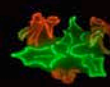




美洲華人生物科學學會



Society of Chinese Bioscientists in America



December 2014

Editor: Gen-Sheng Feng, Ph.D.

Newsletter

Volume 29 Issue 4

Production Editor: Chris Lau, Ph.D.

FROM THE PRESIDENT'S DESK.....

Dear SCBA members:

Welcome to the December 2014 issue of the SCBA Newsletter. First, I take this opportunity to update our progress on organizing the SCBA 2015 International Symposium to be held June 26-29, 2015 in Taipei. After successful recruitment of a star cast of keynote or plenary speakers, we have had an enthusiastic response from our members and colleagues to the call for concurrent session proposals. We have received 66 concurrent session proposals for the 40 concurrent session time slots! The quality of the proposals is outstanding! The scientific program committee has painstakingly worked on the selection of the concurrent sessions, and made difficult decisions to combine certain scientifically related sessions or delete certain sessions. Session chairs will receive instructions soon. I trust that our members will cooperatively work together to build a strong program so that the 2015 Symposium will highlight exciting and valuable scientific progress in the biological and biomedical science and provide excellent opportunities for productive interactions.

Second, the SCBA 2015 International Symposium website will be open soon through which our members and participants can register for the Symposium, submit abstracts for poster presentations, book for housing, etc. SCBA would like to encourage younger generation scientists to participate and submit abstracts for poster presentations. As it is costly for trainees from North America to attend the Symposium in Taipei, SCBA, based on scientific merits, will select 10 abstracts submitted by our trainees from North America for Travel Awards of \$1,000 each (NT\$30,000 each) with registration fee waived. SCBA will also select 10 Poster Awards from all trainee participants at \$100 each (NT\$3,000 each) with registration fee waived. I recommend trainees to apply these prestigious Awards when submitting abstracts.

Third, we will start nomination and election of 2018-2019 SCBA officers under the leadership of SCBA nomination committee chaired by Dr. Kun-Liang Guan. This is very important for the future success of SCBA. I encourage you to

nominate qualified candidates for the coming election follow the link below to see the nomination form with specifications. <http://www.scbasociety.org/2015-Election/2015-Nomination%20form.doc>. The deadline for the proposal nomination is March. 1, 2015. Please submit your nomination via email to both Dr. Kun-Liang Guan and Dr. T.C. Wu at (kuguan@ucsd.edu; wutc@jhmi.edu; and chris.lau@ucsf.edu).

The beginning of the new year is the time of both reflection and anticipation. Looking back at 2014, we had a year that was quite fruitful and dynamic for SCBA. Looking forward, we expect a successful 2015 Symposium and a stronger SCBA.

May this New Year expand our horizons and bring us greater success!

Respectfully yours,



Dihua Yu, M.D., Ph.D.
SCBA President (2014-2015)



NOTES FROM THE SECRETARY

Dear Members of the SCBA,

In this December issue of the newsletter, we report an interesting story about Dr. Amy lee's original work on

GRP78/Bip, a critical regulator of the unfolded protein response and tumorigenesis. Dr. Lee is currently the Associate Director for Basic Research at the Norris Comprehensive Cancer Center of University of Southern California (USC). We share with you the very interesting career path and new accomplishments of Dr. Fu-Tong Liu, Director of the Institute of Biomedical Sciences at Academia Sinica, Taipei. Dr. Liu studied for and got an M.D. degree when he was already an associate professor, having his own laboratory at the Scripps Research Institute. In the September issue, we published an interesting story on Dr. Yang Shi's discovery of the first histone demethylase (LSD1). We are pleased to share with you that the original Cell paper on LSD1 published by Shi's group in 2004 has been selected as one of the 25 historical papers in celebration of Cell 40th Anniversary.

Respectfully yours,



Gen-Sheng Feng, Ph.D.
SCBA Secretary (2014-2015)

NOTES FROM THE TREASURER

Dear Members of the SCBA,

Hope you and your family had a joyous and relaxed holiday season. As you can see from the President's report, the preparation for the 2015 SCBA Biennial Symposium is well underway, and we have received strong financial commitment and support from various organizations in Taiwan to support the Symposium. This quarter we received a \$1,200 donation from Diana Jeang toward the K.T. Jeang Memorial Fund. We have also collected \$3,700 of membership fees. New members are always welcome, especially if you plan to attend the 2015 Symposium in Taipei. Please encourage your colleagues to join this great organization.

Wishing you all the happiness and prosperity in year 2015.

Sincerely,



Hui Zheng, Ph.D.
SCBA Treasurer (2014-2015)

NEWS FROM OUR MEMBERS

DR. AMY LEE ON THE DISCOVERY OF GRP78/BiP, A PIVOTAL REGULATOR OF THE UNFOLDED PROTEIN RESPONSE AND TUMORIGENESIS

Dr. Amy Lee is the Associate Director for Basic Research and Professor of Biochemistry and Molecular Biology at the University of Southern California (USC) Norris Comprehensive Cancer Center, Los Angeles, California. Dr. Lee obtained her B.A. from the University of California, Berkeley in 1970, and her Ph.D. from the California Institute of Technology, Pasadena, California in 1975. Dr. Lee joined USC in 1979 and is currently the holder of the Judy and Larry Freeman Cosmetics Chair in Basic Science in Cancer Research at the Keck School of Medicine USC.



Dr. Lee's research focuses on the mammalian stress response and molecular chaperones. Dr. Lee was the recipient of the American Cancer Society (ACS) Junior Faculty Research Award from 1980-1983 and the ACS Faculty Research Award from 1983-1988. Dr. Lee received the MERIT Award from the National Cancer Institute, USA in 1988. In recognition of her pioneering work on ER stress and its impact on cell and cancer biology, she was elected Fellow of the American Association for the Advancement of Sciences (AAAS) in 2006. She was the recipient of the Chinese American Faculty Association of Southern California Achievement Award in 2008. Dr. Lee chaired a Major Symposium on the Unfolded Protein Response in Cancer at the American Association for Cancer Research (AACR) Annual meeting in 2011. In 2012, Dr. Lee was the recipient of the USC Mellon Mentoring Award.

Here is the story on how she discovered GRP78/BiP

As a graduate student and postdoctoral fellow at the California Institute of Technology, I was fascinated with genes and their regulation. Thus, when I was offered a position as an Assistant Professor of Biochemistry at the University of Southern California (USC) in 1979, I started to search for a model system in mammalian cells to study the mechanism(s) whereby genes that were not physically linked could be coordinately transcribed to fulfil a critical cellular function. The idealism in me urged me to look for something that was entirely new, which had the added advantage that I would have no competitor, at least in the beginning. As I labored at the Caltech library pouring through recent literature, a 1978 letter to Nature caught my eye. There, Jose Melero and Alan Smith (Imperial Cancer Research Fund, UK) described the accumulation of large amounts of three specific proteins of unknown identity with molecular mass of 94, 78 and 58 kilodaltons (kDa) in a temperature-sensitive mutant Chinese hamster cell line (K12) screened for arrest in the G1 phase of the cell cycle. The exciting aspect to me was that this new synthesis was sensitive to actinomycin D, an inhibitor for transcription. However, these same proteins did not accumulate in other G1 arrested mutants in the same screen argued against their function in G1 arrest. So what are these proteins and how are they regulated?

Seizing upon the opportunity to uncover the mystery of the K12 system as the cornerstone of my new laboratory and my first National Institute of Health grant, I contacted Melero and he generously supplied the K12 cells. The National Cancer Institute also liked the novelty so I was in business. In 1981, I succeeded in making a small but amazingly high quality cDNA library of K12 cells overexpressing the three proteins at the non-permissive temperature (40.5°C). Together with my first graduate student, Angelo Delegeane, we selected clones that hybridized preferentially with cDNA made from RNA of K12 cells at 40.5°C, and by using the hybrid selection technique followed by *in vitro* transcription, we identified a full-length cDNA clone containing a 2250 nucleotide insert encoding the 78 kDa protein (PNAS, 1981). Further, *in vitro* translation analysis revealed that this protein was synthesized as a slightly larger species, possessing a slightly more basic charge than the *in vivo* protein. This provided the first hint that this protein was first synthesized and then a short peptide, likely a signal peptide, was cleaved to yield a mature protein. Using the same technique, we also isolated a partial cDNA encoding the carboxyl half of the 94 kDa protein. These cDNA clones became the golden tools to explore the identity, function and coordinated regulation of a novel set of mammalian genes.

It turns out serendipitously that I had cloned the first mammalian cDNA encoding the glucose-regulated proteins GRP78 and GRP94, two major chaperones in the endoplasmic reticulum (ER). The ER is an essential perinuclear organelle where membrane and secretory proteins

are synthesized, assembled and modified, such as N-linked glycosylation. The ER is also the site of lipid synthesis and a major store for intracellular Ca²⁺, which is required for proper protein folding. GRP78 was later discovered to be a major regulator as well as a target of the unfolded protein response (UPR). The UPR is an intracellular quality control system that senses harmful misfolded protein accumulating in the ER and triggers transcription in the nucleus leading to activation of adaptive pathways for survival, or when the stress is too severe, apoptotic death. The protective mechanisms of the UPR include increasing chaperone protein production and degrading the misfolded proteins. The UPR research area is of high significance in both health and disease, as the UPR can protect normal organs against stress, but can also be usurped by cancer cells enabling them to thrive and overcome resistance to therapy. Fast forward to present, GRP78 is now widely recognized as a benchmark of UPR, a multifunctional protein controlling a range of pathways inside and outside of the ER, and plays critical roles in the many facets of cancer development, metastasis and resistance (Nat Rev Cancer, 2014).

However, to start with, how were the proteins induced in K12 linked to the GRPs? Around the time the K12 mutant was isolated, Ira Pastan (National Cancer Institute) reported in 1977 the induction of two viral transformation-sensitive proteins (78 and 94 kDa) in normal fibroblasts by a block in glycoprotein synthesis or glucose starvation, and hence named glucose regulated protein GRP78 and GRP94. While glucose starvation affects the pentose cycle and synthesis of nuclear precursors, one of its consequences is blocking protein glycosylation. In support of this, feeding the cells with N-acetylglucosamine, a metabolite that bypasses the metabolic block at the acetylation step of glycoproteins, restores the amount of these proteins to levels found in normal cells. Thus, these initial studies showed that at least one stimulus for GRP induction is the accumulation of underglycosylated proteins. As the molecular size of the proteins overproduced in K12 cells was nearly identical to GRP78 and GRP94, we quickly tested and confirmed in 1981 that the same proteins overproduced in K12 cells at 40.5°C were also induced when glycosylation was inhibited. This makes perfect sense as in 1986 genetic complementation revealed that the K12 mutation is at the step of transfer of oligosaccharide core to the polypeptides. Between 1984 and 1986, it became clear that the GRPs are also inducible by calcium ionophores, sulphydryl-reducing agents, prolonged anaerobiosis and low pH (Trends Biochem Sci, 1987). Although all these treatments could exert pleiotropic effects, a common denominator is that they all affect ER homeostasis which could negatively impact protein folding. Thus, the induction of GRPs is an ER stress response, not just a glycosylation block response. Importantly, between 1983 and 1986, utilizing the cDNA clones for GRP78 and GRP94 as molecular probes, we demonstrated that stress-induced elevation of the GRPs stems from an increase in mRNA levels, and this provides the first clue of the existence of a monitoring system that allows cells to sense ER stress and

initiate a transcription program in the nucleus to carry out adaptive measures.

To better understand the structure and function of GRP78 and GRP94, it is necessary to obtain the amino acid sequence of two proteins. Taking advantage of the K12 mutant cell line which produced high abundance of both proteins when incubated at 40.5°C, in 1984 we purified these proteins by two dimensional gel electrophoresis and subjected them to amino acid analysis. This approach generated the first amino-terminal sequence for the mature GRP78 and GRP94 proteins and established that they are novel proteins distinct from previously identified heat shock proteins. Subsequently, in 1987, in collaboration with Randal Kaufman (Genetics Institute, MA), the complete amino acid of GRP78 was deduced from sequencing the hamster cDNA clone encoding GRP78, including its ER signal peptide and the C-terminal peptide Lys-Asp-Glu-Leu (KDEL). The latter is the only peptide shared between GRP78 and GRP94 deduced from sequencing of the cDNA clones, and since both proteins are soluble ER luminal proteins, this peptide provides the initial hint that it could be their shared ER retention signal.

At around the same time, Hugh Pelham (Medical Research Council, UK) in search of HSP70 cohorts expressed in non-stressed cells reported the isolation of an unusual cDNA clone (p72) in 1986. Its amino acid sequence as deduced from the cDNA clone was longer at the N-terminus than other HSP70s, and the first 18 amino acids of this extension had typical features of a leader peptide sequence targeting the protein to the ER. Strikingly, the amino acid sequence directly following the putative leader peptide sequence was highly similar to the N-terminal sequence we previously reported for hamster GRP78 and the N-terminus of the mature hamster GRP78 corresponded exactly to the predicted cleavage site of p72, thus p72 is GRP78. Pelham also noticed that p72 shared similar properties with the immunoglobulin (Ig) heavy chain binding protein BiP. The definitive proof came from direct comparison of their peptide maps and cross-reactivity of BiP with an antibody specifically recognizing GRP78. In 1986, Linda Hendershot (U. Alabama) reported that BiP while transiently bound to nascent, wild-type Ig heavy chains, retained incompletely assembled Ig intermediates in the ER. This agrees with work published in 1987 by Kaufman (Genetics Institute, MA) that BiP stably associated with unglycosylated Factor VIII and inhibited its secretion. In 1988, Mary Jane Gething and Joe Sambrook (U Texas Southwestern Medical Center) further demonstrated that malfolded, but not wild-type form of the influenza virus haemagglutinin (HA), was blocked in ER transport and induced the synthesis of GRP78 and GRP94. Hence, the two lines of investigation, stress response and chaperone proteins, were linked, and in 1992, the term "Unfolded Protein Response" was born.

Throughout these exciting years, a major focus of my research team (Augustin Lin, Jerry Ting, Scott Wooden,

Wilfred Li and Peter Baumeister) has been to decipher the mechanism for coordinated stress induction of the GRP78 and GRP94 genes, which are non-contiguous. Using the GRP cDNA clones as probes, we already knew that GRP78 and GRP94 transcription was activated by a wide variety of physiological and environmental stress conditions. To examine this at the promoter level, we used the cDNA clones as probes to isolate the genomic sequences encoding the Grp78 and Grp94 gene from various species, including the human. Then, through the hard work of my graduate student, Binayak Roy, we cracked the genetic code for stress induction of the GRP genes. In 1998, at a meeting in Kyoto, I and Kazutoshi Mori (HSP Research Institute, Kyoto, Japan) announced in tandem the discovery the ER stress inducible promoter element common to UPR mammalian target genes. This was a very gratifying moment considering that two laboratories working continents apart independently reached the same conclusion. This is a highly significant advancement in the UPR research since this makes it possible to work backwards and identify molecular pathways leading to their transcription and then, step by step, find the key players mediating the regulation.

As the UPR research field exploded in the 1990s, I decided that while in vitro studies can provide clean and elegant readouts in a timely manner, I was fascinated by the relevance of the UPR in human disease. My interest in cancer was sparked when Brian Henderson, then the Director of the USC Norris Comprehensive Center, convinced me to move my laboratory there in 1993. Since the Cancer Center is interdisciplinary, the cross fertilization of ideas with immunologists, pathologists, physician scientists and epidemiologists expanded my horizon beyond gene regulation and UPR biochemistry. We obtained the first solid clue linking the UPR and cancer in 1996 when we injected GRP78-knockdown fibrosarcoma cells into syngeneic, immune competent mice. We were totally amazed that tumors either didn't form or they quickly regressed (PNAS, 1996). This is consistent with an earlier observation of our collaborator Gunther Dennert (USC) in 1993 that suppression of GRP78 induction by antisense in these fibrosarcoma cells eliminated resistance to cell mediated cytotoxicity.

Armed with this fantastic early result, we proceeded to understand the mechanism through identifying GRP78 as a potent anti-apoptotic protein. Analogous to the discovery by others that GRP78 forms complex with and maintains the ER transmembrane UPR sensors in an inactive form, in 2003 my postdoctoral fellow Ramachandra Reddy discovered that GRP78 can also complex with and maintain the pro-apoptotic machinery at the ER membrane in an inactive form. In 2004, our team with major effort by graduate student Changhui Mao created a transgenic mouse model using the rat Grp78 promoter for monitoring induction of ER stress in vivo (Nat Med, 2004), and with this model, we visualized transient robust UPR activation in the embryonic heart coinciding with intense glucose utilization and in tumors as they progressed.

To test definitely the role of the GRPs in tumorigenesis, we needed to create knockout mouse models of the GRPs. Anticipating that GRP deficiency might lead to embryonic lethality, my graduate student Shengzhan Luo created a floxed mouse model for GRP78 in 2006, and a floxed mouse model for GRP94 was created in 2010 by Changhui Mao. These provide valuable tools for the creation of conditional and/or inducible knockout models of the GRPs in various tissues. Not only did they provide novel information on the requirement of the GRPs in development and in normal organ function, they allowed us to establish the critical roles and the complex functional interplay of the GRPs in many different types of cancer as well as in diabetes and neurodegeneration. After its initial naming in 1977, GRP78 has now emerged as an exciting new target to suppress tumor growth, metastasis and drug resistance.

Furthermore, with the discovery in 2002 by Salvatore Pizzo (Duke) and in 2004 by Wadih Arap and Renata Pasqualini (MD Anderson) that GRP78 is expressed on the surface of cancer cells, GRP78 can be therapeutically targeted as well as serve as conduits for cancer-specific drug delivery. Promising therapeutics targeting GRP78 include conjugated peptides, toxins, antibodies, small molecules and microRNAs are being developed and a human IgM antibody against a modified form of cell surface GRP78 is in Phase I/II clinical trial (Nat Rev Cancer, 2014). In collaboration with Parkash Gill (USC), a monoclonal antibody against cell surface GRP78 has been isolated capable of suppressing PI3K/AKT signaling, tumor growth and metastasis. In the meantime, drugs against UPR pathways are also actively being exploited in the scientific community as novel agents in treating cancer and other diseases. Looking back, this has been quite a scientific journey and my wish is that the best is yet to come, as we are poised to explore novel angles of GRP and UPR biology, both at the basic science front as well as its applications in medicine.



DR. FU-TONG LIU'S UNUSUAL CAREER PATH AND NEW ACCOMPLISHMENTS AS THE DIRECTOR OF THE INSTITUTE OF BIOMEDICAL SCIENCES (IBMS) AT ACADEMIA SINICA

Professor Fu-Tong Liu assumed the directorship of Institute of Biomedical Sciences (IBMS), Academia Sinica in July 2010. Before he returned to Taiwan he was Distinguished Professor and Chair of the Department of Dermatology at UC Davis.



Dr. Liu is instrumental in the discovery of the family of animal lectins galectins and is a leading investigator in the studies of these family members. His lab discovered the anti-apoptotic function of galectin-3, which is the first

demonstration of the intracellular function of galectins, and demonstrated a number of other intracellular functions of this protein since. By using galectin-3-deficient mice his lab generated, they have provided significant insights into the functions of galectin-3, especially its role in immune and inflammatory responses. His lab has also tackled the biology of galectin-7 and established its pro-apoptotic function, as well as discovered the remarkable tumor suppression activity of this protein. More recently, his lab discovered galectin-12 and has demonstrated its activity in regulation adipocyte differentiation and lipid metabolism.

Early in his career, Dr. Liu developed hybridomas secreting antigen-specific IgE monoclonal antibody, allowing the generation of a large amount of IgE. This revolutionized the field of allergy research and made the studies of a variety of molecular and cellular mechanisms possible. Many of the important discoveries in the field over the last two decades resulted from the use of this IgE antibody.

Dr. Liu was previously Head of the Allergy Research Section at the Scripps Research Institute (1990-1996) and Head of the Division of Allergy at La Jolla Institute for Allergy and Immunology (1996-2001). He is also a board-certified dermatologist (in the US) specialized in medical dermatology. Since he assumed the directorship of IBMS, he has been promoting research in inflammation science and glycobiology in Taiwan. He has also been actively involved in promoting translational research and is currently Associate Director of the Translational Medicine Graduate Program at Academia Sinica and the Program Director of Taiwan Biobank. In 2012, he was elected as Academician of Academia Sinica.

Dr. Liu received his BS in Chemistry from National Taiwan University and PhD in Chemistry from the University of Chicago. He performed postdoctoral research first in Chemistry at University of Illinois, Champaign-Urbana, and then in Immunology at the Scripps Research Institute. He received medical training in a rather unusual fashion. While he was an associate professor with an NIH-funded research group, he attended the two-year PhD to MD program at University of Miami (1985-1987), while maintaining his research group in San Diego. He actually was also serving on an NIH study section during that period. Subsequently, he received dermatology residency training at University of California-San Diego, also while maintaining his NIH-funded research at the Scripps Research Institute.



DR. YANG SHI ON HIS DISCOVERY OF THE FIRST HISTONE DEMETHYLASE, LSD1 (ERRATUM)

Dear Editor,

I am writing regarding a news piece describing the discovery of LSD1, the first histone demethylase in my laboratory, published in the September 2014 issue of the

SCBA Newsletter. After reading of the posted version, I realized that there was a significant typographical error, which needed to be corrected. I am grateful for your subsequent correction and posting of the corrected version online. For those members who had downloaded/received the earliest version, please note on Page 8, 2nd paragraph on the right column, the sentence should read: "The only silver lining was that we were able to exclude the possibility that KIAA0601 was a polyamine oxidase, which, in of itself, was not an insignificant find". Thank you very much for your kind attention.

Sincerely,

Yang Shi, Ph. D.
Boston Children's Hospital
Cell Biology Department, Harvard Medical School
American Cancer Society Research Professor

Editor's note:

The original Cell paper published by Yang Shi's group, reporting identification of the 1st histone demethylase (Cell 119, 941-953, 2004) has been selected as one of the 25 papers into Cell 40th Anniversary Archive, which includes a few Nobel Prize-winning pieces of work.

The First Histone Demethylase Identified
Tony Kouzarides on "Histone Demethylation Mediated by the Nuclear Amine Oxidase Homolog LSD1" by Yujiang Shi, Fei Lan, Caitlin Matson, Peter Mulligan, Johnathan R. Whetstine, Philip A. Cole, Robert A. Casero, and Yang Shi



MEET OUR NEW MEMBERS

New and Renewing Members' Welcome!

The Society of Chinese Bioscientists in America (SCBA) is the largest professional society for elite Chinese bioscientists all over the world. Our SCBA network continues to expand and we encourage everyone to ask colleagues to join and share the many resources and opportunities at SCBA. We would like to give a warm welcome to our newest and renewing members:

Wei-Jen Tang, Ph.D. Life Member, University of Chicago, Chicago, IL

You-Wen He, M.D., Ph.D., Life Member, Duke University, Durham, NC

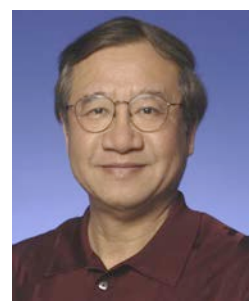
Please refer qualified bioscientists to join the SCBA:

<http://www.scbasociety.org/Membership/Membership.html>

Sincerely,



T.-C. Wu, M.D., Ph.D.
SCBA Co-Executive Director



Chris Lau, Ph.D.
SCBA Co-Executive Director



* Header holly decorations, courtesy of the Laboratory of Dr. Roger Tsien, University of California, San Diego.

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<http://www.SCBAsociety.org/>.

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(chris.lau@ucsf.edu)

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Positions Announcement

Director, Research & Development
Director, Applications & Business Development

Personal Genomics, Inc. (PGI) is a Taiwan-based biotechnology company engaging in the development and manufacturing of a world-leading gene sequencing system by Optoelectronic Single-Molecule Sequencing Technology. The sequencing system offers high-speed, long-read, and low-cost advantages over the current NGS platforms. Once developed and marketed successfully, it will drastically enhance the functionality, speed and cost-effectiveness of sequencing, thus triggering wide-spread healthcare and industrial applications.

PGI, a spin-off from Industrial Technology Research Institute of Taiwan (ITRI) in 2011, was founded by renowned scientists under the leadership of the former President of ITRI, Dr. Johnsee Lee, and invested by reputable electronic and biotech companies including NantWorks, a medical group in USA. The company is located in HsinChu, the silicon valley of Taiwan, and has been able to leverage the technical resources of leading companies and research institutes to develop the state-of-the-art technology and applications. It now employs thirty R&D staff and has an IP portfolio of over hundred patents and applications.

The company is recruiting a Director for R&D to lead its R&D team, and a Director for Applications and Business Development to explore its future applications and business opportunities globally.

The candidate for R&D Director shall possess the following qualifications:

- PhD degree in engineering or related science fields.
- Cross-discipline capabilities covering the fields of biomedical, chemical, and electronics.
- Ability to integrate ideas, initiate product concept, and effectively execute a complete cycle of product development.
- Capable of managing and motivating a R&D organization consisting of technical staff with diverse engineering and science disciplines.

The candidate for Director in Applications & Business Development shall possess the following qualifications:

- PhD degree in genetics, molecular biology or related biomedical fields.
- Good Credential in biology and biomedical research.
- Familiar with genomic and sequencing applications.
- Experienced in business development through collaborations.

Candidates with appropriate qualifications are invited to contact the company by providing their curriculum vitae and a description of past research accomplishment to:

vera@personalgx.com

Ms. Vera Liu

Personal Genomics, Inc.

TEL: +886-3-5910323 Ext.103

195 Chung Hsing Rd., Sec.4, Bldg. 52, 5F-520,
Chutung, Hsinchu, Taiwan 31040

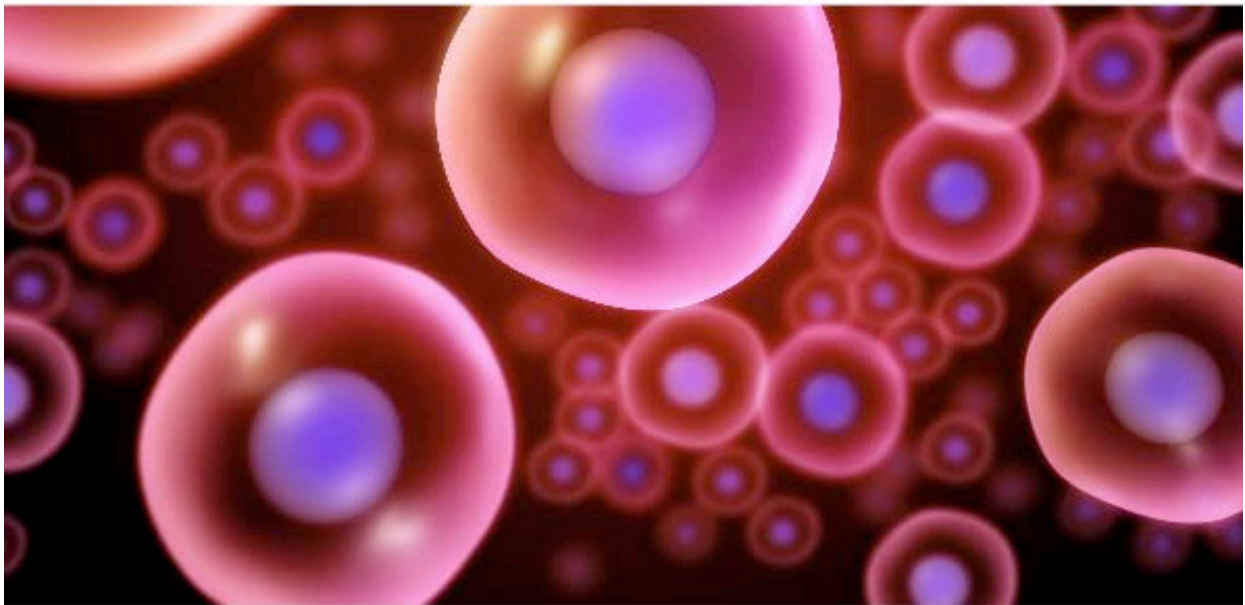
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Society of Chinese Bioscientists in America



The 15th International Symposium



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Yuan Chang



Lieping Chen

Academia Sinica - 中央研究院
Taipei - 台北

June 26 - 29, 2015 . 二零一五年六月二十六 - 二十九日

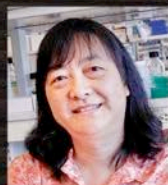
Featured Speakers



Wah Chiu



Ronald DePinho



Xinnian Dong



Susan Horwitz



Nancy Ip



Mu-Ming Poo



Yigong Shi



Thomas Steitz



Li-Huei Tsai



Xiaodong Wang



Chi-Huey Wong



Xiaoliang Xie



Pan-Chyr Yang



Wei Yang

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