For as long as I can remember I wanted to become an academic cardiologist. I was therefore fortunate to have been born 80 years ago in the United States, to have been educated at the beginning of the remarkable expansion of science that followed World War II, and to have had a father, Louis N. Katz, who was a leading figure in academic cardiology. A liberal arts education at the University of Chicago opened my mind to the wealth of human knowledge and taught me how ideas develop. My enthusiasm in a student laboratory led to an invitation to spend the summer of 1951 working with a doctoral student on the inhibition of yeast metabolism by fatty acids. I helped collect enough data for a PhD thesis and a paper, and so was asked to spend the
A liberal arts education at the University of Chicago opened my mind to the wealth of human knowledge and taught me how ideas develop.

After finishing medical school and a medical internship at the Massachusetts General Hospital, I spent 1957-1959 at the NIH with Chris Anfinsen and Marty Rodbell, both of whom were to win Nobel Prizes. My project was to correlate the amino acid sequence of a bacteriophage protein with an existing genetic map. Unfortunately I was unable to dissociate the virus proteins, most likely because it had evolved to survive in sewers, so that my only publications were a methodology paper (which became a Citation Classic), a short paper on hemoglobin, and an article on ancient Greek medicine. I did, however, learn several chemical and physical methods that proved invaluable for my later research. After completing medical residency in 1960 I spent a year in London with Paul Wood, a world-renowned cardiologist whose clinical evaluations were masterpieces of hypothesis-driven logic. Upon hearing the chief complaint, Wood would propose a working diagnosis that he evaluated using the history and physical findings, after which he would predict the electrocardiographic, X-Ray and catheterization findings. He then developed a therapeutic plan and used the patient’s responses to therapy as additional tests of his hypotheses. For the rest of my career I used this iterative approach both to evaluate patients and carry out research.

In 1961 I returned to basic research as an American Heart Association Research Fellow in Wilfried Mommaerts’ laboratory in Los Angeles. Using methods I had learned in Anfinsen’s laboratory I began by measuring the sulphydryl groups in skeletal actin. I found 6, but because earlier literature said there were 4, I was directed to confirm this finding using a second method. After again finding 6 I was told to do additional experiments using a third method, which gave the same result. This experience defined me as a careful worker, which subsequently paid off when I found that cardiac contractile proteins are not affected by digitalis or norepinephrine – I later learned that although the leading journals that published my results rarely accepted negative papers, the quality of my data mandated that my findings be accepted.

The value of knowing standard models became clear when I began my work on the cardiac contractile proteins, which are more difficult to prepare and less stable than their skeletal counterparts. After studying skeletal actin I published a series of reports on cardiac actin, then skeletal and cardiac tropomyosins, and finally skeletal and cardiac myosin that established my expertise on the heart’s contractile proteins. My first discovery occurred in 1963 when I was repeating classical studies using skeletal actomyosin reconstituted from highly purified actin and myosin. After learning that micromolar Ca$^{2+}$ activates muscle contraction I tried to stimulate my actomyosins with Ca$^{2+}$, but without success. One morning, while taking a shower, I thought of adding tropomyosin, which then had no known function. I did not find Ca$^{2+}$-sensitivity, but was overjoyed to discover that tropomyosin inhibited actin-myosin interactions. As I was starting to write up these findings I found to my horror that the inhibitory effect of tropomyosin had become stimulatory. After months trying to understand why the inhibition had disappeared I found that the man who washed our glassware had stopped flushing distilled water through the sintered glass funnels used in preparing myosin. Happily, when the funnels were properly rinsed, the inhibitory effect of tropomyosin returned. This suggested that heavy metals in Los Angeles tap water had catalyzed the oxidation of myosin sulfhydryl groups, so I blocked some of these sulphydryl groups and abolished the inhibitory effect. Although I had enough data for a full paper, I had lost almost a year, by which time others had begun to observe effects of tropomyosin.

After accepting my first faculty appointment in the Physiology Department at Columbia in 1964 I found that unlike my actomyosins, which used actins extracted at 0°C and were not Ca$^{2+}$-sensitive, actomyosins made with actins prepared at room temperature became Ca$^{2+}$-sensitive. The reason, I discovered, was that the latter contained a protein that I called “P2” because it was the second precipitate in an elaborate puri-
fication. At this time Setsuro Ebashi, using different methods, also isolated a protein, which he named “troponin”, that confers Ca\(^{2+}\) sensitivity to actomyosin. Because I was an unknown research fellow, while Ebashi was an established figure, he appropriately received credit for this discovery. I wonder, however, if I might have earned more recognition had I chosen a more mellifluous name than P2 for my Ca\(^{2+}\)-sensitizing protein!

Having decided it was time to move on I began studies of cardiac sarcoplasmic reticulum (SR), which I found transports Ca\(^{2+}\) fast enough and with sufficient affinity to relax the living heart. Because my training in physiology and bedside cardiology allowed me to relate my biochemical findings to the clinical regulation of cardiac performance, these studies made me an "expert" in academic cardiology.

Because I had made and recognized many errors in preparing these membranes I became an expert, which I define as someone who has made every mistake once – unlike a fool who repeats the same mistake again and again!

Two years later, in 1969, I accepted an endowed chair in Medicine and Cardiology at the Mount Sinai School of Medicine in New York where, during the early 1970s, my group described phospholamban. This discovery would not have been possible without three brilliant collaborators: Michihiko Tada who suggested that protein kinase A-catalyzed phosphorylation might stimulate Ca\(^{2+}\) uptake by cardiac SR, Doris Repke my master technician, and Madeleine Kirchberger. My key contribution, based on a lesson I had learned from Anfinsen, was to “poise” our reactions by measuring Ca\(^{2+}\) uptake at half-saturating Ca\(^{2+}\) concentration, others missed the effect of PK-A because they used saturating Ca\(^{2+}\), where stimulation is absent. Having learned the importance of naming things, we quickly named our discovery phospholamban.

In 1967 I joined the Department of Medicine at the University of Chicago where I tried to purify cardiac SR. I had little success, but did not lose my enthusiasm; after I summarized my progress at a national meeting a colleague told me: “Arnie, I have never heard a better presentation of such a small amount of new data.” I did gain an international reputation for my studies of cardiac SR, which is difficult to work with because these membranes are easily inactivated; for example by slow processing of the heart, dull homogenizer blades, and trace impurities which require that “house” distilled water be redistilled in glass. Because I had made and recognized many errors in preparing these membranes I became an expert, which I define as someone who has made every mistake once – unlike a fool who repeats the same mistake again and again!

In 1997 I began to reduce my administrative, research and clinical activities, which allowed more time to think and write about medical history, and more recently to complete the 5th Edition of my Physiology of the Heart. I still teach, in part because few medical educators today are trained in both clinical medicine and basic science, but largely because explaining things keeps my mind agile. I attribute much of my ability to remain active in the rapidly changing fields of cardiology to the intellectual flexibility provided by my liberal arts education and lifelong interest in history, literature and art. Most important has been my marriage to Phyllis, who provided a warm and intellectually rich environment that nurtured my creative abilities.

Arnold M. Katz, M.D.
Professor of Medicine Emeritus,
University of Connecticut School of Medicine,
Visiting Professor of Medicine and Physiology, Dartmouth Medical School,
Visiting Professor of Medicine, Harvard Medical School.

arnold.m.katz@dartmouth.edu

A curriculum vitae and video detailing events discussed in this article can be found on the American Physiological Society’s “Living History” website: http://www.the-aps.org/mm/Membership/Living-History/Katz
Dear Colleagues,

It is my honor to serve as the Chair of the XXI World Congress of the International Society for Heart Research, which will be held in San Diego, California, USA on June 30-July 4, 2013. With the meeting just a little over a year away, we have secured the waterfront San Diego Marriott Marquis & Marina as our hotel venue and wonderful space for the scientific sessions at the adjacent San Diego Convention Center. I have been working with the Congress Organizing Committee (Don Bers, Kirk Knowlton, Mark Mercola, Robert Mentzer, Daria Mochly-Rosen, Rick Moss, Paul Simpson, Mark Sussman, Yibin Wang, James Weiss) to develop the scientific themes of the meeting, to review symposia that will be sponsored by the International, Section and Local program committees, and to plan social events. I am also pleased that Yibin Wang has agreed to serve as my co-chair in the final stages of organizing and orchestrating the meeting.

We recognize that there are many scientific conferences to choose from in the cardiovascular field, many held in the summer months, thus, I hope you will consider just how unique this meeting is. The International Society for Heart Research convenes their World Congress just once every three years; all of the sections of the ISHR (Australasian, Chinese, European, Indian, Japanese, Latin American, and North American) contribute to this congress, insuring its truly international nature. The collegiality of the ISHR members and invitees is legend, having spawned innumerable collaborations, sabbaticals and friendships. Once you are hooked, you will become an integral member of the ISHR community that has already planned to again come together in Buenos Aires, Argentina (2016) and Beijing, China (2019). Please accept my cordial invitation to include the ISHR 2013 San Diego meeting on your calendar as a must-do event for next summer!

Concept and Main Theme

The World Congress is devoted to all aspects of cardiovascular research, providing an international forum for discussion of problems and controversies at the cutting edge of cardiovascular biology, pathophysiology and therapeutics.

The theme for the twenty-first Congress is *Unifying, Invigorating and Translating Cardiovascular Research* and we aim to weave these concepts through the program. *Unifying* current research findings and clarifying underlying controversies will be encouraged through the selection of...
speakers with differing opinions, as well as programmed opportunities to discuss issues in a range of topic areas. The Congress will focus on **invigorating** heart research by including symposia and speakers working on novel systems and technologies, and on emerging research areas that should inform and strengthen all of our research endeavors. We will also include several Bench-to-Bedside symposia to address the goal of **translating** research to improved cardiac health and treatment of cardiovascular diseases through drug discovery and the development of novel therapeutics, biomarkers and stem cell therapies.

Another key aspect of the meeting is the role it serves in recognizing excellence in science through awards and prizes, as described below.

**Organization of the Congress**

The meeting will take place over five days starting in the afternoon on Sunday, June 30 and continuing through the morning of Thursday, July 4 when there will be a half day program. July 4th is Independence Day, celebrated as a family holiday by many in the US, and we were aware of this along with other constraints in choosing the date for the meeting. We see this holiday overlap as an opportunity, however, because the hotel is a fantastic place to be for the traditional July 4th fireworks. San Diego is a vacation paradise, and conference participants and their families are welcome to stay on at the hotel through the holiday and weekend.

San Diego and other major cities on the west coast of the US (including Los Angeles, San Francisco, and Seattle) are home to a remarkable cadre of cardiovascular scientists, several of whom are participating in the Congress Organizing Committee and many of whom will soon be working with us as part of the Local Congress Scientific Program Committee.

As indicated above, in the years that the International Congress is held the program incorporates and encompasses the ISHR Section Meetings, thus regional as well as global issues are discussed in a single meeting. Symposia proposals have already been solicited by the ISHR and many of these have been selected by the ISHR Scientific Program Committee, chaired by Richard L. Moss, Ph.D, for sponsorship. The subject areas covered in these symposia are listed below and the proposed speakers and symposia chairs will soon be receiving invitations. The ISHR Sections will next be asked to identify or propose symposia they will sponsor, as will the Local Program Committee. Overall, we expect that there will be 36-42 total symposia, with up to 6 running concurrently during the meeting. Choosing what session you want to attend may be difficult as there will be many excellent talks, but the Convention Center conference rooms are very close together, making it easy to move between symposia for selected talks. Symposia will be held in the mornings and afternoons, and the poster sessions will be held during an extended lunch break to insure maximal time for scientific interaction.

**Scientific Program**

The Scientific Program will be composed of **Symposia, Nobel and Award Lectures, Young Investigator Award Competition, and daily poster sessions.**

- Principal themes of the meeting include Excitability and Pacemakers, Cardio-protection, Heart Failure, Signal Transduction, Cardiac Regeneration, and
Genomics/Proteomics. There will be approximately 40 symposia in these theme areas, as well as sub-themes such as oxidative stress and NO, calcium and contractile function, remodeling, metabolism, microRNAs, and stem cells.

The Nobel Lecture will be given by Dr Roger Tsien on Monday, July 1.

Because of the integration of Section Meetings into the World Congress, participating Sections will have opportunities to hold business meetings and other Section activities, including a scientific session in their own language if they so desire. As noted above, several Section-sponsored Symposia will be interspersed throughout and seamlessly integrated into the planned scientific program.

Poster sessions will be given prominence in terms of space and time and will form an important focal point of the Congress. Early-career investigators can compete for ISHR International Poster Awards for outstanding scientific research. Winners will receive a certificate, a US$300 prize and a ribbon to be displayed on the poster during the meeting.

The Early Career Investigator Network will stage a pre-Congress ECI event (held on the morning of the first day of the Congress), to be organized by the ECI Network and include scientific symposia and a Career Development workshop.

Social Program and Excursions

The social program and excursions in conjunction with the ISHR World Congress will be rich and varied, with activities that are appropriate for attendees and for their families or guests.

On the first evening, a Welcome Reception will provide an opportunity to renew old friendships and make new acquaintances with delegates from all over the world.

The Fellows of the International Society for Heart Research (FISHR) will have a special dinner sponsored by the ISHR at a venue on the San Diego waterfront on Tuesday, July 2.

The closing banquet on July 3 is open to all participants and is being planned for an outdoor park adjacent to the San Diego Marriott Marquis & Marina.
For conferees and their families, there is a planned excursion to the world-famous San Diego Zoo and historic Balboa Park.

Entertainment opportunities within walking distance of the hotel include a Padres baseball game (if they are in town!), strolling through Seaport Village, shopping at Horton Plaza or visiting the USS Midway museum.

Additional attractions in the area include SeaWorld, Legoland, hiking in Torrey Pines State Reserve, kayaking in La Jolla or a visit to the historic Hotel Del Coronado.

**Travel Awards**

We are pleased to announce that the ISHR will provide travel awards for more than 100 young investigators from all participating ISHR Sections, with the award recipients to be selected by the leadership of the relevant Section. Travel support is intended to facilitate participation of early career investigators in the World Congress by reducing the net cost of attending the meeting.

**Registration and Abstract Submission**

Meeting registration and hotel reservations will be available online – our plan is to activate the website in late May, 2012. Please visit the Congress home page (www.ishr2013.com) regularly for updated information, particularly the format and deadline for abstract submission.

**Venue of the Congress**

A block of rooms has been reserved at The San Diego Marriott Marquis & Marina, a premier San Diego hotel. We have negotiated a good room rate as part of our package and expect that all the conferees will stay at the hotel. The San Diego Marriott Marquis & Marina is adjacent to the San Diego Convention Center (SDCC) providing effortless travel to and from the Congress scientific sessions. The best news of all is that because of the SDCC’s convenient location, pedestrian friendly neighborhoods and near-perfect climate, you should not need a rental car if you stay at the Marriott Marquis & Marina.

Located on the harbor in downtown San Diego, the San Diego Convention Center is the premier meeting and conference facility for the region. The SDCC is located on sparkling San Diego Bay in the heart of a vibrant downtown and offers all the modern amenities while promoting a GREEN conference environment.

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We expect to have approximately 1,000 participants from all over the world. Please be one of them and share in the excitement and success of the 2013 World Congress in San Diego!

Joan Heller Brown, Ph.D.
Professor and Chair
Department of Pharmacology
9500 Gilman Drive, MC 0636
University of California, San Diego
La Jolla, CA 92093-0636

Two mile long bridge over San Diego Bay to Coronado.
Heart Failure after Disaster in Japan

Time has passed quickly – one year has gone by since the disaster in Tohoku, Japan. Restoration of the damaged area has not progressed as expected, and most of the tremendous amount of rubble still has not been removed since the affected area is too large and some areas are contaminated by radioactivity. More than 300,000 people remain in temporary housing. A preliminary regional health care survey of cardiovascular diseases reported that acute myocardial infarction transiently increased during the weeks immediately following the disaster, whereas chronic heart failure (CHF) is steadily increasing even after several months. An increase in acute coronary syndrome (ACS) was also seen following the Kobe quake seventeen years ago; physical and emotional stress in uncomfortable conditions may cause plaque rupture in the coronary arteries leading to the onset of ACS. An increase in CHF and exacerbation of the symptoms may be attributable to sympathetic stimulation due to emotional stress, a lack of good sleep, excessive salt intake (8-16g/day) due to a diet dependent on fast food, and disrupted drug adherence for treatment of hypertension or CHF.

It is well known that neurohormonal activation is a pivotal cause of CHF deterioration. Excessive salt intake also activates ROS generation through the RAAS-NFκB signaling pathway and enhances cytokine production which causes cellular injuries in the heart, vessels and kidneys. Long-term expression of cytokines may produce maladaptive effects. Renin-Ang II, TNFα, TLR-NFκB are not merely markers of myocardial injury, but actively induce myocardial dysfunction which promotes apoptosis and cardiac fibrosis. RAAS activation is also suggested to be involved in the immune reaction.

In innate immunity, the repetitive molecular structure of the pathogen is recognized by the macrophage or monocyte as PAMPs (pathogen-associated molecular patterns) via pattern recognition receptors (PRPs). Upon recognition of PAMPs, toll-like receptors (TLR-4) activate several signaling molecules in which NFκB plays a key role in the inflammatory response to destroy the pathogen and infected cells. Injured or dying cells release endogenous molecules called DAMPs (damage/danger-associated molecular patterns) which also activate the immune system in a fashion similar to PAMPs. A variety of different molecules are recognized as DAMPs, and this chronic inflammation process is activated under sterile conditions by endogenous molecules. My colleagues recently reported that mitochondrial DNA can also activate the inflammatory response via TLR-9 since the unmethylated CpG motif in mitochondrial DNA resembles the PAMPs recognition pattern. During chronic inflammation in which TLRs are activated with these endogenous molecules, high levels of DAMPs are generated and pro-inflammatory DAMPs create more tissue damage which amplifies the tissue levels of DAMPs ad infinitum, resulting in a vicious cycle of tissue damage and heart failure.

After the disaster, a number of people suffered from respiratory infectious diseases; bronchitis and pneumonia were caused by the chilly climate and the dust-polluted air derived from dried sludge. Under these conditions, pro-inflammatory responses are augmented and the immune response is activated. The TLR-4 pathway in immune cells and myocardium may play an important role in cardiac dysfunction via expression of TNFα, IL-1β and IL-6 in the heart, as a recent study demonstrated that the TLR-4 pathway is activated in the heart after lipopolysaccharide (LPS) stimulation.

Taken together, neurohormonal activation, excessive salt intake and concomitant respiratory infections may trigger and enhance chronic inflammation in the heart in patients with CHF in the affected region. DAMPs are usually sequestered by the protease-ubiquitin system and autophagy, however, if accumulation of DAMPs exceeds the sequestration capacity, they induce cardiac injuries causing heart failure. Similarly, the heap of rubble which has not yet been removed one year after the disaster could cause economical failure in the Tohoku area.

Masatsugu Hori, M.D., Ph.D.
President ISHR
Heart disease remains the most common cause of death and disability in our society, and yet the face of heart disease has changed dramatically in the decades since cardiovascular scientists first became able to examine the cellular and molecular mechanisms of cardiac function. In the 1940’s, death was a coin toss for sufferers of myocardial infarction, and survivors were effectively removed from the workforce. Today greater than 9 in 10 patients hospitalized with heart attacks survive to go home and most of those resume normal activities within weeks if not days.

Yet deaths from cardiovascular diseases remain unacceptably common as heart disease has transitioned away from acute crisis to chronic debilitation. As the incidence rates for myocardial infarctions have decreased and the need for acute interventions with stents and other procedures has fallen, the prevalence of chronic heart failure has risen and is increasingly becoming the face of heart disease. It is remarkable that no new effective pharmaceutical therapies for heart failure have been developed in the past two decades. This suggests more than anything a failure to think beyond the old ideas of inotropic dysfunction and neurohumoral imbalance among the critical common factors in the pathophysiology of chronic left ventricular dysfunction and heart failure. The increasingly appreciated consequences of proteotoxicity on cardiac function represent one such new idea.

Protein Homeostasis
The abundance of proteins within a cell is determined by regulating both the synthesis and the turnover of proteins. Protein synthesis is triggered by specific signaling cascades, many of which lie downstream of the master metabolic regulator mTOR, allowing tight coupling of amino acid availability and new protein synthesis. These signaling events coordinate the numerous aspects of ribosomal assembly and function that are required for protein translation. Protein turnover is regulated via two major systems: the ubiquitin-proteasome system (UPS) and autophagy. Autophagy is a relatively non-selective catabolic process for triggering protein degradation in the setting of nutrient deprivation or stress utilizing the lysosomal machinery. In contrast, the UPS is a highly specific mechanism for removing specific proteins that are no longer needed by the cell. The UPS employs a series of enzymes to tag proteins with polyubiquitin chains that serve as a targeting signal for protein degradation via a large catalytic protease called the proteasome. The rate limiting enzymes in this process are ubiquitin ligases, which serve dual purposes by recognizing proteins that require degradation via specific protein-protein interactions and catalyzing the final step in ubiquitin transfer and chain assembly to the substrate protein. Because they are selective, cells contain hundreds of ubiquitin ligases that activate protein degradation programs under specific conditions to modulate the abundance of proteins within cells as conditions vary.

Proteins are removed from a cell for a variety of reasons. The proteins may no longer be needed, or may be toxic to the cell and thus require maintenance at low abundance. However, proteins may also require elimination if they become damaged, misfolded, or are otherwise rendered nonfunctional. Removing such proteins is essential not only because they are non-functional but also because their aberrant conformations may lead to gain-of-function effects that can be highly toxic to a cell. The process of recognizing and removing aberrant protein conformations is referred to as protein quality control (PQC). The PQC system recognizes dysfunctionally structured proteins and activates processes that lead to one of several outcomes. Denatured proteins may be refolded back into their native and active conformations via molecular chaperones, they may be targeted for degradation by the UPS, or they may be sequestered if refolding or degradation fails such that they are rendered non-toxic.

Mechanisms of Regulation of Homeostasis in the Heart
Cardiomyocytes have both general and specific machinery for modulating protein synthesis and degradation. Protein synthesis within the heart is triggered by mechanical forces and signaling events that stimulate cardiac enlargement;
conversely, processes that elicit atrophy of the heart (such as unloading and cancer) suppress protein synthesis. Much of this activity appears to be determined by a regulatory mechanism that involves ubiquitin ligases that are enriched in the heart. The MuRF (muscle ring finger) family of proteins regulates sarcomere quality control and have additional functions within the nucleus to regulate the abundance of proteins in cardiomyocytes as hemodynamic conditions change. Another ubiquitin ligase, atrogin (also known as MafBx), regulates signaling pathways that influence protein synthesis responses in cardiomyocytes. The existence of cardiac-specific responses to stimuli that require protein turnover in the heart suggests that precise interventions are possible to affect PQC within the heart without having an impact on the essential ongoing PQC processes in non-cardiac cells.

Evidence for Proteotoxicity as a Contributing Factor in Heart Failure

The toxic effects of misfolded proteins are well described in chronic degenerative diseases of the brain and other organs. Although there is argument about how misfolded proteins elicit their toxicity, there is little argument that defects in PQC are the proximate mediators of Alzheimer’s disease, Parkinson’s syndrome, and amyotrophic lateral sclerosis. Genetic evidence has linked defective PQC with cardiac dysfunction in uncommon cardiac maladies for decades, but the idea that this might be a general mechanism for cardiac dysfunction has only recently received attention. The development of antibodies that specifically recognize misfolded protein conformations has permitted the discovery of a PQC defect in failing human hearts that leads to the accumulation of toxic misfolded proteins, suggesting that proteotoxicity may contribute to the etiology of heart failure.

To test whether this is indeed the case, transgenic rodent models have been created that allow the temporal control of PQC via regulated expression of conformationally defective protein. The results of these experiments have shown that increasing the abundance of misfolded proteins elicits a proteotoxic response and also causes cardiac decompensation. Remarkably, reversing the accumulation of misfolded proteins also reverts cardiac function back to normal. These observations suggest that proteotoxicity may be a critical component of ventricular dysfunction in the setting of heart failure, and thus might be a therapeutic target that has so far been missed by current pharmacologic strategies.

Therapeutic Implications

In spite of the large increase in the number of patients with chronic congestive heart failure, there have been dreadfully few innovations in pharmacologic approaches to treat this condition. The discovery that proteotoxicity is a contributing factor to left ventricular dysfunction and that reversing proteotoxic effects can lead to improvement in cardiac performance suggests that therapies aimed at reducing the accumulation of misfolded proteins or diminishing their toxic effects may be salutary in the setting of congestive heart failure. A recent screen in motor neurons identified 30 compounds that ameliorated proteotoxic effects. Similar assays may be useful to develop therapies that arrest or reverse the consequences of proteotoxicity within the heart. More broadly, it is critical that we raise awareness that wear and tear on the heart due to protein misfolding is a major and targetable contributor to cardiac dysfunction.

References

8. Cam Patterson, M.D., M.B.A., F.I.S.H.R.
University of North Carolina at Chapel Hill, NC

We are pleased to present this article by Dr Cam Patterson, as the fifth in our series of invited articles written by members of the ISHR International Council. Each member of Council submits one article for publication during his/her 6-year tenure. The content of the article is at the discretion of the author, but should be of broad interest to members of the Society and stimulate constructive debate on important scientific and philosophical issues.
Meet the Future of the ISHR-Japanese Section

Katsuhito Fujiu, M.D., Ph.D.
Winner of the 2011 ISHR-JPN Young Investigator Award – Gold Medal
The 28th Annual Meeting of the ISHR-Japanese Section
December 2-3, 2011, Tokyo, Japan

Dr Fujiu is an assistant professor in the Department of Cardiovascular Medicine at the University of Tokyo. His research interests include chronic inflammation in cardiovascular disease. Recently, his research has focused on the effect of cell-cell and organ-organ communications on the development of heart failure.

Yasutomi Higashikuni, M.D., Ph.D.
Winner of the 2011 ISHR-JPN Young Investigator Award – Silver Medal
The 28th Annual Meeting of the ISHR-Japanese Section
December 2-3, 2011, Tokyo, Japan

Dr Higashikuni is an Assistant Professor in the Department of Cardiovascular Medicine at the University of Tokyo. His research aims to provide new therapeutic targets for heart disease and to develop novel therapeutics. His major research interest is in cellular interaction in the pathophysiology of cardiac remodeling and heart failure.

Satoshi Somekawa, M.D., Ph.D.
Winner of the 2011 ISHR-JPN Young Investigator Award – Silver Medal
The 28th Annual Meeting of the ISHR-Japanese Section
December 2-3, 2011, Tokyo, Japan

Dr Somekawa is a cardiologist at Nara Medical University. His research focuses on the novel functional molecules involved in vascular development and functional regulation. He identified Tmem100 (transmembrane protein 100) as a new endothelium-specific gene regulated by the bone morphogenetic protein 9 (BMP9)/BMP10 and its endothelial receptor, ALK1. This signaling pathway is implicated in the etiologies of human diseases, and his studies may provide a foundation to develop novel therapeutic approaches against these cardiovascular diseases.
ISHR-Japanese Section Poster Award Winners

Ken Shinmura, MD, PhD, FAHA *(Right)*
Winner of the 2011 ISHR-Japanese Section Best Poster Award
The 28th Annual Meeting of the ISHR-Japanese Section (Dec 2-3, 2011), Tokyo, Japan

Dr Shinmura is an Assistant Professor in the Department of Internal Medicine at Keio University School of Medicine. This research focused on the protective role of the transsulfuration pathway against myocardial ischemia/reperfusion. Dr Shintaro Nakano *(Left)*, ex-Project Instructor in the Department of Biochemistry and Integrated Medical Biology at Keio University School of Medicine, mainly performed this experiment.

Yosuke Omori, MD
Winner of the 2011 ISHR-Japanese Section Best Poster Award
The 28th Annual Meeting of the ISHR-Japanese Section December 2-3, 2011, Tokyo, Japan

Dr Omori belongs to Department of Cardiovascular Medicine, Osaka University Graduate School of Medicine, and is in his final year of graduate school. His research interest is the etiology of, and therapy for, heart failure. His recent studies focus on the preservation of ejection fraction (HFpEF) using HFpEF model rats, cultured cardiac fibroblasts and clinical data.

Susumu Hosokawa
Winner of the 2011 ISHR-Japanese Section Best Poster Award
The 28th Annual Meeting of the ISHR-Japanese Section December 2-3, 2011, Tokyo, Japan

Mr Hosokawa is a second year PhD student supervised by Dr Mitsuaki Isobe in the Department of Cardiovascular Medicine at Tokyo Medical and Dental University in Tokyo. His research focuses on the investigation of pathogenesis of pulmonary hypertension from the viewpoint of inflammation. Specifically, he has studied the role of NF-kB signaling in pulmonary hypertension.
ISHR-ECI:
A Network for ISHR Early-Career Investigators

At the World Congress in Kyoto (May 2010), the ISHR Council commissioned a Working Group (Lea Delbridge, David Eisner, Jennifer Van Eyk) to invite Early Career Investigators (ECI’s) to make proposals about how to develop the Section and Congress meetings to be as attractive as possible for ECI’s, and to expand the role of ECI’s in the ISHR. Bringing together ECI’s from across the different Sections of the ISHR, an ECI Network was set up to address these issues and advance suggestions.

Network representatives include:

**Australasian**
Jim Bell  
Helena Viola  
University of Melbourne, Australia  
University of Western Australia, Australia

**European**
Francesca Rochais  
Sarah Briston  
Jessica Clarke  
Institute of Developmental Biology  
University of Manchester, UK  
Marseille-Luminy (IBDML), Université de la Méditerranée, France

**North American**
Grant Budas  
Christopher Murray  
Jeff Erickson  
Eva van Rooij  
Gilead Sciences, USA  
Johns Hopkins Medicine, USA  
University of California, Davis, USA  
miRagen Therapeutics, USA

**Chinese**
Lemin Zheng  
Peking University Health Sciences Center, China

**Japanese**
Atsuhiko Naito  
Osaka University, Japan

**Latin American**
Carlos Valverde  
Emiliano Raúl Diez  
Centro de Investigaciones Cardiovasculares, Conicet La Plata, Argentina  
IMBECU CONICET, Mendoza, Argentina

All Section representatives have provided enthusiastic and insightful suggestions for future ECI schemes. Five core initiatives were presented by the Working Group to the ISHR Council in November for consideration. It has been particularly gratifying.

Drs John Solaro and Roberta Gottlieb talk to early career investigators about a strategic approach to publishing papers and navigating the journal review process at the ECI Workshop (34th ISHR-AUS Section Meeting, Aug 5-8, 2010, Adelaide, AUS).
that the Council has made a commitment to support all of the proposed activities. Briefly, these initiatives are to:

1. Organise a pre-World Congress ECI event
2. Create an ECI-focused webpage on the ISHR world website
3. Deliver ECI-specific articles for publication as a column in the ISHR newsletter, Heart News and Views
4. Establish a program for ECI pre/post Congress visits to local laboratories, funded by ISHR ECI Training Bursary awards
5. Develop a local ECI contact list, to provide general ECI information directly to Section members

The developments related to the World Congress are particularly exciting. Numerous ECI activities have been happening at Section meetings and during the last couple of World Congresses, typically in the form of one-hour career workshops. The proposed pre-World Congress ECI event in San Diego (#1 above) is set to build on these foundations. The idea is to offer an extended forum to provide career training, foster scientific debate amongst ECI colleagues in a relatively informal setting, and create ECI links early in the Congress that can develop as the meeting proceeds and beyond.

Further scientific/career development will be promoted through the awarding of 5 Congress ECI Training Bursaries. These awards will subsidise pre/post-Congress visits to ‘local’ Section labs to train in a new technique/method (#4). More details on these schemes will be made available prior to the World Congress in June 2013. Indeed, the establishment of the ECI-focused webpages/newsletters and local contact lists (#2, 3, 5) will provide multiple media for communicating ECI news, issues and events, including the advertisement of World Congress initiatives. Stay tuned for news about these developments.

Clearly, the enthusiastic support of the ISHR Council for ECI investment is a key element in providing opportunity for ECI mentoring and training at a critical career stage. The emphasis is now on the ECI’s to pro-actively engage in these schemes and extract as much benefit as possible. ECI engagement starts at the local Section level, where programs can be initiated and schemes developed – and hopefully incorporated across all Sections to benefit all Society ECI members. If you are interested in being pro-actively involved in initiatives/events or have ideas on how to enhance the ISHR ECI ‘experience’, please contact your local Section representatives from the list above.

Looking forward to some exciting early career investigator collegiality at San Diego 2013.

Jim Bell
Melbourne, Australia
ISHR MEETINGS CALENDAR

- **August 16-19, 2012.**  **XXXVI Annual Meeting of the Australasian Section.** Brisbane Convention Center, Brisbane, Australia.
  
  Website: [www.csanz2012.com](http://www.csanz2012.com)

- **October 25-26, 2012.**  **XIX Annual Meeting of the Latin American Section.** Santiago, Chile. **Inquiries:** Dr Paulina Donoso, President ISHR-LAT ([pdonoso@med.uchile.cl](mailto:pdonoso@med.uchile.cl))

- **October 26-27, 2012.**  **XXIX Annual Meeting of the Japanese Section.** Fukuoka, Japan. **Inquiries:** Dr Kenji Sunagawa ([sunagawa@cardiol.med.kyushu-u.ac.jp](mailto:sunagawa@cardiol.med.kyushu-u.ac.jp))

- **June 30 - July 4, 2013.**  **XXI World Congress of the ISHR.** San Diego, California. See page 4.

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FELLOWSHIP OF THE ISHR

The International Society for Heart Research invites you to nominate candidates for membership in the Fellowship of the ISHR.

**History of the Fellowship**

The ISHR established the Fellowship of the ISHR as a means of recognizing those members who have distinguished themselves for outstanding contributions to cardiovascular research. Fellows are selected solely on the basis of scientific excellence, as evidenced by an established track record of publications in high-impact journals. The Society looks to its Fellows for leadership and guidance in its various activities; including the organization of topical meetings and selection of recipients for Society awards.

**Fellowship Benefits**

- Fellows will be entitled to add “FISHR” to their degrees and distinctions.
- Fellows will be given free registration at World Congresses.
- The names of new Fellows will be published in the *JMCC* and *Heart News and Views*.
- Fellows will receive a Fellowship certificate.

**Nomination Procedure**

The name and institute of each nominee and a brief (one page only) letter of nomination outlining his/her major contributions to cardiovascular science should be sent by email ([llobaugh@nc.rr.com](mailto:llobaugh@nc.rr.com)) to Dr Leslie Anderson Lobaugh, Executive Secretary – ISHR.

Details of the nomination and application procedures can be found at [www.ishrworld.org](http://www.ishrworld.org). Click on **Fellows** in the righthand column.

The deadline for receipt of nominations is 30th September 2012. Nominations received after that date cannot be considered.
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Dr Irina Elyubaeva - Servier International
35 rue de Verdun - 92284 Suresnes Cedex - France
or webmaster@servier.com

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Editor
L. Anderson Lobaugh
Durham, NC, USA
E-mail llobaugh@nc.rr.com

Founding Editor
T.J.C. Ruigrok
Wijk bij Duurstede, The Netherlands
E-mail t.j.c.ruigrok@xs4all.nl

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London, UK
K.T. Weber
Memphis, TN, USA

Editorial Office
3711 Lochn’ora Parkway
Durham, NC 27705
USA.
Phone/Fax: +1 919 493 4418