

International Hypoxia Symposia 2019

(Celebration of Nobel Prize for Physiology or Medicine 2019 to Hypoxia Biologists)

Theme: **Oxygen Cell Signalling: Mechanisms to therapeutics**

Organized by: Laboratory of Vascular Physiology and Medicine, Department of Physiology

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Date: November 23, 2019 **Time:** 10.00am -10:45am

Venue: Department of Medical Education, 2nd floor, hospital building, BLDE (Deemed to be University),Vijayapur, Karnataka, India

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Prof. Thuppil Venkatesh, PhD
President, International Society of Chronic Hypoxia
Will inaugurate the symposia

Chief Guest

Dr. M.S.Biradar, MD
Hon'ble Vice Chancellor, BLDE (Deemed to be University)

Dr.Aravind V.Patil, MS
Dean of Faculty of Medicine & Principal,
BLDE (Deemed to be University)
Will Preside over the function

Dr.Sumangala Patil, MD	**Dr.Jyoti Khodnapur,** MD,PhD	**Dr.Lata.Mullur,** MD,PhD
Organizing Chair	Organizing secretary	Convener

Programme Schedule

Inaugural Session: 10.00h -10:45h

Scientific Session:

10:45h: Theme Talk: Kusal K.Das, Shri B.M.Patil Medical College, Hospital & Research Centre.

11:00 -11:45h: Gustavo Zubieta-Calleja,MD, High Altitude Pulmonary and Pathology Institute, La Paz , Bolivia (Pulmonology).
Topic: Adaptation to high altitude: the paradox of tolerance to hypoxia; one small step but a giant leap to BioSpace Formin

11:45 -12:30h: Jan Marino (Nino) Ramirez,PhD,University of Washington, USA (Neurological Surgery)
Topic: Oxygen sensing in the central respiratory network and the implications for disordered breathing and cardiorespiratory control

12:30 – 13:00h Jyoti Khodnapur, MD;PhD, BLDE (Deemed to be University) (Physiology)
Topic: Oxygen sensing mechanism in ageing in relation to arterial stiffness

13:00 -14:30h: **Lunch**

14:30 – 15:00h Natalalia Zubieta-DeUrioste,MD; High Altitude Pulmonary and Pathology Institute, La Paz, Bolivia (Internal Medicine).
Topic: Polyerythrocythemia and cerebro-vascular accidents at high altitude

15:00-15:30h Shrilaxmi Bagali,MD;PhD; BLDE (Deemed to be University) (Physiology)
Topic: Low oxygen microenvironment in cardiovascular remodelling: Role of L/N type Ca^{+} channel blocker.

15:30 -16:15h **Panel Discussion:**

Moderator: Lata Mullur,MD;PhD BLDE (Deemed to be University) (Physiology)

Participants: All the speakers 16:15-16:45:

Valedictory Session:

Vote of thanks: Dr.Pallavi Kanthe,PhD

High Tea

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Invitation

BLDE (Deemed to be University)

Shri. B. M. Patil Medical College, Hospital & Research Centre, Vijayapura

International Hypoxia Symposia

23rd November 2019

Registration form

Name:

Designation:

Institute:

Email ID:

Mobile no:

Delegate Fee: Rs 300/-

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Details of Online Payment/ NEFT:

Name of Bank: Account Number:

Amount transferred date: UTR Number:

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1. The DD/Cheque should be paid in favour of		: The Registrar (Deemed to be University), Vijayapura
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	A/c Name	: The Registrar (Deemed to be University), Vijayapura
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International Hypoxia Symposia 2019

Celebration of Nobel Prize for Physiology or Medicine 2019 to Hypoxia Biologists

HYPOXIA LECTURE SERIES

Oxygen Cell Signaling: Mechanisms to Therapeutics

November 23, 2019

Laboratory of Vascular Physiology and Medicine, Department of Physiology, Shri B.M.Patil Medical College, Hospital and Research Centre, Vijayapur, Karnataka, India

International Hypoxia Symposia 2019

Celebration of Nobel Prize for Physiology or Medicine 2019 to Hypoxia Biologists

Date: November 23, 2019
Time: 9 AM to 5 PM
Venue: Department of Medical Education, BLDE (Deemed to be University),Vijayapur, Karnataka, India

Inaugural Session: 10.00h -10:45h

Welcome Address: **Dr.Sumangala Patil**,MD; Organizing Chair

Lighting the lamp & Inauguration- **Prof.Thuppil Venkatesh**,PhD; President, International Society of Chronic Hypoxia

Chief Guest: **Prof.Dr.M.S.Biradar,**MD, Vice Chancellor, BLDE (Deemed to be University)

Guest of Honor: **Dr.Bhaskar Saha**, FNA, FNASc; FASc; Scientist G, (S.S.Bhatnagar Awardee), National Centre for Cell Science (DBT), Pune, Maharashtra

President: **Dr.Aravind V.Patil**, MS; Dean of Faculty of Medicine & Principal, BLDE (Deemed to be University)

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Scientific Session:

Theme Talk: 10:45-11:00h

Kusal K.Das,PhD;FRSB, Shri B.M.Patil Medical College, Hospital & Research Centre, BLDE (Deemed to be University), Vijayapur,Karnataka
Title: *Oxygen Cell Signaling: Mechanisms to therapeutics*

Lecture 1: 11:00-11:45h

Gustavo Zubieta-Calleja,MD, Department of Pulmonology, High Altitude Pulmonary and Pathology Institute, La Paz , Bolivia
Title: *Adaptation to high altitude: the paradox of tolerance to hypoxia;one small step but a giant leap to BioSpaceForming*

Lecture 2: 11:45-12:30h

Jan-Marino (Nino) Ramirez,PhD, Department of Neurolopgical Surgery, University of Washington, USA
Title: *Oxygen sensing in the central respiratory network: Relevance for disordered breathing and cardiorespiratory control*

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Lecture 3: 12:30 –13:00h

Jyoti Khodnapur, MD;PhD, Laboratory of Vascular Physiology and Medicine, Department of Physiology, BLDE (Deemed to be University) (Physiology), Vijayapur
Title: *Oxygen sensing mechanism in ageing with relation to arterial stiffness*

Lecture 4: 14:30 –15:00h

Natalalia Zubieta-DeUrioste,MD; Department of Internal Medicine, High Altitude Pulmonary and Pathology Institute, La Paz , Bolivia
Topic*: Polyerythrocythemia and cerebro-vascular accidents at high altitude*

Lecture 5: 15:00-15:30h

Shrilaxmi Bagali,MD;PhD; Laboratory of Vascular Physiology and Medicine, Department of Physiology, BLDE (Deemed to be University) (Physiology), Vijayapur
Title*: Low oxygen microenvironment in cardiovascular remodelling: Role of L/N type* Ca^{+} *channel blocker*.

Panel Discussion: 15:30 -16:15h

Moderator:
Lata Mullur,MD;PhD; Laboratory of Vascular Physiology and Medicine, Department of Physiology, BLDE (Deemed to be University) (Physiology), Vijayapur.

Vote of thanks: **Dr.Pallavi Kanthe**,PhD

Congratulation to Nobel Laureates of Physiology or Medicine 2019

Gregg L.Semenza Sir Peter J. Ratcliffe William G. Kaelin Jr

William G. Kaelin Jr., Sir Peter J. Ratcliffe and Gregg L. Semenza discovered how cells can sense and adapt to changing oxygen availability. They identified molecular machinery that regulates the activity of genes in response to varying levels of oxygen. The seminal discoveries by this year's Nobel Laureates revealed the mechanism for one of life's most essential adaptive processes. They established the basis for our understanding of how oxygen levels affect cellular metabolism and physiological function by oxygen sensing mechanisms. Oxygen sensing allows cells to adapt their metabolism to low oxygen levels: for example, in our muscles during intense exercise. Other examples of adaptive processes controlled by oxygen sensing include the generation of new blood vessels and the production of red blood cells. Our immune system and many other physiological functions are also fine-tuned by the O_2-sensing machinery. Their discoveries have also paved the way for promising new strategies to fight anemia, cancer and many other diseases.

(Ref.: https://www.nobelprize.org/prizes/medicine/2019/press-release/)

Theme Talk:

Oxygen Cell Signaling: Mechanisms to therapeutics

Kusal K.Das, PhD, FRSB*
Laboratory of Vascular Physiology and Medicine, Department of Physiology, Shri B M Patil Medical College, Hospital and Research Centre, BLDE (Deemed to be University), Vijayapura, Karnataka, India

*Professor

Hypoxia belongs to the most grave factors that can directly impair the function of metabolic pathways in the animal cell. Normally PO2 values of -100 Torr in the alveoli of the lungs to less than 10 Torr in tissues such as the medulla of the kidney and the retina in healthy humans are considered as the range of physiological oxygen levels within the tissues of the body. Hypoxia, which occurs when oxygen levels in the microenvironment of a cell, tissue, or organism are reduced relative to the normal physiological state, is associated with a range of physiological and pathophysiological processes. In vascular diseases such as atherosclerosis and stroke, vascular occlusion leads to acute or chronic tissue ischemia with resultant hypoxia. In chronic inflammatory disease, the greatly increased metabolism of inflamed tissue due to immune cell infiltration matched with vascular dysfunction leads to tissue hypoxia. In cancer, the growth of a tumor away from the local blood supply eventually leads to tumor hypoxia. In all of these cases, the induction of a genetic response to hypoxia leads to the expression of genes that are essentially adaptive (or maladaptive in the case of cancer). Seminal discoveries in the last 20 years have greatly enhanced our understanding of the molecular mechanisms underpinning this critical response. Hypoxia results from conditions such as ischemia, hemorrhage, stroke, premature birth, and other cardiovascular difficulties, among which hemorrhagic shock is the leading cause of death and complications in combat casualties and civilian settings.

Hypoxia has been shown to lead to increases in intracellular free calcium concentration (Ca^{2+}), 5-lipoxygenase, lipid peroxidation, cycloxygenase (COX), constitutive nitric oxide synthase (cNOS), leukotriene B4 (LTB4), prostaglandin E2 (PGE2), interlukins, tumor necrosis factor-α (TNF- α), caspases, complement activation, kruppel-like factor 6 (KLF6), inducible nitric oxide synthase (iNOS), heat shock protein 70 kDa (HSP-70), and hypoxia-inducible factor-1α (HIF-1α). The sequence of their occurrence provides the useful information for studying the mechanisms underlying the hypoxia-induced injury as well as therapeutic targets to prevent or ameliorate the injury.

Low oxygen microenvironment induces generation of ROS (reactive oxygen species) ,increase expression of p53, NF-kβ, AP-1, MAPK and HIF-1α. The increase expression of all these transcription factors leads to either cellular adaptation or cell death. The mechanisms by which mammalian cells adapt to acute and

chronic alteration of oxygen tension is extremely important to understand the exact homeostasis regulation to counteract hypoxia-induced cell damage as therapeutic strategy.

Reference: Bagali S, Hadimani GA, Biradar MS, Das KK**.** Introductory Chapter-Primary concept of hypoxia and anoxia In: Hypoxia and anoxia (Das KK, Biradar MS eds), 2018; ISBN: 978-953-51-6485-2 , InTech Open Science, London

Lecture 1

Adaptation to high altitude: the paradox of tolerance to hypoxia; one small step but a giant leap to BioSpaceForming

Gustavo Zubieta-Calleja, MD*
Department of Pulmonary Medicine
High Altitude Pulmonary and Pathology Institute (IPPA), La Paz, Bolivia

*Professor and Director

Adaptation is the fundamental tool of survival. Geneticists refer to adaptation as a genetic change carried out over many generations. And they use acclimatization to refer to physiologic changes in order to reach a stable function in a new environment. However when it refers to mountain climbing or arriving to high altitude from sea level and vice-versa, acclimatization is the wrong terminology, as it pertains to a climate change (i.e. temperature, humidity, weather). Hypoxia cannot be included because it is not a weather condition. This is why we prefer to use adaptation to high altitude. Furthermore, more and more gene expressions are evident on exposure to hypoxia. The high altitude equation is fundamental to understand the physiologic changes: cardiac, respiratory, biochemical, gene expression and hematologic changes. The tolerance to hypoxia equation shows that paradoxically, the higher humans (and animals) go, the more they are able to survive hypoxia. These concepts along with almost 50 years of experience at high altitude in the city of La Paz, Bolivia at 3500m of altitude, have provided us with a unique opportunity to understand life under chronic hypoxia. We have learned of its benefits, and it has even allowed us to formulate the concept that chronic hypoxia is an advantage for the survival of the species. It provides, greater longevity, and at the same time hearts and brains more tolerable to hypoxia. But it has, likewise, allowed us to propose space travel under chronic hypoxia, which grants several advantages in such hostile microgravity environments. Furthermore, under such circumstances, biology is able to help surpass the physics pressure laws differences in space. This is a remarkable and unique condition in the history of life on planet earth

and beyond. Consequently, we have invented the new terminology BioSpaceForming (adaptation to life in space), where we affirm that humans (and all biological beings) need to adapt to space and change their biology accordingly. Chronic hypoxia becomes one of the first and most useful strategies of biospaceforming in Space.

Lecture 2

Oxygen sensing in the central respiratory network: Relevance for disordered breathing and cardiorespiratory control

Jan-Marino (Nino) Ramirez, PhD*
Center for Integrative Brain Research, Seattle Children's Research Institute; Department of Neurological Surgery, University of Washington School of Medicine, Seattle, WA 98101

*Professor and Director

The continuous regulation of O_2 and CO_2 is critical for survival in mammals. Oxygen homeostasis is particularly important for the brain. The failure to maintain a delicate O_2 /CO_2 balance has been associated

with numerous disorders including epilepsy, sleep apnea, Rett syndrome and sudden infant death syndrome. However, it is important to realize that O_2 /CO_2 homeostasis poses major regulatory challenges, even in the healthy brain. Neuronal activities within the Central Nervous system change in a differentiated, spatially and temporally complex manner. As a consequence, these changes in neuronal activity result in equally complex changes in O_2 demand. This raises important questions that we are just beginning to understand: is oxygen sensing an emergent property, locally generated within all active neuronal networks, and/or the property of specialized O_2 -sensitive CNS regions? There is accumulating evidence that suggests that controlling the brain's redox state involves properties that are intrinsic to many central networks, but that specialized regions and specific neuroglial interactions in the brainstem orchestrate the integrated control of respiratory and cardiovascular functions. Although the levels of O_2 in arterial blood and the CNS are very different, neuro-glial interactions and purinergic signaling are critical for both peripheral and CNS chemosensation. Indeed, the specificity of neuroglial interactions seems to determine the differential responses to O_2 , CO_2 and the changes in pH. In this talk, I will discuss

the important role of medullary networks in regulating not only the respiratory network, but also controlling the interactions with the cardiovagal control systems. This cardiorespiratory control is very sensitive to oxygen and reactive oxygen species (ROS).

Lecture 3

Oxygen Sensing Mechanism in Ageing with Relation to Arterial Stiffness

Jyoti Khodnapur, MD;PhD*

Laboratory of Vascular Physiology and Medicine, Department of Physiology, Shri B M Patil Medical College, Hospital and Research Centre, BLDE (Deemed to be University), Vijayapura, Karnataka, India

*Associate Professor and Presenting author

Objective: The blood pressure, pulse wave velocity (PWV) and arterial stiffness index (ASI) are important markers for age related alterations of vascular function. Further a link between vascular stiffness, vascular endothelial growth factor (VEGF) and erythropoietin (Epo) are least explored in relation to sex and age. *Aim:* The present study was aimed to assess the vascular health in relation to ageing with special reference to vascular endothelial growth factor (VEGF) and erythropoietin (EPO) in both male and female participants of Vijayapur city, Karnataka, India. *Methods:* 204 healthy male (n= 102) and female (n=102) participants (20 to 95 years) were randomly selected among general population of Vijayapur city, Karnataka. Participants were divided into group I (20-29years), II (30-39 years), III (40-49 years), IV (50-59 years), V (60-69 years) and VI (>70 years). Physical anthropometry, physiological parameters, hematology, serum VEGF and serum Epo were assessed. Vascular stiffness indicators like PWV (b-a PWV and c-f PWV) and ASI (brachial ASI and ankle ASI) were also evaluated. Statistical analysis was performed by using one way ANOVA and post hoc test by using SPSS software. *Results:* Increase of MAP, PWV, ASI and VEGF along with concomitant decrease of serum Epo was observed in group V and VI of both males and females. While comparing between male and female all the vascular parameters showed significant differences in group I to IV only. There was significant difference ($p<0.001$) of VEGF between male and female subjects. *Conclusion:* The present study clearly showed impact of ageing on PWV, ASI, MAP, VEGF and Epo in higher age groups of

males and females. Epo is sensitive in older age groups in relation to vascular functions. Epo might be playing a crucial homeostatic role in ageing. Females have an augmented protection against age related alteration of vascular pathophysiology due to greater VEGF concentration as compared to their male counterparts. Endothelial function is not the final protection for arterial function as role of VEGF and its expression in arterial smooth muscles is very important for vascular stability. Understanding of these mechanisms may support greater pharmaco-physiological understanding of arterial stiffness which may possibly improve cardiovascular health of an individual irrespective to their sex.

Lecture 4

Polyerythrocythemia and cerebro-vascular accidents at high altitude

Natalalia Zubieta-DeUrioste, MD*

Department of Internal Medicine, High Altitude Pulmonary and Pathology Institute (IPPA), La Paz, Bolivia

*Consultant Specialist

When sea level humans go to high altitude, they have to increase their red blood cells in order to compensate for the environmental hypobaric hypoxia. What are the normal levels of hemoglobin, hematocrit, and red blood cells for each altitude? What are the specific variations in each civilization? We have over 49 years of work at the 'High Altitude Pulmonary and Pathology Institute;, on this subject. We established the normal values for the city of La Paz, Bolivia at 3500m, in 1979 but it was poorly understood and other centers applied it without considering the variations of altitude. One can take different parameters to evaluate where the top cutoff limit for polyerythrocythemia is and likewise where anemia is present at high altitude. The limits where one can speak of polyerythrocythemia, depend on where the symptomatology (if present) appears. But it can also be the standard deviation. The Gauss curve of distribution can also be visually analyzed to set the limits. The complexity of different altitudes, different nutrition, different genetic build-up, altitude changes, is evident in setting the normal values. This is why there is so much confusion. The importance of this is that many people receive treatments of bleeding unnecessarily, due to this lack of standard values. These parameters will now be applied to a multi-

center study being performed in South America to evaluate if cerebro-vascular accidents (CVA) are related to polyerythrocythemia, or chronic mountain sickness (as it is known). In a preliminary study in a reference hospital for CVA's in La Paz - Bolivia (3600 m), 10% of the CVA's had polyerythrocythemia. However, of that 10% (n=10), all of them had a concurrent diagnosis such as Arterial Hypertension (9/10), diabetes (2/10) and dehydration (1/10) which are all well known causes of CVA at sea level and possibly rule out polyerythrocythemia, as the fundamental ethiopathogenesis. Therefore this suggests that AVC should not be seen as an expectable complication of polyerythrocythemia at high altitude. Likewise, this is an important start to study the complications seen at high altittude, by counting the cases seen, instead of presuming that the viscosity of the blood is directly related to increased morbility.

Lecture 5

Low oxygen microenvironment in cardiovascular remodeling: Role of L/N type Ca^{2+} channel blocker

Shrilaxmi Bagali, MD;PhD*

Laboratory of Vascular Physiology and Medicine, Department of Physiology, Shri B M Patil Medical College, Hospital and Research Centre, BLDE (Deemed to be University), Vijayapura, Karnataka, India.

*Associate Professor

Patients exposed to chronic sustained hypoxia frequently develop cardiovascular disease risk factors to ultimately succumb to adverse cardiovascular events. In view of this, the current study was undertaken to assess the role of L/N type calcium channel blocker cilnidipine in face of chronic sustained hypoxia-induced changes in cardiovascular pathophysiology. Adult male Wistar rats were randomly assigned to one of the four groups - group 1 Control, Normoxia (21% O_2); group 2, Chronic Hypoxia (CH) (10% O_2, 90% N); group 3, Normoxia (21% O_2) + Cilnidipine (Cil); group 4, Chronic Hypoxia (10% O_2, 90% N) + Cilnidipine (CH+Cil). Cardiovascular hemodynamics, heart rate variability (HRV), biomarkers of oxidative stress and hypoxia signaling molecules (serum VEGF, serum NOS3, serum NO) were assessed. Cardiovascular remodelling

was assessed by histopathological examination of ventricular tissues, coronary artery (intramyocardial), elastic and muscular arteries. Normalized wall index (NWI) of coronary artery was calculated. The results demonstrated altered cardiovascular hemodynamics, disturbances in cardiovascular autonomic balance, ventricular tissue oxidative stress, increased expression of VEGF and NOS3 proteins and decreased bioavailability of NO on exposure to chronic sustained hypoxia. The histopathological study pointed towards cardiovascular remodelling. Treatment with cilnidipine ameliorated the cardiovascular remodeling induced by chronic hypoxia exposure which may be due to its inhibitory action on L/N type of calcium channels indicating the possible therapeutic role of cilnidipine against chronic hypoxia-induced cardiovascular pathophysiology.

Keywords: chronic sustained hypoxia, cilnidipine, heart rate variability, Normalised Wall Index, cardiovascular remodelling

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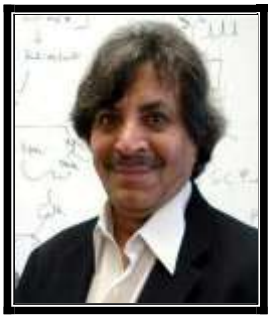
End Note: Remembering ***Nanduri R.Prabhakar***, University of Chicago on Hypoxia biology



Nanduri R.Prabhakar,D.Sc.; Harold H. Hines Professor of Medicine and Inaugural Director of the Institute for Integrative Physiology and Center for Systems Biology of O_2 sensing at the University of Chicago, IL, USA. He is a leading authority on O-sensing mechanisms and physiological consequences of hypoxia.

His immense contribution on carotid body chemoreflex in the progression of autonomic morbidities associated with cardiorespiratory diseases, such as sleep-disordered breathing with apnoea (OSA), congestive heart failure and essential hypertension. His one of the hypothesis is that in addition to OSA, systemic hypertension may involve disturbance of the balance between HIF-1α and HIF-2α, leading to oxidative stress in the carotid body (CB) and adrenal medulla (AM) leading to sympatho-adrenal activation

DEPARTMENT OF PHYSIOLOGY

International Hypoxia Symposia 2019 REPORT

(Celebration of Nobel Prize for Physiology or Medicine 2019 to Hypoxia Biologists)

Theme: *Oxygen Cell Signalling: Mechanisms to therapeutics*

We from Department Of Physiology, Shri B. M. Patil Medical College, BLDE (Deemed To Be) University, Vijayapura submiting the report of Conducted International Hypoxia Symposia (Celebration of Nobel Prize for Physiology or Medicine 2019 to Hypoxia Biologists)Under The Laboratory of Vascular Physiology and Medicine,Dept of Physiology On 23rd Nov 2019. The Theme Of The International Hypoxia Symposia Was "Oxygen cell signalling:Mechanisms to Therapeutics".

International Hypoxia Symposia conducted between 10am to 5pm. At 10:00am Prof.Thuppil Venkatesh,President International Society of Chronic Hypoxia Inaugaerated Our Function With Watering the Plant In The Presence Of Guest Of Honour Dr. Bhaskar Saha, Scientist –G National centre for cell science Pune and Chief Guest Vice Chancellaer Dr. M. S. Biradar, Principal Dr. Aravind Patil presided over the function.

Details Of The Participants:

Total 109 delegates attended the international hypoxia symposia from various parts of the states and the neighboring states made their presence with enthusiasm.

The Scientific Session Of The Day Started At 10:45am With themeTalk on Oxygen cell signalling:Mechanisms to Therapeutics by Prof. Kusal K.Das, Shri B.M.Patil Medical College, Hospital & Research Centre. We Preceeded With 3 Talks by Dr. Gustavo Zubieta-Calleja Dr. Jan Marino (Nino) Ramirez and Dr. Jyoti Khodnapur And Session Was Chaired By Dr. Vijayakuma Kallyanappagol and Dr. Bhaskar Saha.

We Had Break For Lunch At 2 Pm And Proceeded With afternoon Session. In the afternoon session We Had 2 Talks by Dr. Natalalia Zubieta-DeUrioste and Dr. Shrilaxmi Bagali and chaired By Dr. Salim Dhundasi.

We Had 1 Hour Panel discussion moderated by Dr. Lata Mullur with all speakers and had a wonderful discussion with delegates.

Valedicted with Vote of Thanks by Dr.Pallavi Kanthe. We Had High tea And Goodbye.