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### The Cardiovascular Forum for Promoting Centers of Excellence and Young Investigators

**August 15-17, 2013**  
**at**  
**The Galt House Hotel**  
**Louisville, Kentucky**

**Sponsored by**  
International Academy of Cardiovascular Sciences – American Section

**Planned Symposia:**  
- **Autophagy and Cardiac Cell Death; MicroRNA and Cardiac Remodeling; Stem Cells and Cardiac Regeneration; Diabetes and Metabolic Syndrome**

First Announcement with Forum information and links to conference registration, *housing and submission of abstracts is posted on the International Academy of Cardiovascular Sciences webpage: http://www.heartconference.com or contact Irving G. Joshua, Ph.D. or Suresh C. Tyagi, Ph.D. at (812) 852-5371

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**EDITORIAL OFFICE:** Ivan Berkowitz, Editor and Heart Health Scholar, Institute of Cardiovascular Sciences, St Boniface General Hospital Research Centre, Faculty of Medicine, University of Manitoba, 3021/1 351 Taché Avenue, Winnipeg, Manitoba R2H 2A6 Canada. Tel: (204) 228-3193, Fax: (204) 233-6723; E-mail the Editor: ivan@mts.net • Academy website: www.heartacademy.org
Abstract submission Deadline - **July 1, 2013.**

Deadline for submissions for both James Willerson Clinical Award Competition for Residents and Fellows, and Grant Pierce Biomedical Award Competition for Graduate Students and Postdoctoral Fellows **July 1, 2013.**

This will be your final opportunity to submit an abstract for an oral or poster presentation and contribute to the scientific program that will highlight state of the art approaches to cardiovascular research.

Submit your abstract today at:

[http://louisville.edu/conferenceservices/events/abstract/](http://louisville.edu/conferenceservices/events/abstract/)

and register for the meeting at:


Special Rates on Hotel accommodations can be obtained by contacting the Galt House by calling the Galt House Central Reservations Office at 1-800-843-4258 and indicate you are attending this Cardiovascular Forum.

**To get the discounted room rates you must register for your room by JULY 15th, 2013.**

For complete details regarding the Forum please go to our website at: [http://louisville.edu/medschool/physiology/the-cardiovascular-forum.html](http://louisville.edu/medschool/physiology/the-cardiovascular-forum.html)
Preliminary Conference Program

August 15, 2013 - Day 1:

7:00 PM to 10:00 PM – Registration and Welcome Reception

August 16, 2013 - Day 2:

8:30 AM to 10:30 AM

Scientific Symposium: MicroRNA and Cardiac Remodeling (Co-Chair – Suresh Tyagi and Jeff Falcone)

Mechanism of Remodeling in Heart Failure

Suresh C. Tyagi, Ph.D., University of Louisville, Louisville, KY

Molecular Phenotype of Human Right Heart Failure

Hari S. Sharma, Ph.D., VU University Medical Center, Amsterdam, The Netherlands

Cytokines, Innate Signaling and Heart Failure

Dr. Pawan K. Singal, University of Manitoba, Winnipeg, Manitoba, Canada R2H 2A6

Experimental Studies on Association Between MicroRNAs and Cardiac Remodeling in Subjects Under Both Diabetes and Hypoxia

Dr. Belma Turan, Ankara University Faculty of Medicine, Sihhiye / ANKARA

Regenerative Angiogenesis Strategies

James B. Hoying, Ph.D., Professor, Division of Cardiovascular Therapeutics and Department of Surgery, University of Louisville, Louisville, KY

James Willerson Clinical Award Competition for Residents and Fellows (Chair - Roberto Bolli)

(Participants to be selected from abstract competition)

10:30-11:00 Coffee break

11:00 AM to 12:00 Noon

Frontiers in Cardiovascular Science (Chair – Rakesh Kukreja and David Lominadze)

Hypoxia and the Developing Heart

Dr. Bohuslav Ostadal, Czech Academy of Sciences, Prague, Czech Republic

The Control of Hypertension: Is it Time for New Approaches

Dr. Grant N. Pierce, St. Boniface Hospital Research, Winnipeg, Canada.
Clinical Genomics of Sudden Cardiac Death

Dr. Sumeet Chugh, Cardiac Electrophysiology Research Institute, Los Angeles, CA, USA

Frontiers in Cardiac Research – Forum A (Chair – Dinender Singla)

Rescue of Diabetes-related Impairment of Myocardial Angiogenesis: Potential and Challenges

Nilanjana Maulik, Ph.D., University of Connecticut Health Science Center, Farmington, CT

Factor(s) Released from Stem Cells Polarize Infiltrated Monocytes into M2 Macrophages and Provide Cardiac Protection in Diabetic Cardiomyopathy

Dinender Singla, Ph.D., Biomolecular Science Center, University of Central Florida, Orlando, FL

Coronary Artery Bypass Graft: Why is the Saphenous Vein Prone to Intimal Hyperplasia?

Devendra. K. Agarwal, Ph.D., Department of Biomedical Sciences, Creighton University, Omaha, NE

12:00 Noon to 1:00 PM - Lunch and Networking

1:00 PM to 3:30 PM

Scientific Symposium: Stem Cells and Cardiac Regeneration (Chair – Roberto Bolli and James Willerson)

Use of Stem Cells for the Treatment of Ischemic Cardiomyopathy

Roberto Bolli, M.D., Div. of Cardiovascular Medicine, University of Louisville, Louisville, KY

Update on the Use of Stem Cells to Treat Cardiovascular Disease in Humans

James T. Willerson, M.D., Texas Heart Institute, University of Texas Health Science Center, Houston, TX 77030, USA

The Microcirculation as a Therapeutic Target in Ischemic Tissue Repair

Douglas W Losordo, M.D., Department of Medicine, Northwestern University, Chicago, IL

The Human Lung: A Self Renewing Organ

Piero Anversa, M.D., Department of Anesthesia, Brigham and Women’s Hospital, Harvard Medical School, Boston, MA

Interleukin-10 Enhances Endothelial Progenitor Cell-mediated Cardiac Repair

Raj Kishore, Ph.D., Departments of Medicine and Molecular Pharmacology and Biological Chemistry, Northwestern University, Chicago IL

Grant Pierce Biomedical Award Competition for Graduate Students and Postdoctoral Fellows (Chair – Irving G. Joshua)

(Participants to be selected from abstract competition)
3:30 PM to 5:30 PM  - Poster Mentoring and Wine & Cheese
7:00 PM to 10:00 PM  - Networking and Informal Dinner

August 17, 2013 - Day 3:
8:30 AM to 10:30 AM
Scientific Symposium: Autophagy and Cardiac Cell Death (Chair – Steven Jones and Nilanjana Maulik)

Hexosamine Biosynthetic Pathway in Cardiovascular Disease
Steven P. Jones, Ph.D., Department of Medicine, University of Louisville, Louisville, KY

Translational Bioenergetics; Implications for cardiovascular research
Victor Darley-Usmar, Ph.D., Department of Pathology, University of Alabama-Birmingham, Birmingham, AL

Nitric Oxide Signaling and Cardiac Cell Death in Heart Failure
David J. Lefer, Ph.D., Department of Surgery, Emory University School of Medicine, Atlanta, GA

Mitochondrial Dynamics, Myocyte Viability and Heart Failure
Kenneth Walsh, Ph.D., Department of Medicine, Boston University, Boston, MA

Molecular Regulation of Autophagy and Cell Death Signaling Pathways in the Heart
Lorrie A. Kirshenbaum, Ph.D., University of Manitoba, Departments of Physiology and Pharmacology, & Therapeutics, Winnipeg, Manitoba, Canada)

Eric Olson Orations in Cardiovascular Science (Chair – Dennis McNamara)

Granzyme B in Vascular Injury, Inflammation and Repair
David Granville, Ph.D., UBC James Hogg Research Centre, St. Paul’s Hospital, Vancouver, BC, Canada

Hydrogen Sulfide/MicroRNA-21 Partnership for Cardioprotection
Fadi N. Salloum, Ph.D., Department of Medicine and Physiology, Virginia Commonwealth University, Richmond, VA

MLIP, A Novel Modulator of Heart Growth
Dr. Patrick Burgon, Professor of Medicine, University of Ottawa Heart Institute, Ottawa, Canada

Scleraxis: A New Therapeutic Target in Cardiac Fibrosis
Michael Czubryt, Ph.D., St. Boniface Hospital Research, Winnipeg, Canada
miR-133 in the Diabetic Heart

Paras Mishra, Ph.D., Department of Physiology and Biophysics, University of Louisville, Louisville, KY

10:30-11:00 Coffee break

11:00 AM to 12:00 Noon

Frontiers in Cardiovascular Medicine (Chair – Bohuslav Ostadal and Ashok Kumar)

LOX-1 in Mitochondrial DNA Damage, Autophagy and Inflammation.

Dr. Jawahar L. Mehta, University of Arkansas for Medical Sciences, Little Rock, AR 72205, USA

Cellular Time: Practical Insights into the 4th Dimension of Cardiovascular Health and Disease.

Michael J. Sole M.D. University of Toronto, Toronto, Ontario, Canada

Bone Marrow Stem Cell Transplantation for Cardiac Repair

Muhammad Ashraf, Ph.D., Department of Pathology and Laboratory Medicine, University of Cincinnati, Cincinnati, OH

Frontiers in Cardiac Research – Forum B (Chair – Hani Sabbah)

Abnormalities of Mitochondria in Heart Failure: Important Contributors to Disease Progression

Hani N. Sabbah, Ph.D., Cardiovascular Research, Henry Ford Hospital, Detroit, MI,

Role of MicroRNA in Cardioprotection

Rakesh C. Kukreja, Ph.D., Division of Cardiology, Virginia Commonwealth University, Richmond, VA

Taurine: Regulator of Mitochondrial Function

Stephen W. Schaffer, Ph.D., Department of Pharmacology, University of South Alabama, Mobile, AL

12:00 Noon to 1:00 PM - Lunch and Networking

1:00 PM to 3:30 PM

Scientific Symposium: Diabetes and Metabolic Syndrome (Co-Chair – Aruni Bhatnagar and Steven Schaffer)

Role of the Polyol Pathway in Diet-induced Obesity

Aruni Bhatnagar, Ph.D., Division of Cardiovascular Medicine, University of Louisville, Louisville, KY

Vascular Endothelium as a Window into Cardiovascular Health: Focus on Diet and Exercise

David D. Guttermann, M.D., Departments of Medicine and Physiology, Medical College of Wisconsin, Milwaukee, WI

Mitochondrial Proteome Regulation in the Diabetic Heart
Targeting AMPK as a therapy for diabetic cardiovascular complications

Ming-Hui Zou, Ph.D., Department of Biochemistry & Molecular Biology, College of Medicine, The University of Oklahoma Health Sciences Center, Oklahoma City, OK 73104

"reciprocal relationships between endothelial dysfunction and insulin resistance: therapeutic implications"
Michael J. Quon, M.D., Ph.D., Department of Medicine, University of Maryland School of Medicine, Baltimore, MD

Kern Wildenthal Orations in Cardiovascular Medicine (Chair - Naranjan Dhall)

Proteotoxicity in Viral Cardiomyopathy

Honglin Luo, M.D., Department of Pathology & Laboratory Medicine, UBC James Hogg Research Centre, St. Paul's Hospital, Vancouver, BC, Canada

Human Recombinant ACE2 as Therapy for Cardiovascular Diseases: From Bench to Bedside

Gavin Oudit, M.D., Ph.D., Department of Medicine, University of Alberta, Edmonton, AB, Canada

The Art of Preventing Broken Hearts in Breast Cancer

Davinder Jassal, M.D., St. Boniface Hospital Research, Winnipeg, MB, Canada

Epigenetics and renovascular remodeling in hyperhomocysteinemia

Pushpakumar, M.D., Ph.D., Department of Physiology & Biophysics, University of Louisville, Louisville, KY

Role of Micro RNA 499 During Myocardial Infarction

Srikanth Givvimani, M.D., Ph.D., Department of Physiology & Biophysics, University of Louisville, Louisville, KY

3:30 PM to 5:30 PM - Poster Mentoring and Wine & Cheese

7:00 PM to 10:00 PM - Reception, Banquet and Awards
Dr. Robert Roberts: Recipient of the Canadian Cardiovascular Society Research Achievement Award in 2012

Response by Dr. Roberts to presentation of the award

It is a great honour to receive such an award from the Canadian Cardiovascular Society and I am humbled to be among the many outstanding Canadian recipients of this award, which has special significance to me, as I spent a large part of my professional career in the USA. This award, from the prestigious Canadian Cardiovascular Society has a nostalgic fulfillment for me, since it is to Canada I owe my up-bringing, education and training. In fact, most of my early mentors were here, in Toronto, at the University. No award is simply the work of one individual, and this award is no exception. I have many mentors to thank and in particular, I wish to thank Dr. Robert Anderson, Dr. Don Beanlands, Dr. Eugene Braunwald, Dr. Burton E. Sobel and Dr. Antonio M. Gatto. I wish to also thank the hundreds of Fellows with whom I had the honour to rub shoulders throughout my career - not only did the work, but they were always a great source of inspiration and knowledge. Last but
not least, my family, my wife Donna, without whom none of this would have been possible, and my two children Brandon and Alison, who have always been my greatest fans.

For many people, looking back on their career, there are often triggering events, or it was what they always wanted to do. Having grown-up in a small town, for me, a physician was someone who had a black bag and came around and made house calls. My decision to train as an academicians and investigator was without design or prior knowledge. While finishing my Assistant Chief Residency at the Toronto General Hospital, Dr. Robert Young, a Cardiologist in the Roberts Clinic (no relationship) at St. John’s, Newfoundyland called to ask if I would join the Clinic, which was quite famous in St. John’s. It was at this point that I re-read the “The Road Not Taken” by Robert Frost, and on the advice and recommendation of Drs. Don Beanlands and Susan Lenkei (my mentors), I decided to pursue further training. With their support, I secured an Canadian Heart Foundation Scholarship to do research under Dr. Gene Braunwald at the University of California, San Diego, for me, it was certainly the road least traveled. My ambition was to specialize in cardiology, having been inspired in medical school at Dalhousie University by Dr. Robert Anderson, a Cardiologist who had just returned from being with Dr. Paul Wood. I indicated to Dr. Young that I would further my training in order to pursue an academic career.

I had virtually no experience with research and no concept of what an academician would be, other than I would be expected to be a physician and educator. The place to be was San Diego, with Dr. Eugene Braunwald, Chair of Medicine along with a faculty that was driven by discovery and innovation. The focus in cardiology was on myocardial infarction, the number one killer. It was now evident from Jennings work that the amount of myocardial damage (infarct size) occurring during a heart attack evolved over several hours and might be attenuated if appropriate drugs or agents could be administered early in its evolution. Infarct size was the big buzz, research revolved around methods to quantify and limit it, everyone was convinced that Gene Braunwald would get the big prize, he was brilliant, organized and a great administrator. Little did I know then, how much I would owe to him as a lifelong mentor and friend. I arrived in San Diego and Gene assigned me to research on muscle mechanics, this time in conscious animals with Dr. Stephen F. Vatner. While muscle mechanics was fascinating, I believed it had reached its peak and the future would be in biochemistry of the heart, so I made the decision I wanted to work with Dr. Burton E. Sobel, who at that time, was very active in the field. I met with Gene Braunwald and he was extremely encouraging, saying “go for it, I am moving to Boston next year to be Chairman of Medicine, and you are most welcome to join me once you finish your training”. Burt was incredibly intelligent, driven, demanding and even more demanding of himself - he was a great mentor and became a lifelong friend.

Burt’s laboratory was receiving world-wide recognition for estimating infarct size by serially measuring plasma-CK following myocardial infarction. These estimates were based on total CK, part of which was coming from the heart (MBCK) and part from other organs, particularly skeletal muscle. One could measure MBCK, which was quite specific for the heart, however, the assays for MBCK were qualitative, and not quantitative, which was required to estimate infarct size. Thus, my first project was to develop a quantitative assay for MBCK. The qualitative assay for MBCK required separating MBCK from MM on cellulose acetate paper by electrophoreses, since MBCK was more negatively charged than MM, and despite all efforts to standardize the scans for the fluorescent bands, the results were not reproducible. Another concern related to a computer program required to predict infarct size from plasma-CK based on the first 2 to 3 samples after a myocardial infarction. If one could predict, intervene and compare the prediction with the actual observed infarct size, it would be an interesting method to evaluate the effect of interventions on infarct size. This was an exciting time for myocardial infarction and our group in San Diego was at the leading-edge of the whole effort. The excitement was clear and the competition was all too palpable. It was suggested I meet with Dr. Hagivara, a physicist in Pasadena connected with NASA during the cold war era, whose research involved determining the speed and projectile trajectory of a missile launched from Russia enabling its destruction over the Pacific Ocean before it hit the mainland of the USA. Interestingly, this would be somewhat similar to predicting the trajectory curve for plasma-CK after a myocardial infarction. In my discussions with him, although his terminology and knowledge was sometimes beyond my grasp, it became evident that to obtain an early accurate prediction of a plasma-CK curve, we would require sampling over a longer interval than was practical. This was bad news, and on discussing the problem of how to quantify MBCK by fluorometer scanning techniques, he was even less optimistic. After driving back from LA that evening, I continued to ponder the problems I had outlined to him and, while in the shower, a solution occurred to me. The separated bands of plasma MBCK and MMCK on the strips were visualized through the generation of fluorescent NADPH, a reporter molecule. It occurred to me that NA-DPH is water soluble and if I cut the strip into 2 pieces, one containing MB and the other MM and inserted them into separate containers of water, with a little stirring, the water soluble NADPH would elute from the strip into a homogeneous solution to be measured accurately in a spectrometer. I called Burt, we were both late nighters, he met me at midnight, we both knew this would work. Over the next few weeks, I had adequate data with reproducibility; it was presented at the American College of Cardiology and published in the American Journal of Cardiology. I moved with Burt Sobel to Washington University in St. Louis, where he became Chief of Cardiology and I became Director of the Coronary Care Unit at Barnes Hospital. Research on CK flourished and, subsequently, in collaboration with Charles Parker and Burt Sobel, we developed a radioimmunoassay for MBCK based on an antibody to the B-subunit. This was published in Science and the antibody approach became the approach for future diagnostic markers, including today’s Troponins. MBCK became the gold standard for the diagnosis of myocardial infarction and was to remain so for the next 3 decades.

There was still one agonizing problem: could MBCK be released from cardiac cells that are not irreversibly injured, such as with ischemia. To solve this problem, I purified mitochondrial-CK and generated a specific antibody in rabbits and developed
a radioimmunoassay. If mitochondria released its CK, it would indicate the cells were irreversibly damaged. The paper was published in JBC, however with a rather disappointing caveat they indicated that no further manuscripts on mitochondrial-CK would be accepted unless we prove it had its own gene. There was a gene for the M-subunit, a gene for the B-subunit which obviously gave you 3 combinations, MM, MB or BB, but did not provide for a fourth combination, mitochondrial-CK. So the concern was whether mitochondrial-CK was a contaminant of ‘M’ or ‘B’ CK. In 1978, isolating a gene and elucidating its expressed product was far removed from what I considered possible at the time. However, an in vitro translation method became available and in collaboration with Arnold Strauss, we showed that mitochondrial-CK was indeed encoded by a separate gene. We submitted the paper to JBC and the editor promptly accepted it. Little did I know then, it would markedly change my research career, not because of mitochondrial-CK, but because it had exposed me to the techniques of molecular biology and recombinant DNA. I read and reread the book by Jim Watson on DNA to learn the techniques of molecular biology. I took it with me everywhere, probably read it 5 or 6 times and pondered the possibilities of its application to research problems in cardiology.

To digress for a moment, modern molecular biology really began in earnest around 1970. Many of the investigators in molecular biology left the field in the 1960s and went to neurophysiology they realized the human genome with billions of DNA sequences made it formidable to identify a gene responsible for a single protein. That would change with four discoveries from 1969 to 1971, all of which received Nobel prizes. The dogma was that one went from DNA to RNA to protein and there was no going back. The scientists David Baltimore and Howard Temin independently and simultaneously discovered the enzyme reverse transcriptase, enabling one to go from RNA to cDNA, a complementary sequence of the RNA, which was used to locate the sequence of the gene within the genome. DNA molecules are so large (~150 million base pairs), that any attempt to work with DNA induced random breaks, making experimental results un-interpretable. Kelly et al had shown that bacteria possessed specific enzymes called restriction endonucleases that would cut DNA at precise sites and provide DNA fragments of specific length. The third discovery was by Cohen and colleagues at Stanford, who made the first recombinant DNA molecule and expressed it in a bacterial vector. Lastly, Fred Sanger who had received a Nobel Prize for sequencing the first protein, insulin, had now invented a method to sequence DNA. This catapulted molecular biology into the modern era; it was now possible to isolate, clone, express and sequence genes.

After nine glorious and productive years at Washington University, I decided it was time to have my own program and I moved to Baylor College of Medicine as Chief of Cardiology. Baylor College was becoming a hub for molecular biology with Dr. Bert W. O’Malley and molecular genetics with Dr. C. Thomas Caskey. I outlined to Dr. Antonio Gotto, Chairman of Medicine at Baylor, that my plan was to develop a molecular cardiology research and training program and I am very indebted to Dr. Gotto for believing in me and in the future of molecular cardiology. In my first year or two at Baylor College, I did what I was comfortable with at that time – we cloned the genes for MMCK and MBCK which provided experience in the techniques of molecular cardiology. We were fortunate that in 1986, AHA invited grant applications for training (Bugher Centers) to train cardiologists in molecular biology of the cardiovascular system. Three were awarded, one to Children’s Hospital in Boston and two to Texas, one going to Dallas with Brown & Goldstein as Principal Investigators and the other to Baylor College, with me as the Principal Investigator. This was a major boost for molecular cardiology, my career and the cardiology program at Baylor College of Medicine. Having Tom Caskey as a collaborator and as a spokesman at the site visit was certainly a major factor in our success. We trained over 30 Bugher Fellows in molecular cardiology, many of whom today are Chiefs of Cardiology, Chiefs of Medicine, Deans or world-renowned researchers.

At that time, I was also making a connection in my mind with DNA and familial cardiomyopathies. I was convinced cardiomyopathies was an opportunistic field, as little had been done to elucidate the etiology over the previous 3 to 4 decades, except to clinically reclassify them for the sake of new questions for board examinations. Cardiac hypertrophy, a consistent feature of the cardiomyopathies, was a growth problem, and hence a DNA problem, therefore, elucidation of its etiology would require the techniques of molecular genetics. It also reminded me of my rotation with Dr. E. Douglas Wigle, in Toronto, one of the world’s experts on Familial HCM. It also gave me an idea to apply for an NHLBI grant. During my tenure at Washington University, I was an investigator in the prestigious Specialized Center of Research (SCOR) program, of which there were only nine in the USA, all dealing with ischemic heart disease. Having recently moved to Baylor College of Medicine decreased my chance of obtaining a SCOR, since it required a large multi-disciplinary infrastructure in ischemic heart disease. I thought to apply for a SCOR in familial cardiomyopathies would be novel and to our surprise, we were successful. We now had a training grant in molecular cardiology and a large SCOR program grant in molecular genetics. Dr. Christine Seidman (Cricket) would map the chromosomal location for the HCM gene to 14q1 ahead of us, however, we were able to confirm the same gene in one of our families. A lot of excitement was to follow as we mapped the first gene for atrial fibrillation, several genes for HCM and DCM as well as two genes for ARVC, and the gene for Wolff-Parkinson-White syndrome. In collaboration with Dr. A. J. Marian, currently Faculty at the University of Texas Health Science Centre at Houston, and at that time my Fellow, we developed the first transgenic rabbit for HCM which led to several studies on the pathogenesis of HCM. Cricket and I collaborated on several studies and we also became life-time friends.

The cardiology training program attracted the very best Fellows in the USA, Canada and Europe. Certainly, my 23 years as Chief of Cardiology at Baylor College and Methodist Hospital proved to be a very exciting and productive time, for which I am forever grateful.

In 2004, I returned to Canada as President and CEO of the University of Ottawa Heart Institute. I had come full circle, since Dr. Don Beanlands, my first mentor, was the Institute's first Chief of Cardiology and on my arrival Don was Deputy Director
General of the Institute. In terms of my own research career, it was now evident the technology was advancing to take on the big challenge, namely the pursuit of genes responsible for polygenic disorders, such as coronary artery disease and myocardial infarction. It was evident from the 1990s that to discover genes related to polygenic disorders would require, not families, but thousands of unrelated cases and controls and hundreds of thousands of DNA markers. At the time I was moving to Ottawa, Affymetrix was introducing a microarray with hundreds of thousands of DNA markers. Shortly after my arrival in Ottawa, I founded the Canadian Cardiovascular Genetics Center through the combination of a large donation of $5 million from the Ruddy family, a Canadian Foundation for Innovation grant of $11 million, and CIHR grants. This immediately put us in a position to be one of the early sites for genome wide association studies (GWAS) and I recruited Alexandre F.R. Stewart, PhD, a biologist from Pittsburgh. Ruth McPherson had already collected a large group of CAD cases and controls which were being genotyped through collaboration with investigators in the USA. We joined in with the first Affymetrix microarray and were fortunate to be part of the group to discover the first genetic risk variant for CAD, 9p21. This was the beginning of a terrific and exciting story, as we formed an international consortium, CARDioGRAMplusC4D, comprised of several prestigious institutions in the USA, UK, and Germany, which proved to be an extremely productive time, with several publications in Nature Genetics and other prestigious journals. Currently, a total of 50 genes predisposing to CAD have been identified, of which well over half of them were discovered through us and the investigators in the consortium. The first major finding was that 35 of these genes mediate their risk independently of cholesterol, blood pressure or diabetes, which indicates the pathogenesis of atherosclerosis is associated with as yet unknown mechanisms, in addition to the known risk factors (such as cholesterol, blood pressure or diabetes). Due to the predisposing genes, we are identifying new pathways related to the pathogenesis of CAD which undoubtedly will provide targets for the development of new therapies.

In my activities as President and CEO of the University of Ottawa Heart Institute, I have again been fortunate to have superior faculty, staff, senior management and a visionary Board. In 2012, the Scimago Institutions Rankings (SIR) ranked the UOHI to be in the top 2% for research impact among 3,043 institutions world-wide. We tripled our research endowment to $50 million and we were recently approved for funding ($200 million project) to build an extension to the Institute which will enlarge the facility by 50%. My return to Canada has been exhilarating to say the least and I am indebted to the Board, the Senior Managers, faculty and staff for their support and sustained drive for excellence.

My advice to young investigators is to recognize that there is probably nothing more important than your training – every extra year spent in training provides several years of dividends for future success. Fortunately, medicine is a very exciting field and whether you practice medicine, with or without research, it is very rewarding and I can only recommend it with the highest enthusiasm.

PEOPLE AND PLACES

L to R: Grant Pierce, Naranjan Dhalla, Tanya Ravingerová and Bohuslav Ostadal

Dr. Tanya Ravingerová was honoured by the IACS with the “Distinguished Service Award in Cardiovascular Science, Medicine and Surgery” at the International Symposium on Advances in Cardiovascular Research: From Bench to Bedside during May 21-24, 2013 at the Congress Centre of the Slovak Academy of Sciences in Smolenice, Slovak Republic.
Editor's note: Following preliminary discussions at the Chicago 2012 meeting and subsequent meetings in Los Angeles and San Francisco of the IACS “Global Network to Fight CVD” Steering Committee, a project was commenced which has led to a first of its kind process of facilitating development of guidelines in the state of Gujarat in India wherein a few pilot projects have already been initiated with large data pools and the on-going proposal pertaining to it has been designed. It is the intent of the Global Network to provide mentoring of the professionals who will work on the project in India to encourage their being able to facilitate further training and education to extend development of the women's guidelines in other areas of India and appropriate countries, even isolated areas in developed countries such as Canada's North. An IACS Guidelines Development Panel has been formed and will meet by conference call on June 19 including Co-chairs: Parloop Bhatt and Sharon Mulvagh; Members: James Willerson, Naranjan Dhalla, Keyur Parikh, Jawarhal Mehta, Wafia Etieba, Giuseppe Ambrosio, Bohuslav Ostadal, Martha Gulati, Rekha Mankad, Anjali Bhagra, Jagat Narula, Theresa Harvey-Pruden, Nizal Sarraf-Zadegan; Advisors: Noel Bairey Merz and Sonia Anand; Administrative Assistant: Ivan Berkowitz.

With input from Dr. Mulvagh and Bhatt as well as Panel members, Dr. Wafia Etieba has kindly prepared the following paper to help focus the project. I believe it is useful to know more about the awesome Dr. Etieba who wrote the following: “I was born, grew up and lived in a developing country, so I know about real stories, real challenges, real disappointments and yet hopes in this world! I myself worked very hard all my life in Egypt with a group of amazing women cardiologists, young and old. We inspired each other, dedicated ourselves and sacrificed many aspects of our personal lives to achieve targets and dreams in pursuing a scientific path in establishing the first Cardiology Department for women. As a head of the Cardiology Department, with the assistance of those who could help and those who took us seriously; a relatively young female cardiologist could make a difference in the university hospital to alleviate some suffering of people in the community, especially by women who, when sick, were the most vulnerable. After years of hard work, determination and clear vision, our team of women doctors could establish one of the best Cardiology Departments in Egypt. I consider the services which have been provided to low/no income women by that department, was both a mission and an achievement. Now, after I left, and despite the remaining challenges that never end in developing countries, they continue with the same enthusiasm, principles and attitude towards their patients and community.”

CVD Guidelines for Women in Middle and Low Income Countries

by Wafia Eteiba, MD, FACC, FIACS, Cairo, Egypt

Why specific guidelines? Why women?

Cardiovascular Diseases (CVD) prevention guidelines in women were first published by the American Heart Association (AHA) in 2004 in an effort to address a clear gap in knowledge regarding women-specific issues in CVD. These issues were updated in 2007 and again in 2011. The efforts of AHA continue to update, improve and overcome some of the encountered limitations in these guidelines and incorporate new bodies of evidence. Despite these efforts, the guidelines continue to be poorly adopted in emerging countries. The majority of emerging countries still live with the myth that coronary heart disease is a “rich, man’s disease”. This in itself is a major challenge hindering early detection of CVD and implementation of prevention guidelines. For example, in the United States, public awareness increased from 30% to 64% in years spanning 1997 to 2012 yet despite the apparent gains, challenges remain especially in improving awareness in younger women (44%) and women of ethnicity other than Caucasian (34%).

Enormous international and national efforts, both professional and public are required to further increase this awareness much like the scale of those which have evolved for Breast Cancer or Mothers Against Drunk Driving (MADD).

Emerging countries share a higher burden of CVD when compared to developed countries. An estimated over 80% of CVD deaths in women occur in low and middle income countries. For example, poorly educated and economically vulnerable women in India have higher risk of CVD and will be nearly equally affected as men by circulatory disease mortality which is expected to rise by 2015 to 32% vs. 34% respectively. Without female CVD prevention guidelines in emerging countries, the pandemic of CVD will continue to worsen.

Extension of CVD guidelines established in the developed world to women in emerging countries is a possibility especially when applied to conventional CVD risk factors such as smoking, high BP, diabetes, elevated blood cholesterol level and obesity. However, national and regional factors such as ethnic diversity, demographic shifts and environmental influences on diet and physical activity are just some of the factors uniquely affecting women in emerging countries. In other words, it is unlikely that direct application of the existing Western guidelines recommendations will be effective without modifications that tailor approaches to ensure implementation feasibility and efficacy according to the specific epidemiological characteristics and resources of the individual countries.
The need for prevention and/or treatment guidelines specific for women in the emerging world shares several commonalities as for women in developed countries. First, the need to eliminate disparities in CVD prevention diagnosis and treatment in women. Second, the need to reduce the toll of excess CVD mortality reported in women. Third, the recognition of the presence of women-related specific factors and diseases (e.g. autoimmune diseases, rheumatoid arthritis, pre-eclampsia, gestational diabetes, pregnancy induced HTN, and hormone therapy) more commonly occurring in women that heighten their risk for developing CVD. Fourth, the acknowledgement and understanding of why there is a preponderance of the clinical presentation of symptoms and signs of ischemia in the absence of obstructive coronary artery disease (Microvascular Coronary Disease) in women compared to men. Such factors should be considered during the evaluation of women and underscore the need for women-specific CVD guidelines.

On the other hand, there are factors peculiar to emerging countries that may render them different in CVD profile, therapeutic needs, prevention challenges, and public health strategies compared to the developed world for both men and women. Features of the diverse profiles that may impact upon CVD status in different countries include demographic changes, socio-economic growth, racial/ethnic background, age, genetic-environmental factors, culture, occupational status and lifestyle trends. Recognition of this concept was behind the call of the WHO for each country to seek to implement national clinical CVD guidelines directed towards high risk individuals and moreover is to develop appropriate preventive and health promotion strategies towards their low-risk populations.

Potential sex-based variations between developed and emerging countries also include health care accessibility, affordability and medical culture/attitude. Gender-stratified or gender-specific programs to optimize CVD preventive or therapeutic interventions in emerging societies will likely be required. For example, guidelines modifications in emerging countries seem to be mandatory in the face of costly CVD interventions and expensive drug use that are disproportionate to the available health care resources. Instituting national CVD guidelines using treatment eligibility options, rationalizing the use of high cost diagnostic tools, applying more strict criteria for patient selection during indicated interventions, reprioritization of risk factors control, use of generic medications, restructuring stepwise recommendation algorithms in a more cost-effective way and strengthening and catalyzing of initiatives towards professional and public awareness may all represent approaches to solutions to answer the challenges of CVD burden at societal, national and regional levels in the developing world.

Successful women-specific CVD guidelines should prioritize prevention strategies since women are uniquely situated to provide the basic care and nurturing of a heart-healthy lifestyle within their families. Policy development and applied research funding to support and provide effectiveness-based as well as gender-based evidence are required.

Women-specific CVD guidelines have been an important step towards reconciling the gender gap in cardiovascular outcomes in developed countries. Similar guidelines in emerging countries are now necessary to combat the high CVD burden. While such guidelines can utilize some of the fundamental principles in guidelines documents established in developing countries, they must be modified to account for country and cultural specific features, while focusing on prudent resource utilization, and cost effective preventive strategies. Such a tailored approach has the potential to succeed in overcoming gender and national barriers to improving cardiovascular disease outcomes in women while benefitting all societal members, including men women and children.

References:
Dr. Salim Yusuf will deliver 5th Harold Buchwald Heart Health Lecture

Following our extraordinary talks by Drs. Eldon Smith (Canada’s Heart Health Strategy), Jay Cohn (Live More Than 100 Years), Sharon Mulvagh (Women’s Heart Health), and Piero Anversa (Stem Cells), Dr. Yusuf will address the subject on which he is probably the world’s leading cardiologist:

"MOST PREMATURE HEART DISEASE IS PREVENTABLE"

Dr. Yusuf is President-Elect of the World Heart Federation; and Professor of Medicine and Executive Director of the Population Health Research Institute at McMaster University and Hamilton Health Sciences, where he has established international programmes of research in CVD and prevention involving 85 countries.

For details, please contact Ivan Berkowitz
Ph: (204) 228-3193 E-mail: ivan@mymts.net

FOR TICKETS: www.heartcademy.org
$60.00 each; Tables of 10 - $600.00
Jan Slezak honoured by Bratislava, Slovakia

Mayor of Bratislava, Slovakia, Milan Ftáčník (L), in the Primatial’s Palace, recognized personalities who have contributed to the cultural, social and sporting development of Bratislava. A bronze statue of the Knight Roland, defender of city’s rights and privileges, was presented to scientist Prof. Jan Slezak (R) who is honorary citizen of Winnipeg, Canada and Fellow and Member of the Board of Directors of IACS. Prof. D.H.S. Jan Slezak, MD., DSc., accepted the prize from hands of the Bratislava Mayor for his lifetime work as scientist, university professor, communicator and organizer in the field of cardiac medicine. Prof. Slezak worked for 50 years at the Slovak Academy of Sciences. His research activities are devoted to many topics, including Normal and Pathological Physiology and Experimental Cardiology which were his central fields of interest. He has published over 550 scientific papers. He was contributor and editor of eight monographs, and author of textbooks. In 1991, he founded the Slovak League for Prevention and Treatment of Cardiovascular Diseases Heart to Heart. For 10 years, he served as Director of the Institute for Heart Research, Slovak Academy of Sciences and later 11 years as the first Vice-President of the Slovak Academy of Sciences. Currently, Prof. Slezak is the vice-rector of the Slovak Medical University in Bratislava.

AD V A N C E S I N H E A R T H E A L T H

Risks: Blood Pressure Tests May Not Be Enough

By NICHOLAS BAKALAR, New York City, USA

A single blood pressure test, or even several readings over a short period of time, may not be enough to gauge the risk of developing cardiovascular disease.

Many researchers predict a subject’s risk for cardiovascular risk with a single blood pressure reading, but a new study has tracked blood pressure over 14 years beginning at age 41 in more than 61,500 men and women, most not taking blood pressure medicine. The paper was published online last week in Circulation.

Men who developed hypertension during middle age had a 70 percent lifetime risk of developing cardiovascular disease or stroke, compared with a risk of 35 percent among men who had lower blood pressure.

Among women, those who had hypertension from age 41 through 55 had a lifetime risk of 49 percent, compared with 22 percent among middle-aged women with lower blood pressure.

“This study has shown that changes in blood pressure early in middle age affect your lifetime risk,” said the lead author, Norrina Allen, an assistant professor of preventive medicine at Northwestern University. “We need to think about hypertension much earlier in life, around age 40.”

About 30 percent of men and 40 percent of women had increasing blood pressure over the 14 years; about a fifth of men and a tenth of women had decreasing readings.
Pour on the olive oil in good conscience, and add some nuts while you’re at it.

A careful test of the so-called Mediterranean diet involving more than 7,000 people at a high risk for cardiovascular diseases found the diet reduced having heart attacks and strokes when compared with a low-fat diet. A regular diet of Mediterranean cuisine also reduced the risk of dying.

The findings, published online by The New England Journal of Medicine, come from a study conducted right in the heart of Mediterranean country: Spain. A group of men and women, ages 55 to 80 at the start of the study, were randomly assigned to a low-fat diet or one of two variations of the Mediterranean Diet: one featuring a lot of extra-virgin olive oil (more than a quarter cup a day) and the other including lots of nuts (more than an ounce a day of walnuts, almonds and hazelnuts).

The Mediterranean diet is rich in fish, grains, nuts, fruits and vegetables. The diet is low in dairy products, red meat and processed foods. In this study, funded mainly by the Spanish government, the researchers made sure people got regular training sessions in the particulars of each diet. They also checked people’s actual consumption of olive oil and nuts with lab tests. One thing the researchers didn’t do was set any limits on calories or targets for exercise.

While lots of research has found benefits from the Mediterranean Diet, many of the studies have observed what people have eaten and looked for associations. One of this study’s strengths is that it randomly assigned people at high risk of developing cardiovascular disease to diets that stood to help them.

The study was stopped early (after a median follow-up of 4.8 years) because the benefits from the Mediterranean diet were already becoming apparent. Overall, the people consuming the diets rich in olive oil or nuts had about a 30 percent lower risk of having a heart attack, stroke or dying from a cardiovascular cause.

In absolute terms, there were about 8 of those problems for every 1,000 person-years in the Mediterranean diet groups compared with 11 per 1,000 person-years in the low-fat diet group.

How does the Mediterranean Diet work? The prevailing theory is that it lowers bad cholesterol and triglycerides while increasing protective good cholesterol. It may also also help the body’s ability to process sugar.

Heart disease experts said the study was a triumph because it showed that a diet was powerful in reducing heart disease risk, and it did so using the most rigorous methods. Scientists randomly assigned 7,447 people in Spain who were overweight, were smokers, or had diabetes or other risk factors for heart disease to follow the Mediterranean diet or a low-fat one.

Low-fat diets have not been shown in any rigorous way to be helpful, and they are also very hard for patients to maintain — a reality borne out in the new study, said Dr. Steven E. Nissen, chairman of the department of cardiovascular medicine at the Cleveland Clinic Foundation. “Now along comes this group and does a gigantic study in Spain that says you can eat a nicely balanced diet with fruits and vegetables and olive oil and lower heart disease by 30 percent,” he said. “And you can actually enjoy life.”

Not everyone is convinced, though. Dr. Caldwell Blakeman Esselstyn Jr., the author of the best seller “Prevent and Reverse Heart Disease: The Revolutionary, Scientifically Proven, Nutrition-Based Cure,” who promotes a vegan diet and does not allow olive oil, dismissed the study.

His views and those of another promoter of a very-low-fat diet, Dr. Dean Ornish, president of the nonprofit Preventive Medicine Research Institute, have influenced many to try to become vegan. Former President Bill Clinton, interviewed on CNN, said Dr. Esselstyn’s and Dr. Ornish’s writings helped convince him that he could reverse his heart disease in that way.

Dr. Esselstyn said those in the Mediterranean diet study still had heart attacks and strokes. So, he said, all the study showed was that “the Mediterranean diet and the horrible control diet were able to create disease in people who otherwise did not have it.”

A responder to the web site offered important advice “The Olive Garden is not the Mediterranean Diet”!
Predicting heart attacks before they happen

Like NBC’s Tim Russert, most people who die of a first heart attack, have had no symptoms.

The Challenge:
Predict heart attacks before they happen.

What if doctors could identify a heart attack or a stroke in time to prevent it? Humana, along with BG Medicine, academics from the U.S., Denmark and The Netherlands, and other industry leaders, is trying to turn this idea into reality. Most first heart attacks and strokes occur in people with no symptoms and who are considered of low to intermediate risk. NBC’s Tim Russert, for example, died from a massive heart attack in June 2008 after performing well on a stress test in April. The problem for him and about 80 percent of the others who die of a cardiovascular event is that doctors can’t detect the “vulnerable plaque” in vessel walls that ruptures and kills. There are no tests to detect it’s there, so people don’t know to seek treatments that could help.

The Solution:
Create an inexpensive test to detect vulnerable plaque.

Finding a way to identify people like Tim Russert has been called the Holy Grail of cardiology. It’s a quest of tremendous scientific and human value: Cardiovascular disease, after all, is the No. 1 killer. It’s responsible for the deaths of 2,600 Americans each day. If the risk could be reduced by 50 percent, the impact on life expectancy would be comparable to eradicating all cancers. That’s why Humana has partnered in a massive, $30 million international initiative. The mission is to develop inexpensive methods for detecting this vulnerable plaque. Humana’s role is to recruit 7,300 healthplan members in three areas of the country to volunteer for what is the largest biomarkers study of its kind.

Participants will undergo imaging tests and provide physical measurements and blood samples. Their health and health care will be observed for three years.

The Innovation:
Bridge the divide between health care and research

In several ways, this study is breaking the mold in scientific research. Instead of being done in academic medical centers, trucks with imaging and lab equipment drive out to where volunteers live. As a result, the study participants are much more diverse racially, ethnically and socioeconomically. A more representative group means more meaningful results. Up until now, the famous Framingham Heart Study has set the gold standard in how cardiovascular disease is assessed and treated. That ongoing study, which began in 1948, originally involved 5,200 residents of Framingham, Mass. For this study – the Biolmage Study – invitations to participate were issued in South Florida, Chicago and Louisville. It will have a balance that Framingham just doesn’t have. And the Biolmage study is a model for bridging the traditional divide between research and the health care system. Building that bridge is vital: Rapid advancement of personalized medicine – that is, doctors using an individual’s biologic information to better match that person to treatment – will require the help of our nation’s entire health enterprise.

The Benefit:
Money and lives saved.

Why is Humana doing this? Humana and the entire health care system will save money if costs go down – and they will go down if we have simple tests to predict cardiovascular events. But here are two other reasons: Because we know our superior data collection will add to the quality of the research. And because if we can play a role in predicting strokes and heart attacks, that will benefit people around the globe.
International Society of Chocolate and Cocoa in Medicine

On October 16, 2010 a group representing independent laboratories, academic departments and commercial confectionery companies from Europe and the US met in Perugia Italy to establish a new international society focused on the applications of Chocolate and Cocoa in Medicine. The name of the new society is International Society of Chocolate and Cocoa in Medicine (ISCHOM). The purpose of the society is to bring together groups and individuals in an international interdisciplinary organization in order to promote the science of use of cocoa, cocoa and chocolate products in the human diet, health and medicine for the benefit of the public. Individuals from all disciplines interested in the use of cocoa and chocolate in medicine are invited to join the society at one of three membership levels; Full, Associate, Honorary after filling out an application and paying the appropriate membership fee. Members will receive the society’s newsletter, access to the website and other materials prepared by the Executive Board or Society. Those in attendance at the initial meeting of the society elected the following officers and other members of the Executive Committee.

Prof. Norman Hollenberg of Harvard University and an IACS Fellow was elected at Honorary President with Prof. Gian Carlo di Renzo of Santa Maria della Misericordia University Hospital was elected as Chairman. Prof. Claudio Ferri of the University of L’Aquila was elected as secretary General with Prof. W Jeffrey Hurst of the Hershey Company elected as Treasurer. Other members of the Executive Committee were Prof. Roger Corder of the William Harvey Research Institute, Prof. J F Bisson of ETAP, Prof. Margarida Castelli of the University of Barcelona and Prof. Gian Franco Scarselli of Careggi Hospital Florence. For additional information contact Prof. di Renzo at direnzo@unipg.it

Norman Hollenberg

Dr. Norman Hollenberg and Dr. Naomi D. L. Fisher recently wrote, “The use of the word “dark” in dark chocolate, prominent in the title of this article, the article it accompanies, and on chocolate bar wrappers in high-end groceries around the world is symptomatic of this interest in identifying a simple, reliable, and inexpensive assay for what is good in chocolate. What makes it healthy? As is stated clearly in the report by Flammer et al in this issue of Circulation, we have probably identified the major chemical mediators: the subclass of flavonoids called flavanols, including especially the monomers epicatechin and catechin, and possibly proanthocyanins and metabolites. All cocoa is created flavanol-rich. It is primarily the processing of natural cocoa solids into cocoa powder or into confectionary chocolate that determines whether a final product is flavanol-rich or -poor. Because flavanols are bitter, manufacturers have often treated natural cocoa with processing techniques that necessarily destroy the flavanols as they enrich flavor and improve consistency.” They also stated, “The use of the term “dark chocolate” is misleading: There is nothing about the color of the chocolate that will tell you the flavanol content. One of the key places in the manufacturing chain where significant loss of flavanols occurs, after fermentation, is an alkalinization step called dutching. The Dutchman van Houten discovered 200 years ago that adding alkali-potash to cocoa nibs would enhance the taste, texture, and appearance of the cocoa. Dutched cocoa has the bitterness eliminated, together with most of the active flavanols. One relatively underreported effect of alkalinization is, in fact, darkening of cocoa, so that a very dark chocolate might be essentially devoid of flavanols.”
Can’t live without sugar, cakes and sweets? Addicted to sodas? We can assume you haven’t yet heard about Dr. Robert Lustig, the American endocrinologist and childhood obesity specialist, considered the current nutrition prophet of doom. Dr. Lustig declared war on sugar, blaming it for most of the western world’s modern disease, and his statements provoked a flurry of attention. Is this a tempest in a teacup, or should we really be afraid? And what role do artificial sweeteners play?

In a very provocative opinion column called “Sugar: The Bitter Truth,” published in February 2012 in the renowned scientific journal “Nature,” Dr. Lustig proclaimed sugar to be a poison, and that selling it should be regulated in the same manner as alcohol and cigarettes.

When Lustig talks about “sugar,” he primarily refers to “HFCS” – high fructose corn syrup “that since the 80’s is a main ingredient in food products such as cereals, sweet sodas, cereal snacks, cakes and cookies, sweets, alcoholic beverages etc.” Lustig calls it “The most evil supplement mankind has ever known”.

The article went viral in the American media, and Lustig’s claims made headlines, but that wasn’t the opening shot for his struggle. During July 2009, a 90 minute YouTube video was uploaded by Lustig on the subject, where the word “poison” in the context of sugar was mentioned 13 times.

To this day, the video has received more than 2 million views – an impressive number, considering the fact that we’re talking about a 90 minute discussion on the biochemistry of fructose and the human physiology. In April 2012, the New York Times dedicated a long article to Dr. Lustig’s “deadly” opinion regarding sugar.

No doubt about it, the buzz he created worked, but his provocative stance is winning a lot of criticism within the scientific community. His critics – and there are many – claim that the research testimonies Lustig presents aren’t enough.

“What we needed – another obsessive panic-creator obsessing with the sub kind of one nutrition ingredient instead of looking at the big picture,” blogs Allen Argon, a diet and exercise specialist, who also lectures in the national sports medicine academy and the American exercise council, among other places.

In the post published by the headline “Sugar Isn’t Evil: A Rebuttal”, Dr. Katz, head of the preventive research center in Yale University, claims that Lustig is talking nonsense.

Of course, nobody is disputing Dr. Lustig’s claim that high doses of sugar are bad for your health, but is it indeed the source of all evil in our diet and must we abstain from it completely? We checked to see what he’s saying and what other doctors and nutritionists are replying.

The increase in sugar intake is related to increase in illnesses?

Argument: The worrying increase in non-infectious chronic diseases, such as heart attacks, cancer, and diabetes specifically stem from the increase in sugar intake – particularly synthetically produced HFCS – which tripled around the world in the last 50 years.

What do other experts have to say: that the added sugar in our diet has indeed increased in the last decades, but the rates are a lot less than those indicated by Dr. Lustig. However, what has increased big time is our calorie intake.

According to the American agriculture report from 2009 examining nutritional consumption trends between 1970 and 2005, sugar and candy calorie consumption increased by 19% during those decades. But since 1999, there has actually been a decline of 9% in their consumption. Don’t be wrong, this isn’t enough to let out a sigh of relief yet.

According to the report, the average American consumes approximately 30 tablespoons of sugar over and above the sugars naturally existing in food, a very worrying piece of data. And yet, a 19% increase since 1970 is definitely not “thrice the amount of sugar”.

Different data in an earlier report by the American agriculture department from 2008 indicates that the calorie consumption average sum in 1970 was 2,172 calories, whereas in 2007 it reached 2,775 calories – meaning a total increase of 603 calories per day, which apparently landed somewhere in our collective fat cells. Simultaneously, our lifestyles became more sedentary – research published in 2009 in the American Journal of Medicine found a decrease of 10% in exercise rates between 1988 and 2006. These two changes combined to enable the obesity plague and the increase in related illnesses more than sugar consumption.

“Pointing a finger at sugar is as stupid as it was pointing it at fat during the 80’s”, claims Allen Argon in his blog. “A quizzical eye over the data will indicate that the increase in obesity dimensions is mostly caused by the calorie consumption increase in general and lack of exercising, and not necessarily in the increase of blood sugar additives”.

Dr. David Katz writes in his blog, “Like most researchers, I don’t agree with Dr. Lustig’s shortsighted, militant focus on sugar abstinence. He’s missing the forest while barking at a single tree.”
Is sugar bad for you like alcohol and smoking?

Argument: Sugar isn’t just “empty calories” the way health authorities claim, but poison damaging our health just as alcohol and cigarettes do.

What do other experts have to say: like alcohol consumption, sugar consumption in moderate dosages and as part of a healthy diet isn’t dangerous, consumption in high dosages and as a part of a “junk food diet” can be damaging.

“With all due respect to Dr. Lustig, alcohol is not a nutrient,” explains Guy Salmon, clinical dietitian. “We don’t need alcohol to survive. On the other hand, sugar is required for us to survive. The glucose is in our blood, it’s an exclusive substance for the brain, and supplies energy to our muscles.”

According to endocrinologist Dr. Ram Weiss of the Department of Human Nutrition and Metabolism at the Hebrew University in Jerusalem, Israel, research conducted among children and adults showed that drinking one soft drink serving (200-250ml) per day over time doubles the chance of gaining weight. Drinking two servings per day quadruples the risk.

“The increased risk graph is steep. Our body isn’t born with the ability to digest soft drinks, and that’s the reason why half the sugar in it immediately turns into fat, and the other half left doesn’t make you feel full, because the brain doesn’t translates it as a digestive action. In this, Dr. Lustig is completely right, but the problem is that he takes this issue to extremes”.

Is fructose more dangerous than white sugar?

Argument: Fructose is more dangerous than white sugar because it can encourage liver toxicity and a variety of chronic diseases. What do experts have to say: In higher dosages, fructose can lead to an infectious reaction in the liver.

“As opposed to white sugar, fructose doesn’t elevate the blood’s sugar levels, but it causes an increased formation of triglycerides (blood fats),” explains Dr. Raz. “When they elevate, they tend to set in the liver and muscle tissues, for example, and make them turn fatty. The body responds to the situation as an infection and sends white blood cells that secrete materials called inflammatory cytokines. But the cytokines prevent the liver from recognizing the insulin, and it increases the blood production to compensate the false deficiency, and that’s how excess sugar is created. Indeed, the pancreas keeps creating more insulin in response to the excess sugar, but that isn’t enough to overcome the problem, and so a significant amount of fructose accumulates, which can be even more dangerous than excess glucose.”

It’s also important to differentiate between the kind of fructose – natural or processed. Fructose, also known as “fruit sugar,” is found in fruits and vegetables in its natural, unprocessed form, combined with other components such as dietary fibers, vitamins, minerals and antioxidants that benefit your health. But the HFCS rich in fructose added in large quantities to industrial food products is processed and has no nutritional value. And that makes all the difference.

Lustig’s demand to downsize HFC as much as possible is completely justifiable. According to the American agriculture department’s report from 2009 between the years 1970-2005 this sweetener’s availability has grown by 387% per person, at the expense of white sugar, whose use declined by 38%. The industries producing soft drinks, processed foods, and baked goods are the main uses of HFCS, because it’s cheap and sweeter than regular sugar.

This said, Dr. Katz warns us “not to throw the baby out with the water. A diet can contain fructose and be healthy. Even so, it can also be low on fructose, but rich in sodium or trans-fats or light in fiber and omega-3 type fats and be as far away from healthy as can be.” Therefore, the emphasis must be on the kind of fructose and its quantities.

The natural sweeteners challenge

So sugar isn’t poison, but if we consume large quantities of it, it can absolutely damage our health. If so, how can we on one hand downsize our sugar intake, yet on the other hand not forgo sweets completely? Indeed a difficult problem, especially in light of the fact that the main solution offered to us so far, artificial sweeteners, is itself known to be controversial.

The creative solution, which seems to be getting trendier, is natural sweeteners. The grocery store variety that used to hold the only alternatives – honey, molasses and maple syrup – has new players joining in: agave nectar, Stevia, sugar alcohols (i.e. xylitol, erythritol), and monkfruit. Are these natural sweeteners in fact better than sugar and HFCS?

Guy Salmon explains that all of the traditional natural sweeteners contain large amounts of calories, similarly to sugar, and are based on sugar units, such as glucose, fructose, and sucrose. It’s true that if you consume them in their unprocessed form – whole honey, date honey, and real maple syrup (produced from a cedar tree, not the sugar imitation) – they contain some nutrients such as vitamins, minerals, and amino acids, but in large quantities, they can be harmful as well.

So what’s the bottom line?

“In the process of sweetening drinks, the natural sweeteners are preferable and better than the white sugar and corn syrup, but also need to be consumed in moderation”.

A sweetener based on the Stevia plant has been approved by health departments in certain countries. Stevia is a common shrub in South America, mainly in Paraguay and Brazil, and in the last decades has been grown in Japan and China as well. Stevia is actually an old-new sweetener. Old – because the plant has been a popular sweetener among the Inca tribes even before America was discovered, long before its sweet advantage was revealed to the western world. Moreover, Stevia has been used for years in many countries. New – because only during recent years has this usage been officially approved by leading health authorities around the world. The American FDA, and its European parallel EFSA have approved this sweetener, and in these two continents the use of Stevia-based sweeteners is growing immensely, even in sweetened beverages.

Stevia has key benefits. Its sweetness level is 300 times sweeter than sugar, which means that a very small amount is required to create a sweet taste. It is also excreted almost in full by the body, so it barely has any calorie value. It can be used for cooking or baking. Alongside all these benefits it has a bitter aftertaste, which gets stronger the larger the quantity you use.
Unlike other natural sweeteners, Stevia doesn’t contain fructose or other sugars, and it owes its strong sweet taste to special ingredients, called steviol glycosides. There is not a lot of research that has tested its safety, but studies that were conducted thus far have indicated that it isn’t carcinogenic and is safe to use in a home environment as a sweetener for beverages and cereals.

**Is the trendy agave syrup healthy?**

Agave syrup is a relatively new team player on the natural sweeteners shelf, and it’s receiving a lot of publicity. The sweetener is produced from the agave plant, sourced in Mexico and Central America. But if you thought this natural syrup will allow you to sweeten your food and drinks indefinitely, you’re mistaken.

Agave syrup contains about 90% fructose. So not only does it not downsize the calorie intake, but in large quantities it causes the same health problems as HFCS. Bottom line: agave, along with honey and molasses, is preferable over white sugar and HFCS, but it also needs to be consumed in moderation.

**Newer options**

Other natural “non-nutritive” sweeteners on the market today include sugar alcohols, including xylitol, and monkfruit blended with erythritol (also a sugar alcohol). Both of these have slight aftertastes, though less so than Stevia, and have been found to be safe according to the available research. Neither is a significant source of calories, and neither has been implicated in chronic diseases. However, as the saying goes, too much even of a good thing is still too much, and the same applies here. Overconsumption of sugar alcohols has been linked to digestive discomfort.

**ADVANCES IN HEART HEALTH**

Experts predict what we’ll be eating in 2050 online at boston.com by Deborah Kutz

The second annual Food Day was held on October 25, 2012, and the theme centered around predicting what our plates will look like 38 years from now.

Massachusetts Governor Deval Patrick celebrated Food Day by discussing sustainability – the buzzword that’s about maintaining our food supply – with local students and members of his cabinet at the Hayley House Café in Roxbury.

In Washington, a gaggle of nutrition experts gathered at the US Capitol to look into their own crystal balls. “Will we be eating algae burgers produced in a giant sea farm or homemade soups with produce from small organic farms?” asked Michael Jacobson, executive director of the nonprofit nutrition activist group Center for Science in the Public Interest who organized Food Day.

Here’s what experts, who attended the panel discussions or contributed their thoughts to a Food Day publication, had to say about what we should expect in our kitchens in 2050.

1. Healthier processed foods. High-sodium foods won’t be a problem because a variety of salt substitutes will have been developed and added to soups, baked goods, and condiments, predicted Jacobson. “Safe sugar substitutes and sweetness enhancers will end the problem of diets too high in sugar,” he added.

2. We’ll eat less meat and chicken. Plant-proteins in fake meat, seafood, and milk will replace three-quarters of the animal products we now consume today. Limited supplies of energy, water, and land will make it too costly to maintain a steady diet of burgers and franks.

3. We’ll have health planners along with financial planners. “Many of us do a very good job when growing our nest egg, but we don’t invest in our future health,” said Dr. David Katz, founding director of the prevention research center at Yale University who served on the panel. “We need to figure out what and how to eat before we get obese or have that first heart attack.” He predicts that personal health coaches – which could be a nurse or physician’s assistant – will help us plan our daily menus as we learn to value our health as much as our bank accounts.

4. A single computerized device will replace multiple appliances. If we could streamline our lives with smartphones, why can’t we have a single appliance that juices, cools, cooks, and freezes our food? “You’ll be able to walk in, talk to the appliance, and it will do whatever you ask it to do,” Cat Cora, who hosts the Iron Chef program on the Food Network, said in an interview with USA Today that appeared in the Food Day publication. She also predicted that supermarkets have computerized shopping carts that automatically fetch what you need. (Do we really need that with online grocery delivery services?)

5. Most of us will have a home garden. Aeroponic technologies, where plants are grown in an air or midst environment without the use of much soil, will allow us to have refrigerator-sized box gardens that can produce one-fifth of the vegetables and legumes we need, said Eric Meade, vice president of the Institute for Alternative Futures who participated in the panel discussion. We’ll also be more likely to participate in community gardens, he added.

6. We’ll have financial incentives to purchase more nutritious foods. Products will be labeled with a numerical value from 1 to 100 based from least to most nutritious and will be priced inversely proportionally to their nutrient value. Foods higher on the nutrient scale will require fewer food stamp dollars or will be discounted for those not on government subsidies. About 1700 supermarkets have already implemented the grading system called NuVal, said Katz who serves on the company board.

Walmart is already taking a few steps in this direction; last week the store teamed up with Humana and began offering healthy food discounts to those on Humana’s health plan. They get a 5 percent discount if they purchase Walmart’s “Great for You” labeled foods, said Andrea Thomas, Walmart’s senior vice president of sustainability. The label is affixed to fruits and vegetables (frozen, fresh, or canned), fiber-rich whole grains, low-fat dairy foods, nuts, seeds, and lean meats.

Thomas said during the panel discussion that she envisions cost-reductions going further with challenges to create nutritious family meals for under $10 – with cost-efficient recipes that people can scan into their iPhones while shopping and planning a weekly menu.
Dear Colleagues,

It is our great pleasure to invite you to join the VII. International Symposium on Myocardial Cytoprotection (ISMC2013) meeting in Pécs, Hungary. The Symposium will be organized by the Department of Surgical Research and Techniques and Heart Institute of Pécs University in cooperation with the Experimental Section of Hungarian Society of Cardiology and the International Academy of Cardiovascular Sciences. We intend to create a forum for discussion of the latest events in cardiology in both basic science and clinical practice. Thanks to the help of Professor Naranjan S. Dhalla, Executive Director of the International Academy of Cardiovascular Sciences, we can welcome a number of distinguished colleagues from all around the world as invited speakers. However, we intend to rely on active contribution of every participant. To this end, we provide forum for oral and poster presentations. To attract clinicians and young researchers, we are accrediting the symposium by the Continuous Medical Education Committee, and the Philosophy Doctoral Committee. We organise a poster competition, and abstract of all presentation will be published in a special issue of Experimental & Clinical Cardiology.

On behalf of the local organisers, we invite you to join us in Pécs, where together we can explore the future of cardiovascular research, it’s technical development and clinical application.

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Prof. Dr. Ferenc Galyas

Honorary Presidents
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Prof. Dr. Naranjan S. Dhalla
Prof. Dr. Zoltán Papp
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Looking forward to see You in Pécs, the Cultural Capital of Europe in 2010.
Ruminations on Aspartame and Milk

Little rumination is required to reach this conclusion: cows don’t make aspartame. But they don’t make strawberry flavoring, either.

This is relevant to a debate that involves a petition by the dairy industry to the FDA to change what qualifies as milk; a grass roots petition opposing the dairy industry’s petition; and a burgeoning amount of media ink and national attention.

As reported by NPR, the basic issue is that kids are consuming less milk in school, and the dairy industry would like to do something about it. Sometimes, the kids are swapping water for milk; sometimes, they are switching to diet sodas.

And that’s where the aspartame comes in. While the effects of offering flavored milks (chocolate, strawberry) on total dairy intake are themselves somewhat controversial, the dairy industry position is that enticing flavors do indeed help more milk go down.

The trouble with this argument is that flavored milks are sweetened with added sugar, and consequently provide extra calories. I checked on-line for the nutrition details of one prototypical brand of strawberry flavored milk. The low-fat version provides 130 calories and 18 grams of sugar per cup. For comparison, a comparable serving of Coca-Cola has about 26 grams of sugar. As school nutrition standards rise, tolerance for added sugar and calories- in soda or milk- is falling. And that means many schools are abandoning flavored milks, as they have abandoned sugar-sweetened sodas.

To counter such trends, the dairy industry would like to replace some or all of the added sugar in flavored milks with aspartame. The controversial part is that they don’t want to tell the kids about it. Specifically, the dairy industry is petitioning the FDA to omit any reference to ‘aspartame,’ ‘diet,’ or ‘artificially sweetened’ on the front of these milk cartons, contending that kids will find those designations a turn-off. They would like to call aspartame-sweetened strawberry milk just…milk.

As all Shrek fans know, there are many layers to a parfait. Let’s work through them.

First, the overall role of dairy in the diets of school-age children is itself somewhat controversial. There are concerns among some nutrition experts about everything from calories, to saturated fat, to contaminants such as bovine growth hormone. Many studies suggest benefits to diet and health when kids consume dairy routinely, but that may be because the prevailing norm is soda. Whether routine dairy intake is advantageous over water is far less clear. And while benefits of dairy intake to bone health makes good sense, it’s worth noting that most populations around the world whose only dairy intake is breast milk in infancy have greater, not lesser, bone density than we do.

Second, there is the fact with which I began: cows don’t make strawberry flavoring. I signed on to the petition against the dairy industry’s petition, because I believe in people’s right to see the truth, the whole truth, and nothing but the truth on display. If aspartame is added to milk, you shouldn’t have to work hard to figure it out.

But if aspartame-sweetened milk isn’t just ‘milk,’ then neither is sugar-sweetened, strawberry-flavored milk. And while we can make the case that if milk is sweetened with aspartame it should say so clearly on the front of the carton, we might just as readily argue that if chemical flavorants are used to imitate the taste of strawberry, and chemical colorings are used to imitate its color, then the front of pack should list these chemicals as well. What’s good for the goose should be good for the gander- or cow, in this case.

Why the difference? I think it’s just a matter of nonsensical convention. We are accustomed to letting ‘strawberry flavored’ fill in on the front of pack for a host of ills in the ingredient list. A different standard for aspartame probably makes no real sense. The situation is analogous to all of the fuss over medical marijuana, just because we aren’t accustomed to its medical use. Meanwhile, cocaine and heroin analogues even more potent than heroin itself are an established part of the medical pharmacopeia, in routine use.

Third, and finally, there is aspartame itself. A sizable faction in our population considers artificial sweeteners slow poisons. I receive an email almost every day from one such quarter, citing the studies linking aspartame to formaldehyde formation and associated harms. There is, as well, a well-established link between aspartame and headaches, and other neurological ailments, among those sensitive to the chemical.

But, of course, aspartame- the sweetener in Equal and Nutrasweet- has been consumed by tens, and perhaps, hundreds of millions of people for years to decades. If the substance had even remotely common toxic effects, they would long since have shown up as a clear change in epidemiology and national health statistics. They have not. We have less cancer, not more; and, for the most part, no clear evidence of increasing rates of neurological disease.

There is another problem with aspartame, however. It’s sweet. Very sweet. It is 200 times as sweet as sugar when matched for weight. And that concerns me. I think we have it right when we refer to a ‘sweet’ tooth rather than a ‘sugar’ tooth. The message our taste buds send our brains is not specific to sugar-it’s a general message of sweetness.

That message induces a reward, and what’s known as tolerance: the more you get, the more you need. So I have long been...
concerned that all sugar substitutes are at best a lateral move: they take sugar and calories out of your diet right now, but they help grow your sweet tooth into a sweet fang. That sweet fang then goads you to seek out sweeter drinks, dressings, desserts, sauces, spreads, snacks and condiments—probably without even realizing it. By corrupting the palate this way, I think all sugar substitutes have potential to undermine food choice and the quality of the overall diet. I avoid them all.

So, at the bottom of the parfait glass, we find ourselves with imperfect knowledge, and imperfect options. Do kids benefit from consuming more milk? The epidemiology says yes, but only compared to a very questionable status quo. We don't know that less milk, more water wouldn't be trading up.

Should aspartame-sweetened milk indicate up front it's not quite the stuff the cow made? Yes. But frankly, the same standard should apply to strawberry flavoring, and whatever potion is used to make it. Is aspartame toxic? For most people, not per se. But for everyone, it is an intense sweetener that will likely make you want ever sweeter food. Avoiding intense sweeteners is among the strategies I recommend to rehabilitate your taste buds, make them more sensitive, and teach them to prefer less. When you actually prefer food less sweet, you can have that cake- and eat it, too; you can love food that loves you back.

One final thing. If you would like your kids to be able to sniff out the truth about food no matter what the FDA requires on the front of the pack, my colleagues and I offer a free and proven program to educate them, and you, accordingly- help yourself to the free Nutrition Detectives™ DVD at http://www.davidkatzmd.com/nutritiondetectives.aspx.

And there you have it. In the good company of the cows, I am aspartame-free, and devoid of strawberry flavoring. Unlike the cows, I am done ruminating - at least for now.

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FUTURE OF HEART HEALTH

The Calculus of National Medical Research Policy — The United States versus Asia

Since the founding of the National Institutes of Health (NIH) and the National Science Foundation (NSF) more than six decades ago, the United States has maintained a preeminent position as a government sponsor of medical research. That primacy is being tested, however, by potent economic challenges. The NIH's proposed budget for fiscal year 2013 would freeze baseline funding at 2012 levels, continuing a decade-long failure to keep pace with the rising costs of conducting medical research. Across-the-board cuts mandated by the Budget Control Act (BCA) of 2011 will also affect medical research, with the NIH, NSF, and other federal research sponsors sustaining budgetary reductions of about 8% next year.

Cuts to government-funded research will have adverse long-term effects on the health care system and the economy and may irreversibly compromise the work of laboratories long accustomed to receiving stable federal support. Moreover, many medical researchers could transfer their knowledge and resources abroad. In fact, five emerging Asian economic or technological powers—China, India, South Korea, Taiwan, and Singapore—already have medical research policies in place that are filling the void being created by ever more restrictive U.S. funding. Several U.S.-based economists have justified increasing research budgets on the premise that medical discoveries have intrinsically high economic value. For example, Murphy and Topel (in The economic value of medical research. Chicago: University of Chicago Press, 1998.) have suggested that eliminating deaths related to heart disease had an estimated worth of $48 trillion, and a 1% reduction in cancer related mortality could save $500 billion. Beyond these ambitious goals, however, are more practical arguments favoring support for medical research.
HeartBeat Connections: A Rural Community of Solution for Cardiovascular Health

In 2009, the Heart of New Ulm Project (HONU) launched a multiyear initiative to reduce the rate of myocardial infarctions among adult residents of New Ulm, Minnesota, an agricultural region approximately 100 miles southwest of the Minneapolis-St. Paul metropolitan area. The project is a collaborative partnership among Allina Health, the Minneapolis Heart Institute Foundation, the New Ulm Medical Center (NUMC), and the broader community of New Ulm. The HONU Project20 involves interventions in the community, health care, worksites, and the nutrition and built environments. The overarching vision of the HONU Project is to create a sustained culture of health in New Ulm, with programs and initiatives that can be replicated successfully in other rural communities. Within the nutrition environment, for example, the project has partnered with local restaurants to improve menu offerings and is working with convenience stores to increase healthy “grab and go” snack and beverage options. A grant from the U.S. Department of Agriculture is helping the project to train farmers to expand and market their fruit and vegetable offerings through local farmers’ markets and community-supported agriculture drop sites. Volunteers are coordinating events enthusiastically, and employers are embracing worksite wellness initiatives. Community leaders are helping to plan wellness programs, and local organizations are promoting heart-healthy lifestyles. The commitment and engagement of the entire community drive the success of all the project’s activities.

To help inform HONU initiatives, a 36-member steering committee was established at the project’s inception. Volunteer members include representatives from a broad cross-section of the community, including physicians at NUMC, local employers, the City of New Ulm, the chamber of commerce, churches, the school district, local colleges, the Brown County Public Health Department, and the general community. The steering committee meets quarterly under the leadership of the HONU Project director. These meetings generally include a brief update on project progress and related current events (e.g., funding, outcomes reports, and staff changes), followed by a detailed discussion of near-term intervention priorities and associated challenges. Members often are split into smaller groups to encourage more intimate brainstorming and vetting of possible solutions to such challenges and other community engagement ideas. The meetings end with a group wrap-up session, during which an action plan is informally endorsed or modified.

One of the key reasons the community of New Ulm was selected for the project was because its residents are served primarily by a single health care facility: NUMC. About 90% of New Ulm residents are NUMC patients and have an EHR. The EHR enables project planners to address various population segments and disease risk levels with the goal of identifying, implementing, and tracking interventions that will positively influence health.

To obtain a robust baseline assessment, a community screening initiative was conducted in 2009 with a goal of creating a “community diagnosis” that would inform the pending interventions. Nearly half of the target population, defined as residents of the 56073 zip code who were 40 to 79 years of age attended a community screening. The screenings included both biomedical data (e.g., lipids, high-sensitivity C-reactive protein, and blood glucose), which was uploaded automatically into participants’ Electronic Health Records (EHR), and behavioral data (e.g., daily consumption of fruits and vegetables, physical activity level, and perceived stress level), which was entered manually into a specially created flow sheet within participants’ EHRs.

Data collected from the 2009 screening identified many adults in New Ulm who did not currently have CVD but were at high near-term risk for developing it. On the basis of local EHR data, 2,500 residents of the 56073 zip code area who are 40 to 79 years of age were considered to be at high risk for CVD, defined as a 15% estimated probability of a CVD event in the next decade. This group mainly includes people with metabolic syndrome (i.e., those with multiple elevated CVD risk factors).

Because this group of 2,500 community residents is an asymptomatic primary care population without diagnosed CVD or a CVD risk equivalent (e.g., diabetes), there are many expected gaps in their levels of optimal preventive care. One particularly notable observed gap was that 50% were considered clinically eligible for at least one additional preventive medical therapy that they were not getting (e.g., aspirin, a statin, or a blood pressure medication). These initial findings indicated a major untapped opportunity to close gaps in optimal preventive CVD care for a large segment of the target population. The HONU Project leadership identified this as a crucial area to address because 70% of all myocardial infarctions that occur in a given year in Minnesota are first-time events, that is, they occur in people who do not have an existing CVD or diabetes diagnosis.

Personal Care Providers face significant time constraints and other barriers to delivering preventive CVD care, as suggested by national guidelines. Innovative population health approaches that incorporate EHR and community data and engage communities in defining and addressing health needs are necessary to help more patients prevent disease. This is one of the recommendations of the revised Folsom Report (Folsom Group. Communities of solution: the Folsom Report revisited. Ann Fam Med 2012;10:250–60). Identification of patients at high-risk for CVD, coupled with the development of collaborative approaches that engage other members of the health care team in patient care, can help to ensure that these patients receive appropriate therapeutic regimens and have needed access to ongoing coaching and community resources to aid in sustaining lifestyle changes. The HBC program could potentially serve as a model of how to support PCPs and patients in achieving CVD prevention targets and enhance the broader relationship of health care systems with the communities they serve. Evaluation of this program is needed to provide more definitive conclusions regarding its effectiveness.
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