The ISHR is a unique cardiovascular scientific society. It is the only cardiovascular scientific society I know of (at least in the US) that is totally run by scientists! This is both a strength and weakness. Because the society is run by scientists we have total control over where we have our meetings, the cost of registration and dues, and all other aspects of the society. The downside is that we rely on volunteers and sometimes our meetings lose money.

The main strengths of the ISHR-North American Section (ISHR-NAS) are our dedicated section members and our Council. Let me start by introducing the ISHR-NAS leadership. I am currently serving as President, until Steve Houser takes over as president in 2015. Don Bers was the President from 2009 to 2012. Litsa Kranias serves as our Treasurer and Jenny Van Eyk is Secretary. Our current Council includes: Mark Anderson, Chris Baines, Joan Heller-Brown, Pieter DeTombe, Sarah Franklin (ECI representative), Asa Gustafsson, Susan Howlett, Tim Kamp, Wally Koch, Gary Lopaschuk, Chuck Murry, Jeff Robbins, Howard Rockman, Jun Sadoshima, Mark Sussman, Jil Tardiff, and Yibin Wang. We also have 7 Interest Groups that organize symposia for our Section meetings. The Interest Groups encompass the interests of the ISHR: Signaling in Hypertrophy and Failure, Cardiac Metabolism, Stem Cell and Gene Therapy, Contractile and Regulatory Proteins, Excitation-Contraction Coupling, Ion Channels and...

Meet the North American Section

Dr. Elizabeth Murphy
Arrhythmias, and Ischemia, Cardio–protection and Mitochondria. These interest groups are each run by a steering committee and members can join up to 3 interest groups. [Joining interest groups is easy: simply sign in to the ISHR website (www.ishrworld.org) and click on Sections (left navigation), North American, and NAS Interest Groups].

Another major strength of the ISHR is the long standing commitment to Early Career Investigators (ECIs). ISHR-International (ISHR-Intl) has a number of awards – but the first award initiated by ISHR-Intl was the Richard J. Bing Award for Young Investigators that was established and first awarded in 1979, soon after the Society was established. The ISHR-NAS established its own Young Investigator Award competition in 1984. In 2009 we expanded to have two Young Investigator competitions: one for graduate students and early postdocs and a second for more senior postdocs. The ISHR-NAS has also set up an ECI NAS network, and this ECI group is currently planning ECI symposia and helping to select speakers for our 2014 meeting in Miami (May 11 to 15). They will also be involved in planning for our 2015 meeting in Seattle. For the World Congress in San Diego, the ECIs organized a very successful two hour social at Dussini’s Loft.

The introduction of the new ISHR website has been a welcome addition. This new web site was designed and established by ISHR-Intl for use by all the ISHR Sections. The ISHR-NAS will use this website for better communication with our members. We have an ECI NAS webpage on this new site. [Join the ECI NAS group by signing in to the website (www.ishrworld.org) and clicking on Groups (right navigation) and ECI NAS]. NAS is also using this new website for dues payment and to manage our membership database.

For ISHR-NAS, as for all societies and scientists, it seems like it is “the best of times and the worst of times”. We have a very vibrant organization focused on meetings with outstanding science, ECI activities, and social networking interaction among members to foster collaboration. However, the current financial climate has made it a struggle to continue in our mission. As discussed in the first issue in this series by Sian Harding of the ISHR-European Section (ISHR-ES), Sections are struggling to find a way “to preserve their solvency as well as identity”. The ISHR-ES has experimented with joining with other European Societies (e.g. the European Society of Cardiology) for annual meetings. The ISHR-NAS explored the option of joint meetings with the Basic Science Council of...
This is the second in a series of articles entitled “Section Beat” written by Officers of the seven ISHR Sections. These articles address current challenges and successes with the goal of increased sharing and communication between ISHR Sections.

The current article is written by Dr Elizabeth Murphy, head of the Cardiac Physiology Section in the National Heart, Lung and Blood Institute at the National Institutes of Health in Bethesda, MD, and President of the North American Section of the ISHR. Dr Murphy introduces the North American Section and discusses the strengths of the Section, as well as the financial challenges associated with hosting independent Section meetings at costly venues.

Dr Murphy is interested in member feedback regarding the format of future Section meetings, so please follow the directions in the final paragraph to weigh in on these important issues.

However, the American Heart Association (AHA); however the AHA was cool to the idea. Thus, in contrast to some other sections, the ISHR-NAS has only ISHR-sponsored meetings. The only source of “income” for the ISHR-NAS is member dues. We have struggled to keep our dues affordable (it is much lower than other cardiovascular societies). Dues revenue is used to fund all activities of the ISHR-NAS, including funding the Young investigator Award competitions held at our section meetings, paying for mailing the Society newsletter, Heart News and Views, to ISHR-NAS members, paying for our part-time Executive Secretary (Leslie Lobaugh), and covering deficits in meeting expenses. Over the past few years, as pharmaceutical industry support has become more limited, we have been spending more on meetings than we collect in dues – clearly this is not sustainable! You might say – why not just reduce support for the meetings. We have tried to cut back on meeting expenses – but the hotels where we hold our meetings have us over a barrel. In order to get the rooms needed for the speaker presentations and posters – the hotels require organizations to sign a contract guaranteeing that we will book a minimum number of rooms at the hotel and spend some minimum (often around $100,000) on food! If fund raising is low and/or if attendees stay at other (often less expensive) hotels, our expenses exceed our income (registration and fundraising are the only two sources of income). Because of these issues – the ISHR-NAS Council is exploring other models for holding meetings. We are exploring renting a conference facility (rather than getting meeting rooms for “free” by promising room and food purchases).
In this model, if fundraising is low that year we can cut back on lunches and receptions (which we can’t do if we have a food minimum). We are exploring this model for our 2015 meeting in Seattle. However, in this model we have no rooms blocked off for the meeting and it is possible that attendees might have trouble finding a room near the meeting site. Another model being considered for future meetings is to hold the meeting at a University, where we can use University facilities at a cost that will hopefully be less than at a hotel. It would be useful to get feedback from members and meeting attendees as to what they “want” in a meeting.

**Do members want the ISHR-NAS to guarantee a block of hotel rooms?** This will mean that you will be able to get a room near the meeting; however if people chose to stay somewhere else – we will have to pay for these rooms and this will cost the society money. How should we balance this?

If we rent a conference facility or hold the meeting at a University, we will not have a food minimum. **How important, for the meeting and for networking, are the lunches, receptions and the banquet?**

We look forward to member feedback on these issues.

[To post a comment regarding these topics in our online forum, go to the ISHR website (www.ishrworld.org), click on Sections (left navigation), North American, Directory and Features (top navigation), Forums, and General Discussion. We value your opinion!]

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**Dr Elizabeth Murphy**

**Bethesda, MD**

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**ISHR-International Council 2013-2016**

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R-P Xiao, China

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**2013 ISHR Award Winners**

Peter Harris Distinguished Scientist Award
Masatsugu Hori, MD, PhD

Research Achievement Award
Eric Olson, PhD

Outstanding Investigator Award
Deepak Srivastava, MD

Distinguished Leader Award
Alicia Mattiazi, MD, PhD

Keith Reimer Distinguished Lecture
Karin Sipido, MD, PhD

Janice Pfeffer Distinguished Lecture
Michael Marber, MB, BS, PhD, FRCP

President’s Distinguished Lecture
Richard Kitsis, MD
Prenatal Genetic Testing in Japan

Two months ago, a Japanese firm (Tokyo) announced the start-up of a new business to send pregnant women to the US to check if their conceived babies have chromosomal abnormalities, such as Down syndrome. This service would cost a client about 4,000 US dollars which covers travel and handling expenses. Cell and Genetic Laboratory would take the blood samples at a medical institution on the West Coast, Guam or in Hawaii and send them to Sequenom Inc. in San Diego for DNA analysis. This announcement aroused a big controversy in Japan as the Japan Society of Obstetrics and Gynecology (JSOG) was mapping out guidelines for prenatal genetic testing using pregnant women’s blood samples. Considering the social criticism, Sequenom changed their policy so that they now offer the test through national medical centers and university hospitals in Japan.

The JSOG adopted guidelines in April for conducting prenatal blood tests to detect three types of chromosomal abnormalities in fetuses: Down syndrome (Trisomy 21), Edwards syndrome (Trisomy 18) and Patau syndrome (Trisomy 13). A body of the Japanese Association of Medical Sciences screened about 20 medical institutions for performing the blood tests. The guidelines request that the selected medical institutions be staffed with an adequate number of genetic counselors. The guidelines also limit the prenatal genetic testing to women giving birth at an advanced age or who have a history of fetal chromosomal abnormalities in previous pregnancies. The new blood tests were first offered at the end of April in Japan. The genetic test can be done around 10 weeks into a pregnancy; the test is carried out earlier than amniocentesis without the risk of miscarriage or infection. The difference in methylation of specific DNA sequences between mother and fetus can be used to identify fetal-specific DNA in the maternal blood. This technique was first developed in 2011, and became commercially available soon after the massive clinical evaluation of the screening test.

In general, prenatal diagnosis has three purposes: (1) to enable timely medical or surgical treatment before or after birth, (2) to give the parents the chance to abort a fetus if necessary, and (3) to give the parents the chance to prepare psychologically, financially and medically for a baby with a health problem. Down syndrome is often associated with cardiac defects which may require appropriate surgical intervention upon birth. Although fetal corrective interventions are rare, prenatal surgeries are now being used for fetuses with spina bifida. Early diagnosis gives the parents time to research postnatal treatment and care, or in some cases, abortion. Genetic counselors are usually called upon to help families make informed decisions.

Many expectant parents would like to know the sex of their baby before birth. This is easily diagnosed by amniocentesis with karyotyping, and/or prenatal ultrasound. In some countries, however, health care providers are expected to withhold this information from parents. In Japan, health care providers are expected to reveal this information so that the parents can better prepare themselves for naming the baby and purchasing the layette. As for the chromosomal abnormalities, a survey was conducted to find how women felt about noninvasive diagnosis of fetal aneuploidy. It was reported that 82% of pregnant women and 79% of female medical students view this type of diagnosis in a positive light, agreeing that it is important for prenatal care. However, it is critically important that the parents should be well informed if they have to consider abortion vs continuing a pregnancy when the result of the prenatal genetic test is positive.

Society’s keen interest in this issue may be attributable to the high prevalence of deliveries by women of advanced age and to the decreasing population of children in developed countries. We are always fighting against age–related genetic abnormalities – cancer may be another target of this effort.

Masatsugu Hori, M.D., Ph.D.
President ISHR
Mortality in patients with acute myocardial infarction (MI) decreased about 80% since the 1960s, when between a quarter and a half of these patients died in hospital (1-4), to 8.4% and 4.6% in 1999 and 2006 (5). Although these comparisons are limited by differences in diagnostic criteria and patient characteristics, the improved survival is clearly a major success of modern cardiology. Prevention of arrhythmia deaths by coronary care units, which were introduced in the 1960s, accounts for much of the improved outcome, but two discoveries regarding hemodynamic collapse in these patients also played a role. The first, made possible by understanding of the pathophysiology of hypotension following large, usually anterior wall, infarctions (cardiogenic shock), occurred in the 1950s. The second, recognition of the role of a vagal (parasympathetic) reflex in reducing blood pressure and slowing the heart in patients with inferoposterior infarctions, was made in the early 1960s. Both reduced iatrogenic deaths by discrediting potentially lethal treatment strategies.

**Cardiogenic Shock**

In the 1950s, it seemed logical to treat hypotension complicating MI using inotropic drugs and vasoconstrictors. Regarding the latter, Hellerstein et al. suggested:

> The ideal pressor drug would elevate blood pressure, increase peripheral resistance, produce a proportionate increase in coronary flow, have minimal side effects, and would not decrease cardiac output or produce serious arrhythmias (6-18).

Although most sympathomimetic amines had been observed to stimulate the heart and cause vasoconstriction, it was becoming clear that responses to different drugs in this class vary. The reason had been suggested in 1948 when Ahlquist (7), using the concept of receptors that had been proposed at the beginning of the 20th century, postulated that responses to sympathomimetic amines depend on the type of adrenergic receptor to which the drug binds. These differences led Gazes *et al.* (8) to compare two sympathomimetic amines that increase blood pressure: phenylephrine, a powerful vasoconstrictor that binds to \( \alpha \)-receptors on blood vessels, and norepinephrine which, because it also activates cardiac \( \beta \)-receptors, is an inotrope in addition to a vasoconstrictor. Their finding that 6 of 7 patients who received norepinephrine recovered, whereas only 2 of 7 given phenylephrine “emerged from shock”, indicated that combined vasoconstriction and inotropic stimulation is superior to vasoconstrictor therapy alone. Evidence that increasing peripheral resistance reduces cardiac output in dogs with cardiogenic shock (9) also argued against the use of drugs that are primarily vasoconstrictors. However the next logical step, using vasodilators to increase cardiac output in these patients, met strong resistance, even though these drugs relieve angina. In 1966, for example, Friedberg’s authoritative text argued that “because of its hypotensive effect nitroglycerin should not be taken for the pain associated with the infarct” (10).

Resistance to using vasodilators in patients with cardiogenic shock reflected emphasis on the easily measured fall in blood pressure rather than the less obvious decrease in cardiac output. Yet there had been clues that increasing tissue perfusion could also be important. In 1954, as a second year medical student, I participated in a course run by Lewis Dexter, one of the first to catheterize cardiac patients, in which we discussed the view that the most appropriate goal in treating shock should be to maintain cardiac output rather than blood pressure. Herbert E. Kaufman, a classmate who had worked in cardiovascular research before entering medical school, went so far as to suggest that vasodilators might be used to increase cardiac output in these patients, even if blood pressure was lowered. I recall that all of us, including Dr. Dexter, nodded our heads in agreement, but because neither the understanding of pathophysiology nor the technology for measuring cardiac output were sufficiently advanced, our discussion moved to other topics.

It was not until 1972 that Franciosa *et al.* (11), using nitroprusside, and Gold *et al.* (12), using nitroglycerin, demonstrated that vasodilators could increase cardiac output and reduce left atrial pressure.
without causing a significant fall in blood pressure in patients with acute MI and high left ventricular filling pressure.

The von Bezold-Jarisch Reflex
In 1954 Dawes and Comroe, reviewing chemoreflexes arising in the heart and lungs, described the ability of sensory receptors in the heart to reduce blood pressure and slow heart rate (13). First observed in 1867 by von Bezold and Hirt and confirmed in the late 1930s by Jarisch, this reflex induces an intense vagal discharge that causes sinus bradycardia, atrioventricular (AV) block, peripheral vasodilatation and hypotension. The reflex was subsequently found to be triggered by posterior and inferior MI, where it can lead to a syndrome that resembles cardiogenic shock caused by large left ventricular infarcts, but because the reflex is reversible and not associated with extensive myocardial necrosis the prognosis is much better. I observed, but did not understand this reflex in 1956, when as an intern I managed an 80 year old man with an inferior MI complicated by bradycardia and severe hypotension. In accord with accepted practice I gave him norepinephrine intravenously, but although this increased his blood pressure it initiated long runs of ventricular tachycardia for which the only treatment at that time was intravenous procaine amide. The latter caused severe hypotension, which required additional norepinephrine, which again led to ventricular tachycardia, for which I gave more procaine amide, which was again followed by hypotension, more norepinephrine, etc. These cycles continued every ~30 minutes for half a day and then abated. The patient eventually left the hospital and returned to his farm where several months later, I was amazed to learn, he built a barn. I had no idea at the time what had caused all this, but in 1963 a landmark paper by Costantin (14) identified the mechanism as the von Bezold-Jarisch reflex. This made it clear that instead of using high doses of a sympathomimetic amine, I should have treated the patient with atropine, a muscarinic receptor blocker (15). The advantage of the latter is that it reverses the underlying pathophysiological abnormality, whereas norepinephrine initiates a new set of problems that includes triggered ventricular tachycardias. The latter occur when β-adrenergic stimulation activates L-type calcium channels which, by allowing more calcium to enter the cardiac myocytes, increases the depolarizing current generated during the vulnerable phase of the cardiac action potential when the sodium/calcium ex−changer removes calcium from the cytosol.

Conclusion
Until the 1960s two potentially lethal treatment errors increased mortality in patients with acute MI and hypotension. The first, administration of vasoconstrictors to already severely vasoconstricted patients with cardiogenic shock, not only failed to correct the underlying pathophysiological abnormality but made these patients worse by increasing afterload and reducing tissue perfusion. The second, use of sympathomimetic amines to correct hypotension caused by the von Bezold-Jarisch reflex, alleviated some adverse consequences of the vagal stimulation but caused arrhythmias that added to mortality. The interplay between basic science and clinical medicine that corrected these errors improved survival by causing the inappropriate therapies to be abandoned.

References

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The Keith Reimer Distinguished Lecture 2012
(May 2011; Belgrade, Serbia)

HDAC Regulation in Cardiac Hypertrophy: Novel Phosphorylation-Dependent and Independent Mechanisms

Metin Avkiran, Ph.D., D.Sc.

Professor Avkiran is Professor of Molecular Cardiology and Deputy Director of the Cardiovascular Division at King’s College London. He trained at the University of Bath, United Kingdom, where he received a BSc Honours in Pharmacology and a PhD in Cardiovascular Pharmacology. In 2002, he was awarded a DSc by the same University, in recognition of his published research on the cardiac Na+/H+ exchanger. Since 1987, Professor Avkiran has held various research and academic appointments at the United Medical and Dental Schools of Guy’s and St Thomas’ Hospitals in London, and subsequently at King’s College London. These included a prestigious 10-year personal Career Development Award from the British Heart Foundation. He was appointed as Professor of Molecular Cardiology at King’s College London in 2001.

Professor Avkiran is currently the President of the International Society for Heart Research (ISHR), which he has previously served as Secretary General for consecutive terms (2004-2007, 2007-2010). He has also served as the Secretary (1996-1998) and Chairman (2000-2002) of the British Society for Cardiovascular Research, as a member of the ISHR European Section Council (1995-2001) and the ISHR World Council (2001-2004), and as the Chairman of the Myocardial Biology Panel of the British Cardiac Society. In 2000, he was the recipient of the Pfizer Award in Biology (in recognition of his work on the cardiac Na+/H+ exchanger). He is currently an Associate Editor of the Journal of Molecular and Cellular Cardiology and serves on the Editorial Boards of Basic Research in Cardiology, Cardiovascular Research, Dialogues in Cardiovascular Medicine and the Journal of Physiology. He has previously served as an Associate Editor of Cardiovascular Research (1991-1995) and Pharmacology & Therapeutics (2001-2005) and on the Editorial Boards of Circulation (2000-2004) and the British Journal of Pharmacology (2002-2005). Professor Avkiran was Chairman of the Scientific Programme Committees of the XIX ISHR World Congress in 2007 (Bologna, Italy) and the XX ISHR World Congress in 2010 (Kyoto, Japan).

The research in Professor Avkiran’s Molecular Cardiology Group is focused on the mechanisms underlying cardiac injury and dysfunction (e.g. arrhythmias, contractile dysfunction, infarction), particularly in the contexts of ischaemic heart disease and heart failure, with the work aiming to contribute to the development and evaluation of novel interventions for the inhibition and/or reversal of such injury and dysfunction. The Group’s approach integrates physiological and pharmacological approaches with biochemical and molecular biological techniques, with recent and ongoing studies focused on: (1) Cardiac signalling by G protein-coupled receptors and downstream transduction pathways, with emphasis on protein kinases (particularly protein kinase D [PKD] and p90 ribosomal S6 kinase [RSK]), phosphatases (particularly type 2A protein phosphatase) and redox-sensitive mechanisms; (2) The pathophysiological roles and molecular regulation of the cardiac sarcolemmal Na+/H+ exchanger (NHE) and the therapeutic potential of pharmacological NHE inhibitors. Recent contributions include the identification and functional characterization of novel sarcomeric substrates for PKD and RSK, and the delineation of phosphorylation-dependent and independent mechanisms for the neurohormonal regulation of histone deacetylases implicated in the transcriptional control of cardiac hypertrophy.
The President’s Distinguished Lecture 2012 (October 2012; Fukuoka, Japan)

The Role of Calcium in Cardiac Hypertrophy and Failure

Dr Steven Houser

Dr. Steven Houser, Laura H. Carnell Professor of Physiology and Medicine, Director, Cardiovascular Research Center and Chair of Physiology started at Temple as an Assistant Professor in 1978. His research initially focused on the electrical and mechanical properties of the heart and the alterations in these properties that contribute to depressed cardiac performance in heart failure. This was a relatively stagnant field in the late 70’s due to the fact that cellular systems amenable to rigorous biophysical analysis were not available for heart cells. Dr Houser was the first person to develop techniques for isolation of Ca\textsuperscript{2+} tolerant myocytes from large animals. He and his colleagues used these isolated single cells to uncover exquisite details of cardiac ion channel biology, excitation-contraction coupling and Ca\textsuperscript{2+} regulation in the normal and diseased heart. The Houser laboratory has been at the forefront of the field of cardiac Ca\textsuperscript{2+} regulation and excitation-contraction coupling for the past 30+ years. His laboratory has been instrumental in the current understanding of the cellular and molecular bases of contractility defects in the failing human heart. In collaboration with his former trainee (K08) Dr Kenneth Margulies, his work has focused on the alterations in myocyte Ca\textsuperscript{2+} regulation that cause depressed contractile reserve in the failing human heart and improvement in these processes that contribute to improved cardiac function after support of failing hearts with mechanical assist devices.

Dr Houser’s recent research has focused on the idea that that heart failure does not result from fundamental defects in myocyte Ca\textsuperscript{2+} regulation, but instead, from Ca\textsuperscript{2+} mediated cell death. Current studies are exploring the idea that the increased Ca\textsuperscript{2+} necessary for the myocytes of the dilated, failing heart to work against increased systolic wall new stress, induces alterations in myocyte Ca\textsuperscript{2+} handling (in an attempt to prevent cellular Ca\textsuperscript{2+} overload) and predisposes the myocyte to apoptosis and necrosis. These studies are being performed in cellular systems and transgenic mouse models with cardiac specific, inducible expression of T-type Ca\textsuperscript{2+} channels. A recent study from the Houser laboratory showed that the adult heart has some capacity to repair itself via the generation of new cardiac myocytes.

The newest research in the Houser laboratory is related to the remodeling of the heart that takes place after an ischemic insult (myocardial infarction, MI). Dr Houser is the PI of an NHLBI PPG to study post MI remodeling and develop novel therapeutics for the treatment of post MI remodeling. This PPG is being performed in collaboration with Drs Walter Koch and Jeffrey Molkentin.

Another major aspect of the ongoing work in the Houser laboratory challenges another long held dogma that the adult heart is incapable of generating new cardiac myocytes. The Houser laboratory has recently shown that myocyte number increases in late adolescence and these new myocytes appear to be derived from a resident cardiac stem cell. Ongoing studies suggest an important role of T-type Ca\textsuperscript{2+} channels in stem cell derived new myocyte generation. These topics are being carried out with cell culture systems and in transgenic mice with cardiac specific, inducible expression of T-type Ca\textsuperscript{2+} channels. The role of calcium in cardiac hypertrophy and failure...
Report on the XI Congress of the Chinese Section (November 20-24, 2012; Guangzhou, China)

This congress was organized by Guangdong Academy of Medical Sciences (Guangdong General Hospital) and Guangdong Provincial Cardiovascular Institute. The theme of this congress was “Today’s Science for Tomorrow’s Medicine”. The meeting was well-organized and well-attended by registrants, which contributed to making this an important event for elevating the academic level of cardiovascular pathophysiology and translational medicine in China.

The XI Congress of the ISHR - Chinese Section was held in conjunction with the 14th Scientific Meeting of the Chinese Association of Pathophysiology, Cardiovascular Section and the 9th Scientific Meeting of the Chinese Association of Pathophysiology, Receptor Section. The venue, the conference building of Guangzhou Pearl River Hotel, was an ideal place for scientific discourse. This meeting attracted over 400 participants (more than 20% of the participants were clinical physicians), and this meeting provided both physicians and scientists with a wonderful platform for academic communication.

There were eight plenary lectures and twelve sessions of special reports on stem cells, cardiovascular remodeling and heart failure, atherosclerosis and coronary heart disease, cardiovascular protection and microRNA modulation, regulatory molecules in signaling transduction and more. The congress also included two mini symposia, five luncheon seminars, the Young Investigator Award (YIA) sessions and poster sessions.

Five plenary lectures took place during the opening ceremony, and were, of course, not to be missed. Dr De-Pei Liu (Chinese Academy of Medical Sciences) gave a talk on the protective roles of SIRT1 in cardiovascular diseases. Dr Masatsugu Hori, President of ISHR-Intl (Osaka Medical Center for Cancer and Cardiovascular Diseases, Japan), talked about the role of autophagy in heart failure and aging. Dr David Eiser, JMCC Editor (Univ of Manchester, UK), gave a nice talk on the control of intracellular calcium in the heart and the link to arrhythmias. Dr Yibin Wang (UCLA, USA) talked about “Stress kinases and phosphatases in cardiac remodeling and heart failure”. Finally, Dr Ping Wang (Feinstein Institute for Medical Research, USA) also presented a nice talk on milk fat globule-EGF factor 8 attenuating inflammation and injury.

In the first mini-symposia entitled, “Research Grant Application and Research Design”, Dr Rui-Juan Sun (National Natural Science Fund Committee of China) elucidated guidelines and strategies for applying for funding for medical research in China, and Dr Chao-Shu Tang (Peking University Health Science Center) enthusiastically discussed experimental design in medical research. In the second mini-symposium entitled, “Communicating with the editors”, Dr David Eiser gave a talk on “The Journal of Molecular and Cellular Cardiology: how to (and how not to) get a paper published”. Dr Xiao-Jun Du (Baker IDI Heart and Diabetes Institute, Australia) talked about “Scientific English writing: feedback from the editorial office of the Clin Exp Pharmacol Physiol”. Both mini symposia were extremely popular and successful. The audiences were quite satisfied with the interesting information on research design, scientific paper writing and publication that they received.

One of the highlights of this meeting was two sessions of “Clinical Research” featuring talks by 10 cardiovascular scientists. Dr Xiao-Ying Li (PLA General Hospital) reported on a survey of coronary heart disease therapy and secondary prevention in elderly patients in China. Dr Dong-Feng Gu (Fu Wai Hospital, CAMS & PUMC) gave a talk about “Genetic risk factors of atherosclerosis and coronary heart disease and clinical significance”. Dr Ning Tan (Guangdong Provincial Cardio–
vascular Institute) talked about “Late events in patients after implantation of drug-eluting stents”. The remaining speakers addressed the clinical applications of β-receptor inhibitor and RAAS blockers and some other interesting clinical issues.

Another highlight of the meeting was the YIA competition and poster display. Seventeen candidates presented their distinguished work in two separate sessions. All the presentations were excellent and eleven winners were announced at the closing ceremony. The winners of first grade were: Dr Yuan-liang Ma (Peking University Third Hospital) and Dr Yu-liang Feng (Guangdong General Hospital). There were also three poster sessions in which 128 investigators presented their data. Dr Hao Li and 29 additional participants were selected for Excellent Poster Awards.

Another very important event that took place during this meeting was the ISHR-CHI Section assembly where Dr Xi-Yong Yu from Guangdong Academy of Medical Sciences was elected as the new chairman of the Chinese Association of Pathophysiology, Cardiovascular Section and
Dr. Thomas Eschenhagen is Professor of Pharmacology and Director of the Department of Experimental Pharmacology and Toxicology at the University Medical Center Hamburg Eppendorf, Germany. Dr Eschenhagen studied Medicine (1978-1986) and completed his experimental M.D. thesis in Pharmacology and Toxicology in 1988 at the Medical School Hannover under the direction of Prof. Hans Herbert Wellhöner. After a three years residency in Internal Medicine at the Vinzenz Hospital Hannover (1986-1989) he trained as Pharmacologist at the Institute of Pharmacology at the University Hospital Hamburg Eppendorf under the direction of Prof. Hasso Scholz. After his habilitation in Experimental and Clinical Pharmacology and Toxicology in 1994 he received a prestigious Heisenberg Stipend of the German Research Foundation (DFG) and spent seven short-term fellowships (2-6 months) in Washington University School of Medicine, St. Louis, MO (Prof. Elliot L. Elson), Stanford Research Institute, Menlo Park, CA (Dr Nanette Bishopric), National Institute on Aging, Baltimore, MD (Dr Edward G. Lakatta), and INSERM Unit 544 in Chatenay-Malabry, Paris, France (Dr Rodolphe Fischmeister). In 1998 he became Chair of the Institute of Clinical Pharmacology at the University of Erlangen, Germany and in 2002 he took his present position in Hamburg.

Dr Eschenhagen has received numerous awards and honors, including the Martini-Award, University of Hamburg (1991), the Rudolf Thauer Award, German Society of Cardiology (1992), Sandoz Award for Translational Science (1995), Fraenkel Award of the German Society of Cardiology (1997) and the Ursula M. Händel Award for the Replacement of Animal Experiments of the German Research Foundation in 2011. He was named Fellow of the American Heart Association in 2004, Fellow of the European Society of Cardiology in 2010, and Fellow of the International Society of Heart Research in 2010. Since 2004 he is member of the Academy of Science, Göttingen and since 2008 member of the German Academy of Science Leopoldina. He is currently President of the ISHR European Section (2010-2012) and was recently voted member of the Senate’s commission on Sonderforschungsbereiche (center research grants) of the German Research Foundation. Since 2011 he is coordinator and speaker of German Center of Cardiovascular Research, a long term, high volume grant initiative of the German Ministry of Research involving 7 partner site and 24 institutions. Between 2004 and 2012 he served as panel member of the reviewing board of the German Research Foundation and is frequently involved in international grant reviewing including INSERM, ANR (France) Netherland Heart Foundation, British Heart Foundation, and Swiss National Fonds. He participated in two roadmap procedures for the NIH (2007) and the German Ministry of Research (2007). He is in the editorial board of numerous peer-review journals, and serves at the Board of the German Foundation for Heart Research, Foundation for Chronic Heart Disease, ADUMED-Foundation, Hufeland-Award, and Galenus-von-Pergamon-Award Committee.


Dr Eschenhagen is perhaps best known for his pioneering work on 3-dimensional engineered heart tissue (EHT) from primary cardiac cells. In collaboration with Dr Elson’s group in St. Louis he first described a method to generate spontaneously beating, force-generating 3D heart tissue from embryonic chick hearts (FASEB J 1997). The original lattice technique was
Vice-President of the ISHR-Chinese Section. The 6th Executive Committee (2013-2016) of the ISHR-CHI Section consists of 30 council members and 13 young members. The leaders of the ISHR-CHI Section are:

Honorary President: Chi-De Han
President: Ru-Tai Hui
Vice President: Huang-Tian Yang, You-Yi Zhang, Xi-Yong Yu
Secretary: Yi Zhu
Treasurer: Li-Ling Wu

During this congress, eighty-six participants applied for membership in the ISHR-CHI Section and were successfully certified as members. Currently, the ISHR-CHI Section has 607 members.

The enthusiasm and support of all of the participants and the hard work of the local organizing committee contributed to the extremely successful ISHR 2012-CHI Section congress. This meeting definitely encouraged all of the cardiovascular scientists and clinicians to achieve further scientific progress in cardiovascular translational research. After such a fantastic and impressive gathering, we look forward to the 2014 meeting in Harbin with much anticipation!

Zhi-Xin Shan and Yi Zhu

quickly adapted to neonatal rat cardiac myocytes (Biotechnol Bioengin 2000) and modified to a ring-EHT format with better tissue development and easier handling (Circ Res 2002, 2005). Originally designed as an improved in vitro model for drug testing and target validation, this technique, in combination with recently established protocols to generate EHTs from human embryonic stem cell- and human induced pluripotent stem cell-derived cardiac myocytes (PloS One 2011), has opened new perspectives in biomedicine (e.g. medium throughput drug screening, LQT and cardiotoxicity testing, and iPS-mediated disease modeling) and cardiac repair (Circulation 2002, 2006, 2007, Nat Med 2006).

Dr Eschenhagen’s fourth research focus, in collaboration with Dr Lucie Carrier, Paris (Dr Carrier is in Hamburg since 2005), explores the functional genomics of hypertrophic cardiomyopathy. The joint research effort resulted in the first evidence that impairment of the ubiquitin proteasome system may play a pathophysiological role in HCM (Cardiovasc Res 2004), and found that dysfunction/deficiency of cardiac myosin binding protein C (cMyBP-C), a common affected protein in HCM, leads to increased Ca^{2+} sensitivity of myofilaments and thereby to relaxation deficits and diastolic dysfunction (Circ Res 2007, 2009), suggesting that the latter is a primary defect in HCM and not the consequence of cardiac hypertrophy. This hypothesis and therapeutic approaches are presently being tested in a hiPS-EHT disease modelling approach.

Dr Eschenhagen is also an engaged teacher and mentor. He authored several textbook chapters on cardiovascular pharmacology and drug politics, and he is an active contributor to patient information via the German Heart Foundation. He has trained more than 60 M.D. thesis and 20 Ph.D. thesis students. Dr Eschenhagen promotes training of young scientists on several levels including career talks at the German Society of Cardiology, the creation of an MD/PhD program and an interdisciplinary Cardiovascular Research Center in Hamburg as well as an extensive training program launched in the framework of the German Center for Cardiovascular Research. His young, international research team in Hamburg and the open-minded human touch testify the success of this engagement.
Dr. DARIA MOCHLY-ROSEN is a Professor in the Chemical and Systems Biology, the Senior Associate Dean for Research and the director of SPARK, a translational research program at Stanford University School of Medicine. She leads a multi-disciplinary research lab that includes chemists, biochemists, biologists and physician scientists and has used her basic research discoveries to develop a number of drugs for human diseases.

Her basic research focuses on understanding how protein-protein interactions govern cell signaling. A protein chemist by training, she predicted and then confirmed that intracellular protein-protein interactions can be interfered with short peptides derived from one of the partners that mimic the interaction site (JBC, 1991; PNAS, 1994, 1995; Science, 1995). This approach led to the discovery of the only highly selective protein kinase C (PKC) inhibitors and activators (Nature Biotechnology, 1998; PNAS, 2001; Circulation, 2003; etc). These peptide regulators of PKC identified the role of this family of enzymes in a variety of diseases and therapeutic modalities including cell therapeutics and acute and chronic treatments for ischemic diseases, such as myocardial infarction, stroke, heart failure, peripheral artery disease and organ transplantation.

As part of her long standing interests in understanding the molecular basis of cardiac protection, her lab used an unbiased proteomic approach that unexpectedly identified aldehyde dehydrogenase 2 (ALDH2), the rate determining enzyme in ethanol metabolism, as a key regulator of cell survival under oxidative stress. These observations were possible because the lab designed a novel assay to screen for activators of ALDH2, called Aldas (for ALDH activators) Science, 2008). Importantly, Aldas correct a structural mutation in ALDH2 found in ~0.5 billion East Asians and therefore represents a new class of drugs that serve as molecular chaperons (Nature Structure and Molecular Biology, 2010). Aldas also prevent nitroglycerin-induced tolerance and improve outcome after myocardial infarction in animal models (Science Translational Medicine, 2011). Very few selective activators of enzymes have been described. Further, because defense from oxidative stress is a common factor in determining cell survival, current research in the lab examines the benefit of activating ALDHs in a variety of diseases and therapeutic modalities including cell therapeutics and acute and chronic treatments for ischemic diseases, such as myocardial infarction, stroke, heart failure, peripheral artery disease and organ transplantation.

Her interest in translational research led her to teach drug discovery classes for the past 18 years and to her establishing SPARK, a drug development program at Stanford University School of Medicine, May 2012; Banff, AB, Canada). Very few selective activators of enzymes have been described. Further, because defense from oxidative stress is a common factor in determining cell survival, current research in the lab examines the benefit of activating ALDHs in a variety of diseases and therapeutic modalities including cell therapeutics and acute and chronic treatments for ischemic diseases, such as myocardial infarction, stroke, heart failure, peripheral artery disease and organ transplantation.

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About the Award...

Each year, the International Council selects a speaker to deliver the Janice Pfeffer Distinguished Lecture at the World Congress or at the annual section meeting of one of the three largest ISHR Sections. The purpose of this lecture is to honor the memory of Dr Pfeffer and to recognize her contributions to cardiovascular research. The topic of the lecture must be in the field of remodeling, heart failure and/or hypertrophy. The speaker receives a plaque and $1,000 honorarium in addition to travel expenses.
University School of Medicine. This unique program provides education in drug development and supports the school’s mission in translational research. Now in its sixth year, ten projects out of twenty six that completed the program are in human clinical trials and ten inventions were licensed to biopharmaceutical companies. The ability to take over 50% of these early inventions through “the valley of death” between academic research and patient care demonstrates again her non-orthodox yet effective approach to address problems and lead to a change – SPARK a change.

Dr Mochly-Rosen received her Ph.D. at the Weizmann Institute in Israel in Chemical Immunology. After two-years of post-doctoral training in the laboratory of Dr Daniel E Koshland, Jr., where she began the biochemical study of protein kinase C (PKC), she moved in 1986 to the University of California San Francisco, where she rose in the ranks to Associate Professor in Residence in the departments of Neurology and Pharmacology. In 1993, she was recruited to the department of Molecular Pharmacology, now called Chemical and Systems Biology, at Stanford University. She became the Chief and then the Chair of the department in 2001, helped catalyze the scientific direction of the department to increase the chemistry strength, led the hire of three new faculty members and established a high-throughput screening facility. In 2006, she stepped down as the Chair and became the Senior Associate Dean for Research at the School of Medicine, a position that she still holds. She held the Reed-Hodgson Endowed Chair in Human Biology between 1996 and 2001 and in 2006 became the Inaugural George D Smith Professor in Translational Medicine.
HEART NEWS AND VIEWS

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