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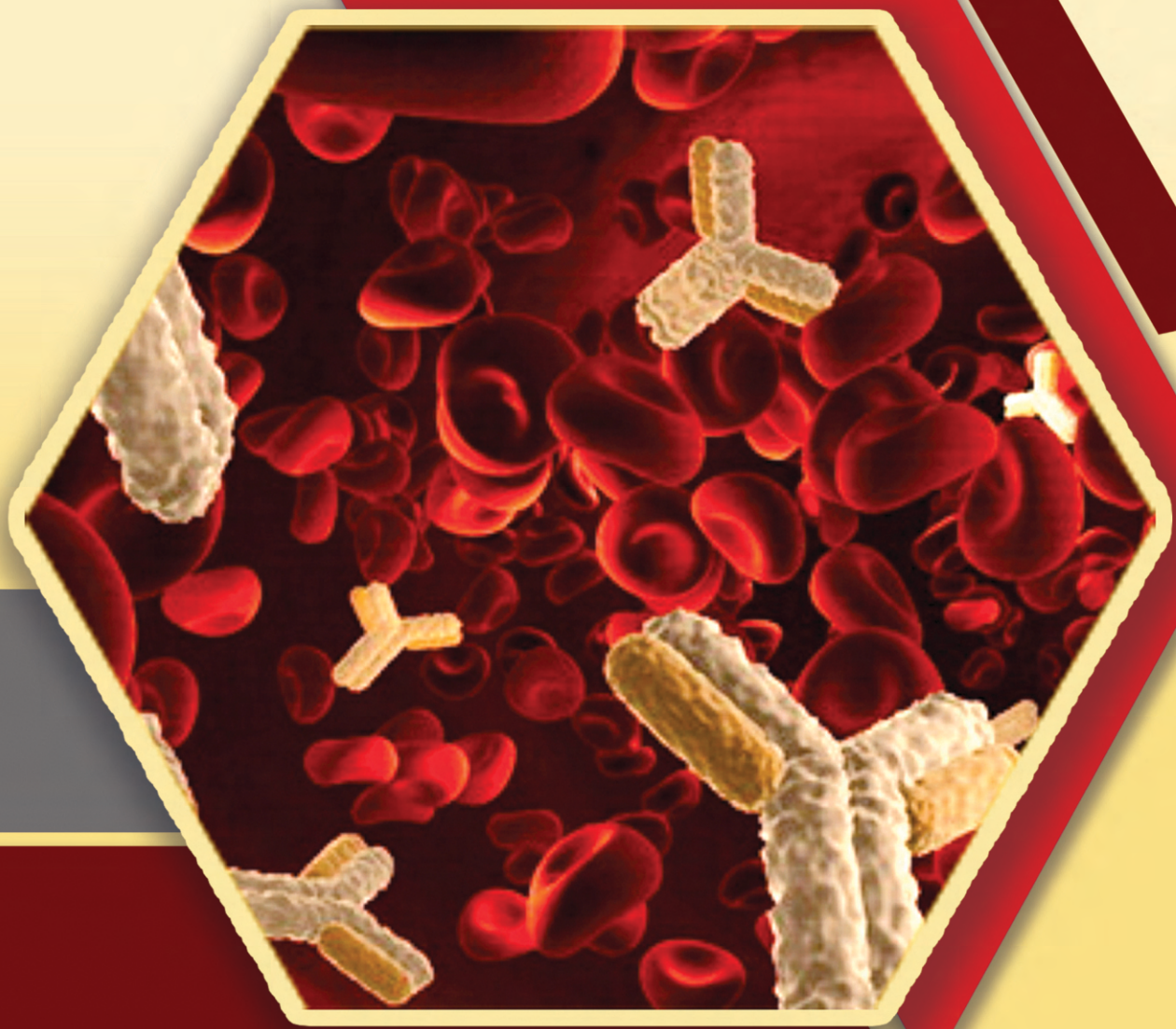

ICMR - NATIONAL INSTITUTE OF IMMUNOHAEMATOLOGY

NIIH DIAMOND JUBILEE CELEBRATIONS 15th - 17th Feb, 2017

Book release during inaugural ceremony of the Diamond jubilee conference

Inaugural address by DG, ICMR & Secretary DHR, Dr. Soumya Swaminathan

Adipokines: the new players of inflammatory response in Systemic Lupus Erythematosus

Durga A Chougule & Vandana D Pradhan

Since the discovery of leptin, adipose tissue (earlier referred as the inert organ), is being extensively studied for its autocrine, endocrine and exocrine functions. The cellular components of adipose tissue are involved in secretion of chemical mediators called adipocytokines or adipokines. As a hormone, adipokines play a regulatory role in appetite maintenance, glucose metabolism and lipid metabolism; whereas, as a cytokine they are involved in inflammatory response and regulation of immunity. The pathophysiology of pro-inflammatory and anti-inflammatory adipokines is being explored in autoimmunity, which is a consequence of inflammatory response and breach of self-tolerance. Systemic Lupus Erythematosus (SLE) is an inflammatory rheumatic disease involving imbalance of inflammatory mediators and production of autoantibodies leading to systemic tissue and organ damage. Studies among patients with systemic rheumatic diseases have shown varied results in circulating adipokine levels and disease severity. The interplay of adipokines still remain questionable but once deciphered might prove as important therapeutic targets in treatment of systemic rheumatic disease.

Introduction

SLE is a prototypic systemic autoimmune disease characterized by autoantibody production and inflammatory response in multiple organs. Though environmental factors and genetic predispositions are said to be the initial triggers for induction of SLE; not much is known about the etiology and pathogenesis of SLE. Lately, adipocyte-associated cytokines have been extensively studied for their independent and significant association with the disease severity and risk of co- morbidities in SLE [1, 2, 3]. The adipose tissues in mammals are of two types: white adipose tissue (WAT) and brown adipose tissue (BAT). Adipose tissue consists of adipocytes and stromal vascular fraction (SVF) cell population which includes pre-adipocytes, fibroblasts, vascular endothelial cells and a variety of immune cells like adipose tissue macrophage [4]. These cell populations are involved in secretion of chemical mediators called adipocytokines/adipokines which have a pro-inflammatory and anti-inflammatory effect [4, 5]. The interplay between pro-inflammatory and anti-inflammatory adipokines plays a regulatory role in lipid metabolism, inflammation,

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immune-stress response and cardiovascular function [6]. Alteration in adipokine levels have been observed in a variety of diseases like metabolic syndrome, obesity, diabetes, cardiovascular diseases etc. Increase in adiposity is marked with an elevation in production of pro-inflammatory adipokines and infiltration of immune cells giving rise to low grade systemic inflammatory response; a phenomenon closely related to metabolic and obesity related diseases [7]. Inflammation is an important phenomenon in SLE and hence adipokines have been considered as a potent candidate for SLE disease pathogenesis [8].

Regulation of Immune Response in Adipose Tissue

Apart from the fat storing adipocytes, adipose tissue also consists of resident immune cells like monocytes/macrophages, T & B lymphocytes, dendritic cells, mast cells, neutrophils and eosinophils. Adipose tissue is located in close proximity to lymph nodes; due to which the interaction between the adipocytes and migrating immune cells is evident [4].

An increase in the mass of adipocytes initiates a low grade inflammatory response in the microenvironment of adipose tissue [6,8] (figure 1). In an obese condition, changes in immune cells are marked with a decrease in eosinophils and T regulatory (T-reg) cells and an increase in $CD4^+$ and $CD8^+$ T-cells. An increase in $CD4^+$ T-cells augments secretion of T_H1 cytokines. Lumeng et al [9] have reported a shift in differentiation of resident macrophages from the anti-inflammatory cytokines producing M2 (alternatively activated) macrophages to pro-inflammatory cytokines producing M1 (classically activated) macrophages in obese patients. Disruption in the pro-inflammatory and anti-inflammatory adipocyte-associated cytokines (adipokines) balance results in metabolic dysfunction which eventually leads to apoptotic and necrotic cell death (Table 1).

Adipokines in Immunity

Adiponectin

Adiponectin also known as Acrp30, GBP28, adipoQ or apM1 is primarily secreted from adipocytes. It exists in three forms: light molecular weight, medium molecular weight and high molecular weight. Adiponectin reduces T cell responsiveness and B cell lymphopoiesis [10, 11]. Studies have shown anti-inflammatory effect of adiponectin by inhibiting production of pro-inflammatory cytokines. Adiponectin also exerts an anti-athersclerotic effect by inhibiting the expression of pro-inflammatory adhesion molecules and suppressing the endothelial nuclear factor kappa B (NFκB) signalling [12].

Leptin

Leptin, a pro-inflammatory adipokine, modulates immune system at various stages of development, proliferation, anti-apoptosis, maturation and activation [3]. Presence of a primary amino acid sequence similar to the long helical cytokines like IL-2, and sequence homology of leptin receptor to class I receptor includes this hormone as a member as in cytokine family. Leptin exerts a pro-inflammatory effect via JAK-STAT, PI3K and MAPK signalling pathway, thus promoting T_H1 responses and production of pro-inflammatory cytokines [13]. Presence of leptin receptor mutation

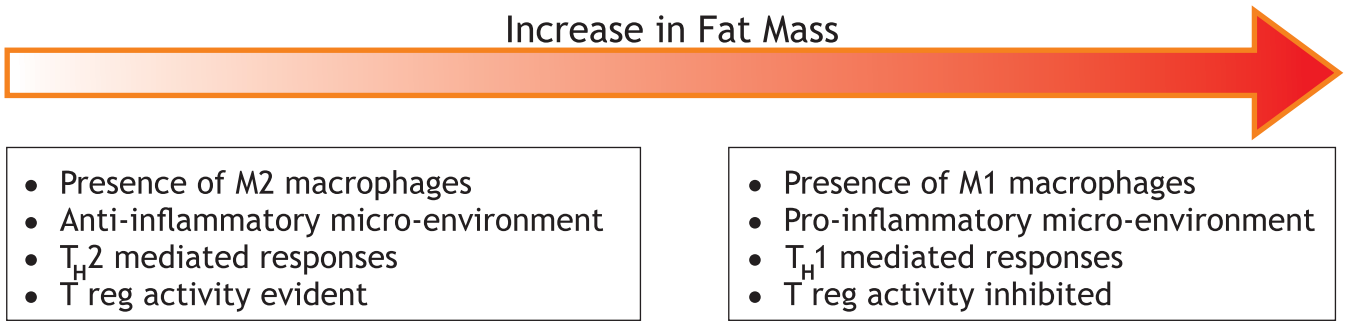

Figure 1 Recruitment of immune cells in adipose tissue due to an increase in mass size of adipocytes

and low leptin levels showed low immune response and high susceptibility to infections in mice. Leptin stimulates the expansion and differentiation of naive $CD4^+$ into pro-inflammatory T_H 1, T_H 2 and T_H17 subsets or anti-inflammatory T-reg cells. Low levels of leptin impair maturation of dendritic cells and favours stimulation of T-reg cells [14].

Visfatin

Visfatin, also known as pre-B-cell colony-enhancing factor (PBEF), is an insulin-mimetic molecule. Visfatin is involved in upregulation of pro-inflammatory cytokines TNFα, IL-1β and IL-6. Upregulation of visfatin in activated neutrophils of septic patients inhibits their apoptotic cell death by caspase-3 and caspase-8 mechanisms. Visfatin acts as a potent chemoattractant for monocytes and lymphocytes and induces T cell activation by promoting expression of co-stimulatory molecules on monocytes [10, 11, 15].

Resistin

Resistin belongs to the family of resistin-like molecules that are involved in regulation of inflammatory response. This adipokine shows pro-inflammatory activity by upregulating pro-inflammatory cytokines via the NFκB signalling pathway. Though the role of resistin in regulation of immunity is not much explored, studies show an upregulation in the endothelial adhesion molecules which is an important phenomenon in onset of cardiovascular disease [10, 11, 15].

Chemerin

Chemerin is a pro-inflammatory adipokine, expressed as prochemerin (143-amino acids) which is activated by serine protease C-terminal cleavage. This active chemerine (137-amino acids) promotes chemotaxis of macrophages and plasmocytoid dendritic cells by binding on GPCR ChemR23/CMKLR1 receptors present on them [16]. It also inhibits production of anti-inflammatory cytokines from anti-inflammatory macrophages [17].

Role of Adipokines in SLE

The involvement of adipokines in autoimmunity has been of major interest in last few years. Though adipokines have been extensively studied mainly in rheumatoid arthritis (RA) not much information is available in SLE. (Table 2) The serological data available in different ethnic groups of SLE patients provide conflicting results. Elevated serum

Table1: Effect of adipokines on innate and adaptive immunity

Adipokines	Effect on immunity	
	Adaptive	Innate
Adiponectin	• Inhibits macrophage phagocytic activity • ↓ Endothelial adhesion molecules • Suppress NFκB pathway • ↓ Pro-inflammatory cytokines • ↑ Anti-inflammatory cytokines	• ↓ T-cell response • ↓ B-cell lymphopoiesis • T-reg proliferation
Leptin	• Activation of monocytes & macrophages ↑ cell proliferation ↑ phagocytosis ↑ TNFα, ↑ IL-6, ↑ IL-8, ↑ IL-12 • Mediates chemokinesis • Activation of neutrophils • Inhibits apoptosis • Mediates or inhibit chemokinesis depending on the presence of chemoattractants ↑ IL-1β, ↑ IL-6, ↑ IL-8, ↑ MCP-1, Effect on NK cells ↑ IL-2 & perforin, ↑ IL-12 & ↓ IL-15	• T_H 1 cell activation • T_H 2 cell inhibition • Naive T-cell proliferation • ↓ T-reg proliferation • Pro-inflammatory cytokine induction
Visfatin	• Acts as a chemoattractant for monocytes & lymphocytes • ↓ Neutrophil apoptosis • ↑ Pro-inflammatory cytokines • ↓ Anti-inflammatory cytokines	• T-cell activation • Growth factor for B lymphocyte precursors
Resistin	• ↑ Endothelial adhesion molecules • Activates NFκB pathway • ↑ Pro-inflammatory cytokines	• Expansion of T-reg cells
Chemerin	• Activates plasmacytoid dendritic cells • Inhibits anti-inflammatory macrophages	• N/A

Table 2: Role of adipokines in pathogenesis of SLE

Adipokines	Potential role in SLE	Pro-or Anti-Inflammatory
Adiponectin	• Inhibits macrophage to foam cell transformation • Suppresses IL-2 induced NK cytotoxicity • Decrease T cell recruitment • Expresses anti-inflammatory cytokines IL-10, IL-1ra	Anti-Inflammatory
Leptin	• Proliferates & activates macrophages • Promotes T_H1 response • Upregulates IL-1, IL-6 & TNF-α	Pro-Inflammatory
Resistin	• Upregulation of TNF-α, IL-1β & IL-6 • Upregulation of adhesion molecules	Pro-Inflammatory
Chemerin	• Skin barrier defence • Chemotactic function for dendritic cells • Upregulation of TNF-α, IL-1β & IL-6	Pro-Inflammatory

adiponectin levels have been associated with premature atherosclerosis and renal involvement in SLE patients [19]. Meta-analysis study by Li et al [20] in SLE patients revealed no significant difference in leptin as compared to healthy controls. Evaluation of serum visfatin levels in Egyptian population showed an association with disease activity especially in lupus nephritis (LN) patients [21]. A significant association was observed between serum resistin levels and inflammatory markers like ESR, low complement levels, renal disease and glucocorticosteroid treatment [22]. Not much can be commented on the pathophysiological significance of adipokines in SLE and further studies are required to know the mechanism behind these associations.

Indian Scenario

The adipokine levels and their gene polymorphism have been studied in metabolic syndrome, diabetes, obesity and obesity-related co-morbidities, not many studies have been reported in rheumatic diseases. Afroze D et al had reported an increased susceptibility of SLE in Kashmiri population with elevated leptin levels and LEPRQ223R polymorphism. [23] This was the first study reported in Indian patients. Hitherto there have been no published reports on adipokines in Indian SLE patients from other parts of the country.

Experience at NIIH

In preliminary study at NIIH Mumbai, a total of 80 subjects consisting of naively treated SLE patients (n=45) fulfilling the American College of Rheumatology (ACR) criteria and age-sex matched healthy controls (n=35) were enrolled. The sera adipokines levels (Adiponectin, Leptin, Progranulin, Omentin and Adipsin) were evaluated by ELISA. Sera were screened for Anti-nuclear antibodies (ANA), anti-dsDNA and anti-neutrophil cytoplasmic antibodies (ANCA) by indirect immunoflorescence (IFA) technique. ANA positive samples were further processed for qualitative detection of ANA profile using Immunoassay blot.

We saw statistically significant lower serum leptin ($p<0.05$) levels in SLE patients as compared to healthy controls. These results are different from the higher leptin levels reported in Kashmiri SLE patients by Afroze et al. [23] Similarly, statistically significant reduced omentin levels ($p<0.05$) were seen in SLE patients as compared to healthy controls. The serum progranulin and adipsin levels were significantly higher in SLE patients when compared to healthy controls ($p<0.05$). The serum adiponectin levels of SLE patients and healthy controls were comparable.

Our study also showed that the adiponectin levels were significantly increased in SLE patients with renal involvement (LN) when compared to SLE patients without renal involvement ($p<0.05$). Leptin, progranulin, omentin and adipsin levels did not show a statistically significant difference when SLE patients with renal and without renal involvement were compared. Further studies in larger cohort are required to understand the pathophysiology of adipokines in SLE and genetic susceptibility to SLE considering the adipokine profile.

Conclusion

Once an inert tissue, but now considered as a major endocrine organ of human body, adipose tissue has been extensively studied for its adipokines and their regulatory functions in immune responses and inflammatory responses. Considering the vital role that adipokines play in inflammatory responses, they are now area of interest in autoimmunity. Until now more than hundred adipokines have been discovered and not much is known about their pathophysiological role in SLE. Though in-vitro and in-vivo models showed an impending role of adipokines in SLE pathogenesis, studies in human have shown varied findings. More research is required to establish associations between adipokines and pathogenesis of SLE. The decoding of adipokines networking in SLE pathogenesis shall avail new therapeutic targets and prognostic markers. Furthermore, adipokines gene polymorphism studies shall evaluate an association between adipokine gene variants and susceptibility of an individual to SLE.

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सारांश

लुपस की बिमारी में प्रदाह (Inflammation) के कारणों में ॲडिपोकाईन का योगदान

मनिषा पटवर्धन

सन 1995 में लोप्टिन की खोज हूई। अबतक वसा उतक (adipose tissues) को निष्क्रिय अंग माननेवाले जान गये की वसा उतकों की कोशिका कई रासायनिक द्रव्य स्त्रावित (Secrete) करती हैं। इनमे से एक महत्वपूर्ण स्त्राव है, ॲडिपोकाईन। भूख लगना, ग्लूकोज का चयापचय, चर्बी का संतूलन, स्वरोगक्षमता (Autoimmunity) को अबाधित रखना, आदि शारिरिक क्रियाओं में ॲडिपोकाईन की महत्वपूर्ण भूमिका होती है। अभ्यासकोंने स्वरोगक्षमता में ॲडिपोकाईन के प्रदाह के अनुकूल और प्रतिकूल काम करनेवाले अंगो पर काफी काम किया है। शरीर में किसी भी अवयव में प्रदाह उत्पन्न करने का कारण ॲडिपोकाईन भी हो सकते है। लूपस स्वरोगक्षमता की क्षति के कारण उत्पन्न होनेवाली बिमारी है। अनूसंधान में पाया गया है, की शरीर में मौजूद ॲडिपोकाईन की मात्रा और रोग की गंभीरता का सीधा अनुपात (directly proportionate) देखा जा सकता है।

ॲडिपोकाईन के प्रमुख चार प्रकार है। लेप्टिन, रेजिस्टन और केमेरीन प्रदाह उत्पन्न करनेवाले है, और ॲडिपोलोक्टिन प्रदाह की रोकथाम करता है। कई समिक्षालेखों में लूपस के रुग्णों में (सीरम में) मौजूद ॲडिपोकाईन की मात्रा की तुलना निरोगी लोगों में मौजूद ॲडिपोकाईन से की गयी है। हालांकि परिणामस्वरुप अलग अलग जातियों में ॲडिपोकाईन की मात्रा असमान है। जहां मुंबई की आबादी में लूपस के रुग्णों में लेप्टिन घटता हुआ पाया गया, वही कश्मीर राज्य में रुग्ण और सुदृढ जनसंख्या इस तरह का निरिक्षण नहीं दिखाती।

लुपस के रुग्णों का सामान्य वर्गीकरण गुर्दे प्रभावित लुपस और गुर्दे अप्रभावित लुपस होता है। कई जगह के अनुसंधानों में ॲडिपोकाईन की गुर्दे को प्रभावित करने की भूमिका उभरकर सामने आयी है। ॲडिपोकाईन पर होने वाले अनुसंधान गतिमान हो रहे है। किंतु इस दायरे को और विस्तारित करने की जरुरत हैं। ॲडिपोकाईन के आनुवांशिक अध्ययन (genetic studies) से इनके बारे में अबतक अज्ञात तथ्यों का खुलासा हो सकता है।

SPEAKERS AT NIIH DIAMOND JUBILEE CELEBRATIONS

DIAMOND JUBILEE CELEBRATIONS OF NIIH

INTERNATIONAL CONFERENCE ON "REVOLUTION OF LABORATORY MEDICINE IN MODERN BIOLOGY" HELD FROM 15TH TO 17TH OF FEBRUARY 2017 AT MUMBAI

The International Conference on "Revolution of Laboratory Medicine in Modern Biology" was held at Nehru Center on the occasion of Diamond Jubilee of the Institute from 15th to 17th of February 2017. A total of 15 International and 78 National faculty members delivered lectures/chaired various sessions/ conducted panel discussions. One hundred and seventy four delegates from various parts of the country attended the three day Conference. The Maharashtra Medical Council has awarded four credit points.

The Conference was inaugurated on 15th February at 7 PM by the Chief Guest Dr. Soumya Swaminathan, Director General, ICMR and Secretary, Department of Health research. Prof. N.K. Mehra, Chairman, NIIH Scientific Advisory Committee and Dr. Mukesh Kumar, Director, CEPIFRA were the guests of Honour. The Chief Guest Dr. Swaminathan congratulated the Staff members and reiterated the importance of translational aspects of each of the research program which the Institute is undertaking and also highly appreciated the performance of the Institute during the last 60 years. Prof. Mehra and Dr. Mukesh Kumar in their address also appreciated the good research work the institute has been conducting over the years which has direct benefit to the society. Four books were also released by the dignitaries during this occasion which included the Conference Souvenir, a Technical Manual of Immunohaematology, a book on the history of the Institute and a compendium of Hemoglobinopathies in India.

The overall Scientific program included 49 invited lectures, 18 oral papers, four panel discussions, 86 poster presentations and Dr. H.M. Bhatia oration. The topics discussed were quite diverse ranging from transfusion medicine, bone marrow failure syndromes to primary immunodeficiencies, hemostasis and thrombosis and hemoglobinopathies. Some of the highlights of the invited guest lectures are as follows

- The pathophysiology of sickle cell disease beyond the primary mechanism i.e. polymerization of deoxyhemoglobin in the form of cell-cell interactions between all types of circulating cells, abnormalities of vascular tone and nitric oxide metabolism
- The importance of source of factor VIII in eliciting immune response specifically the low immunogenicity of plasma derived factor VIII
- Importance of GATA2 transcription factors in the clinical manifestation of several primary immunodeficiency disorders


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- Negative association of thrombophilia with the first clinically evident thrombotic episode
- Importance of molecular biology investigations in personalized medicine
- The exponential growth of hematopoietic stem cell transplantation in India over the last few years and the role voluntary marrow donor registries

Besides these there were lectures on ethical guidelines to be followed for genetic research in Indian context. There was an interesting lecture on research programmes supported by the Wellcome Trust/DBT India Alliance.There were invited lectures on various aspects of hemoglobinopathies, both on international and national perspectives. There was an update on the gene therapy trials for hemophilia. The session on Hematological malignancies covered the entire landscape of hematological malignancies, while the session on primary immunodeficiencies mainly focused on diagnostic approach to different types of PIDs and newer criteria for diagnosis. There were two sessions in the area of Transfusion Medicine, one highlighting the rare blood group antigens discovered on Indian soil, importance of minor blood group antigens and molecular aspects RhD variants. The session on transfusion medicine also covered a ringside view on the extent of transfusion transmitted diseases in India. There were four panel discussions on important aspects of different disorders which covered topics like inhibitors in hemophilia patients, diagnostic challenges in PID, preventive and management strategies for haemoglobinopathies and controversies and challenges in transfusion medicine. The panelists were laboratory experts as well as clinicians who covered various aspects in laboratory medicine and disease management.

There were 18 highly competitive oral papers in two sessions which were judged by six experts. A total of 86 posters were displayed during the first two days of the conference which were also judged by experts and were awarded prizes.

The conference besides providing information on the basic aspects of each of these immunohaematological disorders also enumerated numerous newer aspects in the diagnosis and management protocols. The panel discussions were highly informative. The recent developments in laboratory diagnosis in different areas of immunohematology by experts were highly beneficial, to medical, paramedical personnel and students who otherwise have limited access to these topics.

AWARDS GIVEN DURING
NIIH DIAMOND JUBILEE CONFERENCE

DEPARTMENT OF LIBRARY & INFORMATION SCIENCE 2017

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GENERAL INFORMATION

NIIH Library and Information Centre has been the key source for the research activity of the institute. At present, it is equipped with modern amenities like WI-FI, LAN, Online database, e-journals and e-books and e-consortiums. Library has print media like journals, books, annual reports, technical reports, reprints, dissertations and thesis on specific topics of Medicine like Hematology and Immunohaematology.

To facilitate end users, NIIH library has renewed the e-journals consortiums like J-Gate Plus, NML-ERMED consortium and ICMR consortium of e-journals like Nature, Science, NEJM and Lancet. Library is automated with SLIM21, advanced automated library software for performing house-keeping activities like acquisition, cataloguing, circulation, serial control and web OPAC online catalogue services. For retro-conversation of Library, digitalization of the resources are made through digital library software.

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- Current Science (P+O)
www.currentscience.ac.in 1932-2017
- Indian J Pediatrics (P+O)
www.ijppediatricsindia.in

6. **WHO Online Journals**

www.who.int

- Bulletin of the WHO (P+O)
www.who.int/bulletin 1947-2017
- Weekly Epidemiological Review(P+O)
www.who.int/wer 1998-2017
- Technical Report Series (P+O)
www.apps.who.int/iris 1951-2017

7. **NML-ERMED e-journal consortia URL's**

- Lippincott Williams and Wilkins
www.lww.com
- WILEY www.onlinelibrary.wiley.com
- BMJ www.journals.bmj.com
- Cambridge University Press
www.journals.cambridge.org
- Oxford University Press
http://oxfordjournals.org

Or

www.ermed.in

8. **J-Gate plus/ICMR e-consortia**

- JCCC-ICMR Consortia
www.jgateplus.com
- ICMR e-consortia
www.icmr.nic.in/icmrnews/e_coortia.htm

Facilities:

- Library is equipped with high speed broadband Wi-Fi internet facility.
- Updated Library details are linked to institute website. www.niih.org.in
- Electronic resource sharing from the nearby library.
- Audio visual and multimedia facilities for video conferencing, webcasting.
- Digitalization of resources which will help the readers to acquire the information at the finger tip.
- Bibliometric analysis of institute publications.

Some Recent Additions

(Apr2016 - Mar 2017)

- Books -10
- Journals -International 27
-National 10
- Bound Volumes -Nil
- CD-ROM/DVD -24

Foundation Day Programme - 28th Feb, 2017

State Govt sponsored workshop on Haemophilia,
Thalassemia and Sickle cell anemia 20-25th March, 2017

Visit of Director General, ICMR & Secretary DHR, Dr. Soumya Swaminathan
10th February, 2017

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