratories and the Princess Haya Biotechnology Center.

6. Are there any late stage biological products to be commercialized in your center?

Not that I know.

7. Are there significant biotechnology centers in your country?

Yes, there are a few biotechnology centers such as Princess Haya Biotechnology Center at the Jordan University of Science and Technology.

8. Are there any academic training courses in biotechnology in your country? If yes, to above question, at which level and how many students are trained annually?

Yes, there are several universities offering B.Sc. degrees in biotechnology. For example, the Jordan University of Science and Technology offers B.Sc. and M.Sc. in biotechnology and genetic engineering, with a total number of students at both levels reaching 500. The department graduates around 100 students per year. There are also other universities in Jordan that offer either B.Sc. or M.Sc. in biotechnology. I do not have figures for the enrolment and graduates of these universities.

9. Are you familiar with EMRO countries and EMGEN (Eastern Mediterranean Health Genomics and Biotechnology Network)? Would you please tell us how you know EMGEN?

Yes, I am familiar with EMRO and the EMGEN network from the emails that I receive and some information from the website.

10. Do you have any suggestions for establishing/extending collaborations with EMRO countries?

(I) To initiate a regional conference on biotechnology that is held each year in one of the member states. (II) To have a vigorous advertising campaign for enrolling every biotechnologist in these countries in its database. (III) Initiating a qualified journal that publishes the research outcome in these countries; and of course, for the world. It would give it a momentum and will certainly maximize its impact not only in the Middle East but worldwide. (IV) Initiating a consortium of biotechnology journals that is available for members.

11. Are there any possibilities for young researchers from EMRO countries to participate in training courses in your biotech centers?

Yes, there is possibilities and it could be done with the proper communication with the administration of the Princess Haya Biotechnology Center at the Jordan University of Science and Technology.

12. What kinds of difficulties do you face, in research and commercialization of medical biotechnology in your country and the region?

The biggest difficulty here in Jordan is the teamwork. I mean to be able to commercialize products, a big team effort is needed and that is affected by promotion rules and regulations and some other intrinsic issues.

13. Do you have any training courses or workshops in your research center?

The Biotechnology Center which is a part of the Jordan University of Science and Technology is adopted as a training center for many biotechnology techniques and workshops for students in Jordan and out-side organizations such as WHO and others.

14. What is your idea about genomics and its applications in improving public health?

Certainly, the field of genomics is pivotal for public health. It has already been in use for pre-marriage testing in thalassemia. In addition, it has been used for testing for the presence of many other genetic disorders. In the near future, the biotechnology center and the graduates from departments of biotechnology in Jordanian universities will certainly have its big share in improving public health in Jordan and will hopefully be a tool for preventing marriages that predispose to many of the genetic disorders.

15. What is your idea about commercialization of researches in the field of bioscience?

It is a great idea to initiate spin off companies as a yield of the bioscience research. However, in order to ripe the fruit of such a research, we need to have organized groups of researchers for certain fields from different areas; but, close disciplines with a strategic plan that is advertised among the scientific community in order to initiate the research groups accordingly and to have a good source of funding will be helpful.

16. What is your opinion about the development of the biotechnology and genomics in your place?

Certainly there is development in this field, however, it is moving at a slow pace.

17. Would you please tell us about the differences of genomics and its applications between developed and developing countries? What should we do in this regard?

One of the major differences between developed and developing countries are the lack of communication between academia and industry in developing countries as opposed to the excellent connections between these two entities in the well-developed countries. In addition, as I said in my answer for question 8, there is the lack of overall teamwork. To overcome these obstacles, I think the aim of the research should be modified i.e., to move it from being done merely for promotion purposes; and every effort should be exercised to bridge the gap between academia and industry. For that, I urge the industry to start by approaching the academics with their problems seeking solutions or the vision of new products from academics rather than the other way around.

Thank you Dr. Ziad W. Jaradat for sharing information and your opinion with us. Also, we are grateful for your kind and useful cooperation.

APOPTOSIS

The term of apoptosis was first introduced by a researcher named Ker to describe physiological cell death based on morphological changes and its difference from necrosis. Apoptosis is a Greek word and it means shedding the leaves. In most of the references, two words i.e. apoptosis and programmed cell death are used as synonyms; in some others apoptosis also figured as the most important kind of programmed cell death. These researchers have used programmed cell death as a general term to describe physiological cell death and classified it in terms of the causative agent of cell death, action mechanism, morphologic and biochemical changes, into two kinds of apoptotic and non-apoptotic programmed cell death.

Apoptosis is physiological cell death which in normal conditions provokes elimination of aged, damaged, excessive and harmful cells and is essential for tissue development and homeostasis. Apoptosis plays a role in tissue repair and renovation and elimination of self-reacting T cells. Any disorder in the apoptosis process will result in a disease that could be caused by reduction in cell death which gives rise to emergence of cancer cells and their growth or autoimmune disorders. Contrarily, unnatural increase of cell death is seen in diseases like neurodegeneration and AIDS. Chemotherapy drugs cause apoptosis induction in cancer cells.

Apoptosis and necrosis

Two main mechanisms of cell death are apoptosis and necrosis and their differences are the following:

1. Apoptosis is physiological cell death and occurs in special stimulations. While necrosis is pathological cell death and this kind of cell death occurs in severe damages such as hypoxia, hyperthermia and external toxins. Necrosis is an inactive process and occurs in the absence of ATP, while apoptosis is active and is energy dependent.
2. In apoptosis a cell becomes smaller and wrinkled, while in necrosis, it will be swollen and bigger.
3. In apoptosis the cytoplasmic membrane envelops apoptotic bodies, while in necrosis membranes will be destroyed causing internal cell contents to be released.

4. Chromatin is compressed and divided in apoptosis.
5. Cytoplasmic organelles remain intact in the process of apoptosis, while they will be demolished in necrosis.
6. Tissue necrosis is associated with inflammation responses, while it occurs without inflammation in apoptosis.

Apoptotic pathways

A. External pathway

The plasma membrane of most cells include death receptors. Death receptors are members of tumor necrosis factors (TNF) receptor super family. When these TNFs are activated with the related ligands, they result in activation of caspases and apoptosis induction. Existence of a cysteine sequence in the exterior section of the cell is the specialty of this super family. These receptors have a sequence called death domain in their cytoplasmic part; hence they participate in transferring apoptotic communication into the cell. The most notable death receptors are: TNFR2, DR4/TRAIL-R1, TNFR1, CD95/Fas/Apol, and DR5/TRAIL-R2 (Fig.1).

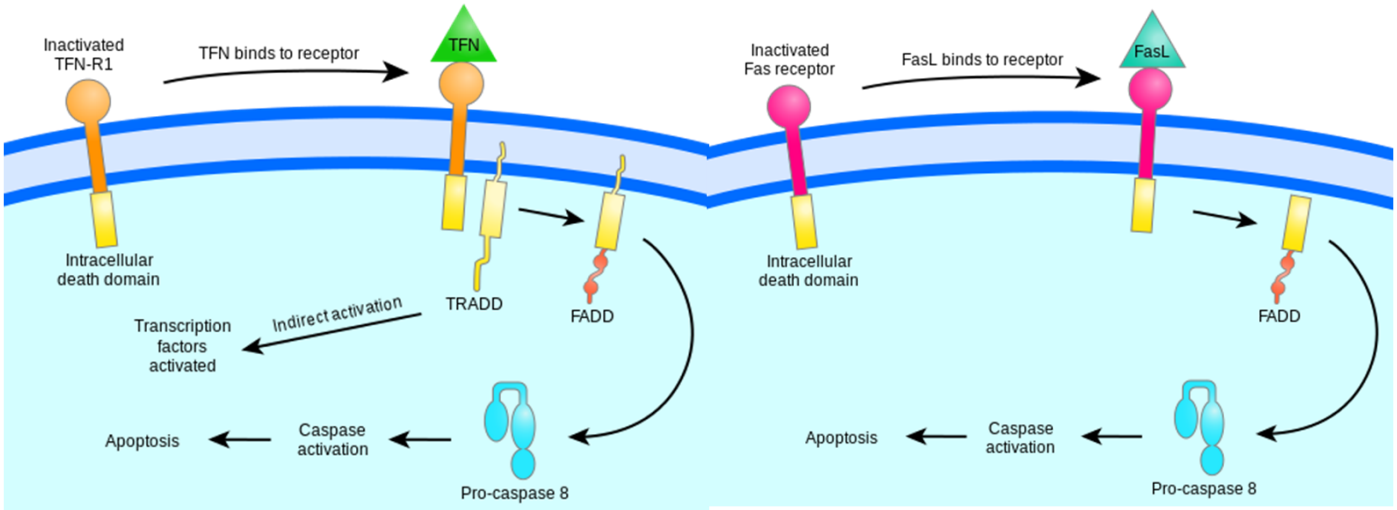

Fig. 1: Overview of TNF (left) and Fas (right) signaling pathway.

Stimulation of death receptors through related ligands, leads to receptor trimerization using adaptor proteins. For example, ligand CD95 (CD95L) interacts with the related receptor and induces its trimerization. This action gives rise to death cell cluster in the cytosolic domain of the receptor and causes adaptor proteins such as FADD/Mort1 to connect to it. FADD has death domain (DD) in C-terminal that enables this protein to connect to the trimerized receptor through death domain-death domain interaction. This protein also, via N-terminal including "death effective domain" (DED), interacts with similar DED domain in Caspase-8 prodomain. These protein complexes (ligand- death receptor, adaptor molecule and pro-caspase) are called "death inducing signaling complex" (DISC).

In this manner, Pro-caspase-8 turns into active Caspase-8. Caspase-8 provokes activation of executive caspases and as a result apoptosis takes place.

B. Internal pathway

Mitochondrion is involved in both life and death of the cell. This organelle provides current energy of the cell in the form of ATP. It maintains intercellular homeostasis relating to ions and oxidative stresses. In response to cell death signals, permeability of extra membrane causes releasing of pro-apoptotic molecules such as apoptosis inducing factor (AIF), Cytochrome C, Smac/DIABLO and endonuclease G from between two mitochondrion membranes into the cytoplasm. Smac/DIABLO imposes an antagonistic effect on caspase inhibitors. Apoptosis inducing factor is a 57 kilo Dalton flavo-protein which in natural conditions has an anti-oxidant role in mitochondrion.

It is suggested that released AIF from mitochondrion during the apoptosis process causes damage to nuclear DNA in an independent pathway of caspase. Release of cytochrome C seems to be a common event in apoptosis and the mechanism controlling its release is under consideration. Possible mechanisms are opening mitochondrial permeability transfer pore, existence of special channels for cytochrome C in extra membrane of mitochondrion and/or swelling and wearing out of mitochondrion extra membrane without loss of membrane potential.

Under normal conditions, Apaf-1 exists in the cell in an inactive form and in the apoptosis process becomes active due to cytochrome C release from mitochondrion. Molecular weight of this protein is 130 kilo Dalton and includes CARD domain in the amine end and several repetitive sections of WD-40 motif in the carboxylic end. Interaction between this protein and cytochrome C is essential for activation of Apaf-1 protein. With a mechanism which is not completely known, cytochrome C and dATP/ATP make Apaf-1 to oligomerize. CARD sequence of procaspase-9 causes activation of administrative caspases such as caspase 3 and 7. The collection of protein Apaf-1, cytochrome C and Procaspase-9 is called Apoptosome.

Caspase 9 is the starter caspase in the internal or mitochondrial pathway. Active caspase 9 causes activation of executive caspases (Caspase-3, 6, 7) and as a result executive caspases act on their substrates and apoptosis takes place.

In some cell lines, caspase-3 independent apoptosis occurs following treatment with different compounds; while apoptosis is dependent on caspase-3 in most cell lines. Possibly cytotoxic T-lymphocytes and natural killer cells (NK-cells) prompt apoptosis through a pseudo-receptor dependent pathway in target cells. In this state, Ger-enzyme B (GrB) (which is a serine protease) containing granules is delivered to plasma membranes of object cells along with porphorine.

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NEW GENERATION OF GAP-PCR:

GAP-PCR COUPLED WITH HIGH RESOLUTION MELT (HRM) ANALYSIS IN THALASSEMIA

Thalassemia is one of the inherited blood disorders in which the body makes an abnormal form of hemoglobin, a protein that is responsible for transporting oxygen. There are many different forms of thalassemia each including different subtypes.

In α-Thalassemia disorder the synthesis of α-globin protein is defective or missing. In people with α-Thalassemia-1 Southeast Asian (SEA) trait, which is a genetic disorder with a high frequency in the Asian population, parents who both carry the α-Thalassemia gene, are at risk of having a severe and lethal form of α-Thalassemia child, called Hb Bart`s hydrops fetalis who dies either in utero or shortly after birth. Furthermore, the high percentage of mothers carrying the fetuses affected by α-thalassemia-1 homozygotes will develop toxemia through their pregnancy. These types of mutations, which have specific overlap in hematological data, cannot simply be diagnosed by a single laboratory test. Therefore, a rapid and reliable technique of prenatal diagnosis to identify the carrier of this type of α-thalassemia is highly requisite.

GAP-PCR

A reliable diagnostic test for detecting the carriers of α-Thalassemia-1 gene is GAP-PCR, which is a DNA-based analysis applied for preventing and controlling this severe form of α-Thalassemia homozygotes. Gap-PCR is a mutation type molecular diagnostic test for detecting the common forms of alpha thalassemia deletion mutations when the deletion break points have been defined already.

Primers in GAP-PCR

The primers are specific for the most common alpha thalassemia deletions such as Hb Lepore (delta/beta globin gene crossover), HPFH (large deletion) and some alpha gene duplications.

These primers are flanking a known deletion, producing an amplicon which is shorter than wild type (Table 1).

Table1: Forward and Reverse Primer sequences at concentration of 400 nM, used in GAP PCR coupled with HRM analysis using SYBR Green1 to detect α-Thalassemia-1 Southeast Asian (SEA) trait.

Primer	Sequence (5′→3′)	GenBank accession No.
Forward α-thal-1 SEA	AGA AGC TGA GTG ATG GGT CCG	Z84721
Reverse α-thal-1 SEA	TGG ACT TAA GTG ATC CTC CTG CCC	Z69706

The disadvantage of conventional GAP-PCR is that the technique requires post PCR analysis which is rather time consuming.

In a recent study, Melting Curve Analysis (MCA) with high sensitivity, is applied to detect thalassemia 1 trait. The analysis of MCA data is based on the number of peaks detected by comparing with the normal subjects (αα/αα), with two peaks of normal fragments at specific T_m value. For example, for one form of the α-thalassemia 1 trait, the Southeast Asian type of α-thalassemia 1 trait ($--^{SEA}$/αα), three peaks could be identified, the two peaks of normal fragments and an additional corresponding peak indicating the deletions.

High Resolution Melt (HRM)

A rather novel assay is reported in which the GAP-PCR is followed by another rapid and reliable assay, called High Resolution Melt (HRM) Analysis. The technique is real-time gap-PCR based, cost-effective with high resolution and accuracy, simply analyzing the amplified fragment without gene sequencing that is rather time consuming. In this single-tube multiplex real-time PCR, the α-thalassemia-1 mutant allele can be detected from wild type α-globin gene allele simply by amplifying the DNA samples using a double stranded nucleic acid stain, SYBR Green-1 fluorescent dye, which is low cost compared to other probes applied in FRET (Fluorescence Resonance Energy Transfer) and TaqMan.

The assay will be continued by analyzing the melting curve of each allele with specific peak heights, to distinguish the wild type from heterozygotes or carriers. The fluorescent activity of SYBR Green-1 is highly dependent on the presence of double-stranded DNA fragments. The DNA amplification during PCR is being monitored and quantified by the fluorescent activity of the mentioned dye. By decreasing the PCR temperature the amplified DNA fragment is denatured and the fluorescent dye released, resulting in dropping of fluorescence level.

Post PCR curve analysis is used to detect non specific products by applying a florescent dye which is not a novel approach. The assay is established on the concept of T_m of the DNA duplex at which 50% of the DNA sample is double-stranded. The amount of T_m is dependent on the length and GC content of DNA. However, in novel HRM assays the sensitivity and accuracy of this technique has been improved. In conventional PCR curve analysis which is called Low Resolution Melt (LRM), the temperature step increases 0.5^{o}C, whereas in recent HRM the temperature is reduced to 0.008 - 0.2^{o}C increments which allows a more sensitive analyzing. Furthermore, in HRM a wide range of DNA intercalating dyes are used to increasing the sensitivity and reliability of the technique.

Other applications of HRM include SNP genotyping, DNA mapping, DNA fingerprinting, DNA methylation analysis and some other molecular assays.

The Intercalating Dyes in HRM

The intercalating dyes are required to be used in HRM to provide data on the melting characteristics of DNA target instead of giving information on signal-to-noise ratio which is important in other PCR based techniques including qPCR technique. The amplification efficiency is also not the key point in HRM but in its place the intercalating dye should not change the T_m of the amplicon.

The most common dye used in HRM is a non-saturating SYBR Green-1 fluorescent dye with the limitation that a high concentration should be applied. To overcome this, saturating dyes which do not alter the T_m of the product have been applied. These dyes, including SYTO9® and LCGreen®, can be used at higher saturating concentrations than SYBR Green-1 which is used in HRM.

Data Analysis

Multiple sample analysis is possible in HRM by selecting an accurate software. To properly align and analyze data, a range of fluorescence readings should be defined properly including pre-and post-melt areas, without selecting melt region, the HRM should be repeated without amplification rate to adjust temperature range. Following this step, the results must be normalized.

Summary

To detect one of the most high frequency genetic disorders in the Asian population, α-Thalassemia-1 Southeast Asian (SEA) trait, the conventional PCR based technique is coupled with a High Resolution Melt (HRM) Analysis, which is a rapid assay with high resolution and accuracy.

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A SPECIFIC TREATMENT OF CERVICAL CANCER CELLS BY NANOPARTICLES

A research group at the National Autonomous University of Mexico, Cell Differentiation and Cancer Unit, established a therapy by targeting the cervical cancer tumors in an animal model study. The treatment consists of a protein, interleukin-2 (IL-2), encapsulated in a nanostructured composition that is toxic to cancer cells. The mechanism involves activating an antitumor response, in which nanoparticles act as a bridge between tumor cells and T lymphocytes, according to the head of the project, Rosalva Rangel Corona. The nanoparticle causes the accumulation of interleukin-2 near the tumor site by carrying it to receptors on cancer cells. The side effect of the nano vector is reduced compared to the large amount of the protein administered, as it does not enter the blood flow. The treatment using the nanoparticles would be beneficial for immunosuppressed patients with insufficient interleukin, but still pharmaceutical approval is required to provide for public prescription.

Reference: http://www.sciencedaily.com/releases

CANCER STEM CELLS DESTROYED BY CRYOABLATION AND NANOPARTICLE-ENCAPSULATED ANTICANCER DRUG

A research team at the Biomedical Engineering Center of the Ohio State University suggested an effective combination therapy, applying nano drugs chemotherapy and cryoablation together, toward suppressing the cancer stem-like cells (CSCs) with higher safety and effectiveness compared to the conventional therapies. Cryoablation also known as cryotherapy or cryosurgery is a low invasive surgical technique in which the damaged tissue is treated irreversibly by freezing to subzero temperature. This technique is mostly applicable in breast cancers with minor tissue scar formation following the surgery. The new strategy could increase the destruction of CSCs, with very promising results in the elimination of almost all of the damaged cells. Nano drugs with a positive charge are attracted to the negatively charged cell membrane, specifically to the CD44, a protein that is highly expressed on CSCs. Future studies will focus on *in vivo* studies to monitor the tumor decline at micro scale following the combination therapy.

Reference: http://www.sciencedaily.com/releases

THE GENES IN HUMAN SKIN STEM CELLS BEHIND THE INTERNAL CLOCK

The human skin stem cells have an internal clock that permits them to very accurately know the hour of day and helps them know when it is best to carry out the correct function. This is significant because it seems that tissues require an precise internal clock to stay healthy. A diversity of cells in our body possess internal clocks that help them carry out particular functions depending on the hour of day, and skin cells as well as some stem cells represent circadian behaviors. Researchers already discovered that animals missing usual circadian rhythms in skin stem cells age prematurely, representing that these cyclical models can protect against cellular injury. Also, they indicate that specific genes in human skin stem cells indicate peak activity at different hours of the day. Genes engaged in UV preservation become more active over the daytime to protect these cells while they are duplicating; that is, when they amplify their DNA and are more sensitive to radiation-induced injury.

Reference: http://www.sciencedaily.com/releases

A GENE FAMILY DISCOVERED TO SUPPRESS PROSTATE CANCER

According to a research study, miRNA gene family, MicroRNA-34 (miR-34), were reported to be effective in tumor suppressing. According to the previous findings, p53 is also shown to be involved in positive regulation of miR-34, since in half of the different types of cancers, the mutation of this gene, p53 has been found. The cross talking of p53 and miR-34 in a mouse model study showed the genes jointly suppressing another gene called MET which can cause different cancers. It is the first mouse model study showing these findings, according to Alexander Nikitin, a professor at Biomedical Sciences department of Cornel University. The suppression of either gene alone, in prostate epithelial cells did not lead to reduction of cancer cells. The joint silencing of the two genes, miR-34 and p53 resulted in reduction of the prostate cancer cells which could be a promising target for cancer therapy in future.

Reference: http://www.sciencedaily.com/releases

PATHOGENIC *ESCHERICHIA COLI*: MOLECULAR AND CELLULAR MICROBIOLOGY

Publisher: Caister Academic Press

Editor: Stefano Morabito

EU Reference Laboratory for E. coli, Veterinary Public Health and Food Safety Department, Instituto Superiore di Sanità, Rome, Italy.

ISBN: 978-1-908230-37-9

BIOINFORMATICS AND DATA ANALYSIS IN MICROBIOLOGY

Publisher: Caister Academic Press

Editor: Ozlem Ta and Scediltan Bishop

Rhodes University Bioinformatics, Department of Biochemistry, Microbiology and Biotechnology, Rhodes University, South Africa.

ISBN: 978-1-908230-39-3

MYXOBACTERIA: GENOMICS, CELLULAR AND MOLECULAR BIOLOGY

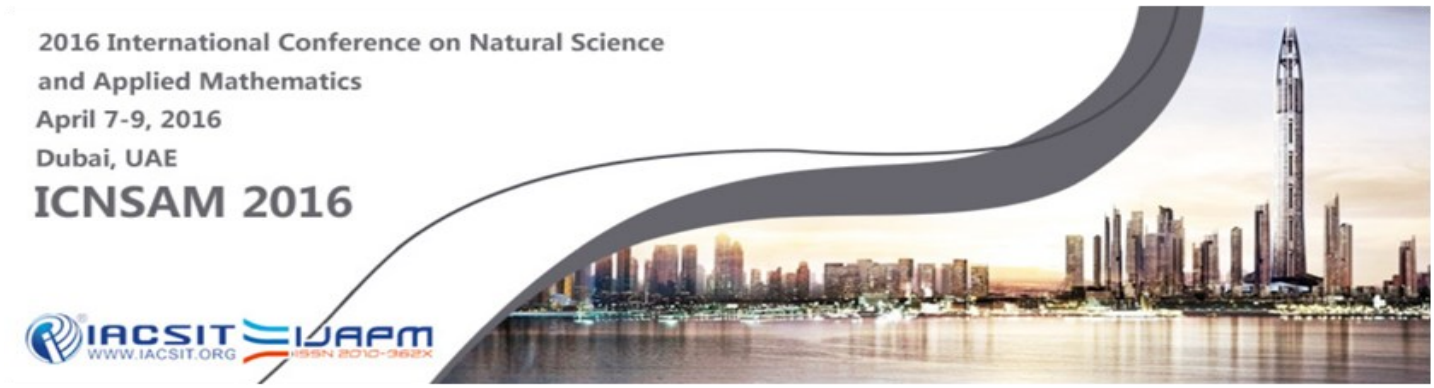
Publisher: Caister Academic Press

Editor: Zhaomin Yang and Penelope I. Higgs

Biological Sciences, Virginia Tech, Blacksburg, VA 24061-0910, USA; Dept. of Ecophysiology, Max Planck Institute for Terrestrial Microbiology, Marburg, Germany (respectively).

ISBN: 978-1-908230-34-8

Announcements



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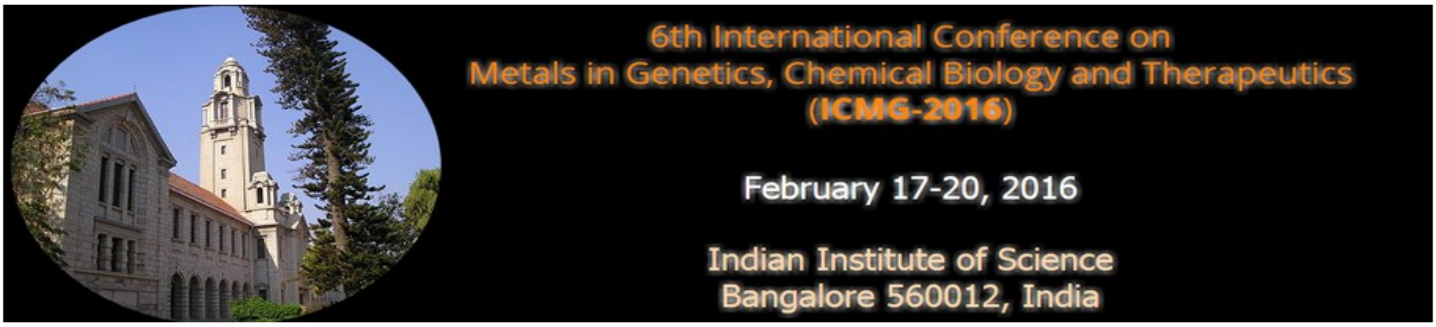
http://itcs2016.org/index.html



http://www.icmg2016.com/

Announcements

http://www.indc.cz/en/

http://autoimmunity.kenes.com/

College of Science and Engineering > Medical Devices Center

Design of Medical Devices Conference

April 11, 12-14, 2016

The Commons Hotel & McNamara Alumni Center
Minneapolis, MN

15 CELEBRATING 15 YEARS

http://www.dmd.umn.edu/

TITLE: DNA ligase

DNA ligase, a specific enzyme that binds DNA strands together by catalyzing phosphodiester bridges has a special role in sealing single-strand DNA breaks. Some forms of DNA ligase, such as DNA ligase IV, are able to repair breaks in both complementary strands. In addition, to repair DNA duplex, the enzyme is involved in DNA replication as well as in molecular biology experiments to construct recombinant DNA. The purified form of the DNA ligase is also used in molecular cloning to make DNA recombinant.

Reference: http://en.wikipedia.org/wiki/DNA_ligase

TITLE: Endothelial stem cells

Endothelial stem cells (ESCs) comprised of a group of stem cells observed in bone marrow. ESCs are multipotent and are able to change into many cell types. They have the specific attributes of a stem cell differentiation and self-renewal. Also, ESCs are parents of stem cells, which change to progenitor cells and intermediate stem cells. ESCs will subsequently create endothelial cells (ECs), which produce the thin-walled endothelium that covers the internal area of both lymphatic vessels and blood vessels. ESCs have the unparalleled ability to produce similar copies of themselves. EPCs are more differentiated than ESCs, and ECs are more differentiated than EPCs. The more differentiated a cell is, the more specialized it is.

Reference: http://en.wikipedia.org/wiki/Endothelial_stem_cell

TITLE: Coronavirus

Coronaviruses belong to enveloped, positive sense RNA viruses with a genome length of 26–32 kb. The virus particles, virions, appear in spherical (120–160 nm, in *Coronavirinae*) or bacilliform (170–200 by 75–88 nm, in *Bafinivirus*) or in a form of a combination of both, in *Torovirus* bearing crescents particles. The spherical particles are seen in forms of the solar corona using electron micrographs. The viral envelope contains two virus-specific proteins including, S and M, comprising large surface and triple membrane spanning protein respectively. Furthermore, phosphoprotein N is also recognized as another viral structural protein accountable for helical symmetry of RNA virus nucleocapsid.

Reference: http://en.wikipedia.org/wiki/Coronavirus