Cardiovascular disease (CVD) claims the lives of millions every year around the globe. If those at risk for developing CVD could be identified early and receive preventive care, many of these people would live longer and healthier lives.

Learn about important guidelines that will enable labs to provide data on available biomarkers and improve the clinical diagnosis and management of acute cardiac conditions.
# TABLE OF CONTENTS

**ACKNOWLEDGEMENTS**
- Program Co-Chairs.................................................................................................................. 3
- Exhibitors................................................................................................................................ 3

**PROGRAM SCHEDULE** ........................................................................................................ 4

**SPEAKER BIOS AND DISCLOSURES**
- Fred Apple, Robert Christenson, Chistopher deFilippi James de Lemos............................... 5
- James Januzzi, Allan Jaffe, Peter Kavsak, David Morrow....................................................... 6
- Kristin Newby, Frank Peacock, Alan Wu................................................................................... 7

**POSTER ABSTRACTS**
- Poster Abstracts 1 — 2.............................................................................................................. 8
- Poster Abstracts 3 — 5.............................................................................................................. 9
- Poster Abstracts 6 — 7............................................................................................................. 10
- Poster Abstracts 8 — 9............................................................................................................. 11
- Poster Abstracts 10 — 11 ...................................................................................................... 12

**CONTINUING EDUCATION RECORDING FORM** ..................................................................... 13
Program Co-Chair — Robert Christenson, PhD
University of Maryland School of Medicine, Baltimore, MD

Dr. Christenson is Professor of Pathology and Professor of Medical and Research Technology at the University of Maryland School of Medicine in Baltimore, Maryland. Clinically, Dr. Christenson is Director of the Clinical Chemistry, Toxicology, and Core Laboratories at the University of Maryland Medical Center, where he is also Medical Director of Point of Care Services. Dr. Christenson has an active research program in the area of biomarkers of cardiovascular disease and renal dysfunction. Dr. Christenson directs the Clinical Chemistry Research Laboratory, a CLIA-licensed and CAP-accredited research laboratory at University of Maryland School of Medicine that specializes in Government and Industry sponsored clinical trials. Dr. Christenson also directs the University of Maryland School of Medicine’s ComACC training program and is active in the Pathology Residency Program.

Dr. Christenson holds four patents and has published over 265 peer-reviewed manuscripts, over 250 abstracts, 4 books, and 50 book chapters and monographs. He is an associate editor for the journal Clinical Biochemistry and has served for 10 years on the editorial board member for Clinical Chemistry Journal and chaired the editorial board of AACC’s Clinical Laboratory News.

Program Co-Chair — Alan Wu, PhD
University of California, San Francisco, CA

Dr. Wu is Chief of Clinical Chemistry and Toxicology at San Francisco General Hospital and Professor of Laboratory Medicine, University of California, San Francisco. He received B.S. degrees in chemistry and biology at Purdue University, West Lafayette, Indiana, and a Ph.D. degree in analytical chemistry at the University of Illinois, Champaign-Urbana, Illinois. He completed a postdoctoral fellowship in clinical chemistry at Hartford Hospital. He is certified by the American Board of Clinical Chemistry in Clinical Chemistry and Toxicological Chemistry.

Exhibitors

As a global leader in diagnostics and pharmaceuticals, Roche is focused on helping patients live longer, healthier lives. We are leading the way in the development of tests and customized treatments that enable personalized healthcare solutions, tailored to the needs of unique groups of patients.

In the lab, Roche helps optimize testing efficiency with flexible, scalable solutions like cobas® integrated chemistry/immunoassay analyzers with broad menus and comprehensive automation options. Molecular labs use our PCR-based solutions to deliver innovative companion diagnostics and actionable information that help clinicians provide personalized care. Anatomical labs use integrated slide staining and workflow management platforms from Ventana Medical Systems Inc. to optimize efficiencies.

From labs to hospitals, clinics and patient homes, Roche is dedicated to providing exceptional customer support, enhanced testing efficiency and reliable clinical information that can be used to improve the lives of patients.

To learn more, visit www.roche-diagnostics.us

AACC is a global scientific and medical professional organization dedicated to clinical laboratory science and its application to healthcare. Our leadership in education, advocacy and collaboration helps lab professionals adapt to change and do what they do best; provide vital insight and guidance so patients get the care they need.

Visit the AACC booth in the exhibit hall to learn about the organization’s activities and upcoming educational programs.

To learn more, visit www.aacc.org
<table>
<thead>
<tr>
<th>Time</th>
<th>Session Title</th>
</tr>
</thead>
</table>
| 9:00 AM | **Welcome Remarks**  
Alan Wu, PhD, University of California, San Francisco, CA |
| 9:05 AM | **Session 1: Insights into the Development of Clinical Guidelines**  
Ground rules for clinical guidelines development  
Robert Christenson, PhD, University of Maryland School of Medicine, Baltimore, MD |
| 9:40 AM | **SESSION 2: Biomarkers for Acute Coronary Syndromes (ACS)**  
Clinical Perspectives  
Allan Jaffe, MD, Mayo Clinic, Rochester, Minnesota |
| 10:15 AM | **Analytical Perspectives**  
Fred Apple, PhD, Hennepin County Medical Center, Minneapolis, MN |
| 10:45 AM | **Ask Questions and Chat with Drs. Christenson, Jaffe, and Apple**  
(in the Networking Chat Lounge with Dina Greene, PhD, University of Washington, Seattle) |
| 11:05 AM | **Short-term risk stratification for cardiac patients: Emergency Department perspective**  
Frank Peacock, MD, Baylor College of Medicine, Houston, Texas |
| 11:35 AM | **Short-term risk stratification for cardiac patients: Cardiology perspective**  
David Morrow, MD, Brigham and Women’s Hospital, Boston, MA |
| 12:00 AM | **Ask Questions and Chat with Drs. Peacock and Morrow**  
(in the Networking Chat Lounge with Dina Greene, PhD, University of Washington, Seattle) |
| 12:15 PM | **LUNCH BREAK — VISIT VIRTUAL EXHIBITS AND POSTER HALL** |
| 1:00 PM | **SESSION 3: Use of Acute Cardiac Biomarkers for Primary Care and Other Disease Etiologies**  
Primary Care  
James de Lemos, MD, UT Southwestern Medical Center, Dallas, TX |
| 1:35 PM | Other Disease Etiologies  
Peter Kavsak, PhD, Juravinski Hospital and Cancer Centre and McMaster University, Hamilton, Ontario |
| 2:10 PM | **Renal failure and Acute Kidney Injury**  
Alan Wu, PhD, University of California, San Francisco |
| 2:40 PM | **Ask Questions and Chat with Drs. de Lemos, Kavsak and Wu**  
(in the Networking Chat Lounge with Dina Greene, PhD, University of Washington, Seattle) |
| 3:05 PM | **SESSION 4: Biomarkers for Heart Failure**  
Acute Heart Failure  
Christopher deFilippi, MD, University of Maryland |
| 3:40 PM | Chronic Heart Failure  
James Januzzi, MD, Massachusetts General Hospital and Harvard Medical School, Boston, MA |
| 4:15 PM | **Novel Heart Failure Biomarkers**  
L. Kristin Newby, MD, Duke University Medical Center, Durham, NC |
| 4:45 PM | **Ask Questions and Chat with Drs. DeFilippi, Januzzi, and Newby**  
(in the Networking Chat Lounge with Dina Greene, PhD, University of Washington, Seattle) |
| 5:00 PM | **End of Conference**  
Playbacks of the presentations will be available after the program ends. |
Dr. Apple’s research interests have been centered in the areas of cardiac biomarkers in acute coronary syndrome and heart failure, and forensic toxicology. His CLIA-certified research laboratory is the “Cardiac Biomarkers Trials Lab” at the Minneapolis Medical Research Foundation of Hennepin County Medical Center. Dr. Apple has served as an Associate Editor of Clinical Chemistry for the past 14 years. He has served or serves as a member of the ‘National Academy of Clinical Biochemistry’ committee, the ‘Joint ESC-ACC-FAH-WHA Global Task Force’ and the ‘Universal Definition of Myocardial Infarction’ committee. He is also a member of the IFCC “Task Force on Clinical Application of Cardiac Biomarkers”. He has served on the Institute of Medicine’s Committee on Qualification of Biomarkers as Surrogate Endpoints of Chronic Disease Risk and on the NHLBI Working Group for Onsite Tools and Technologies for Clinical Cardiovascular Research and Point-of-Care.

Disclosure: Speaker has disclosed grant/research support from Abbott Diagnostics, BD, Beckman Coulter, Siemens’ Healthcare, Roche Diagnostics, Ortho Clinical Diagnostics, Nanomix, Trinity BioTech, and Alere; consultant fees from Philips Incubator; honorarium/expenses from Abbott Diagnostics, and Roche Diagnostics.

Robert Christenson, PhD
University of Maryland School of Medicine, Baltimore, MD

Dr. Christenson is Professor of Pathology and Professor of Medical and Research Technology at the University of Maryland School of Medicine in Baltimore, Maryland. Clinically, Dr. Christenson is Director of the Clinical Chemistry, Toxicology, and Core Laboratories at the University of Maryland Medical Center, where he is also Medical Director of Point of Care Services. Dr. Christenson has an active research program in the area of biomarkers of cardiovascular disease and renal dysfunction. Dr. Christenson directs the Clinical Chemistry Research Laboratory, a CLIA-licensed and CAP-accredited research laboratory at University of Maryland School of Medicine that specializes in Government and Industry sponsored clinical trials. Dr. Christenson also directs the University of Maryland School of Medicine’s ComACC training program and is active in the Pathology Residency Program.

Dr. Christenson holds four patents and has published over 265 peer-reviewed manuscripts, over 250 abstracts, 4 books, and 50 book chapters and monographs. He is an associate editor for the Journal Clinical Biochemistry and has served for 10 years on the editorial board member for Clinical Chemistry Journal and chaired the editorial board of AACC’s Clinical Laboratory News.

Disclosure: Disclosed no relevant financial relationships.

Fred Apple, PhD
Hennepin County Medical Center, Minneapolis, MN

Dr. Apple is Medical Director of Clinical Laboratories, Clinical Chemistry, POC Testing and Clinical and Forensic Toxicology Laboratories at Hennepin County Medical Center in Minneapolis, MN, and Professor of Laboratory Medicine and Pathology at the University of Minnesota School of Medicine.

Disclosure: Speaker has disclosed grant/research support from Abbott Diagnostics, and Roche Diagnostics, Ortho Clinical Diagnostics, Nanomix, Trinity BioTech, and Alere; consultant fees from Philips Incubator; honorarium/expenses from Abbott Diagnostics, and Roche Diagnostics.

Christopher deFilippi, MD
University of Maryland School of Medicine, Baltimore, MD

Dr. deFilippi has an active clinical research program in collaboration with cardiologists, nephrologists, emergency medicine physicians, and clinical chemists throughout the country evaluating how blood tests can be best used to identify patients with kidney disease and how such patients may benefit from further heart tests or treatments.

Currently Dr. deFilippi is conducting multi-center studies to establish novel uses for established blood based cardiac biomarkers and evaluating the utility of new cardiac markers. The focus is on those with underlying renal disease and identifying significant coronary disease in asymptomatic but potentially higher-risk populations such as the elderly.

Dr. deFilippi oversees the cardiac evaluation program of patients who are being considered for kidney transplant. He has clinical expertise in echocardiography and diagnostic cardiac catheterization and is actively involved with both at the University. Although his general cardiology practice focuses on patients with renal disease, Dr. deFilippi also sees general cardiology patients with ischemic heart disease. His teaching responsibilities include cardiology and general internal medicine trainees.

Disclosure: Disclosed no relevant financial relationships.

James de Lemos, MD, PhD
UT Southwestern Medical Center, Dallas, Texas

Dr. de Lemos is Professor of Medicine at UT Southwestern Medical Center and holds the Sweetheart Ball-Kern Wildenthal, MD, PhD Distinguished Chair in Cardiology. He graduated from Harvard Medical School and completed an Internal Medicine Residency at UT Southwestern Medical Center, where he also served as Chief Medical Resident. He completed a fellowship in Cardiovascular Medicine and served on the faculty at the Brigham and Women’s Hospital before returning to UT Southwestern Medical School. He has served as the Cardiology Service Chief at Parkland Memorial Hospital, and the Cardiology Fellowship Director at UT Southwestern.

He has held positions on multiple committees of the AHA and ACC, including the STEMI Guideline Committee, and as Chair of the Research and Publications Committee for the NCDR ACTION-GWTG registry. He is a standing member of the FDA’s Cardiorenal Advisory Panel. He has been named the incoming Executive Editor for Circulation, and has served on the editorial boards of the Journal of the American College of Cardiology, the American Journal of Cardiology and the American Heart Journal. His primary research interests are in early detection, risk assessment and management of cardiovascular disease, with a particular focus on the role of cardiovascular biomarkers. His research has evaluated existing biomarkers such as B-type natriuretic peptide, C-reactive protein and cardiac troponins as well as novel biomarkers reflecting biological pathways of disease. He was the lead author of the Z phase of the A to Z trial, an international trial investigating different cholesterol lowering strategies in patients with acute coronary syndromes. He has mentored >30 post-doctoral research trainees and has authored or coauthored over 300 manuscripts or book chapters. He has won several teaching and mentorship awards, including the 2015 Women in Cardiology Mentoring Award by the AHA. He has...
been elected to the Association of University Cardiologists and the American Society of Clinical Investigation.

**Disclosure:** Speaker has disclosed receiving consultant fees from Roche and Abbott.

### James Januzzi, MD
**Massachusetts General Hospital, Boston, MA**

Dr. James Januzzi is currently the Roman W. DeSanctis Endowed Distinguished Clinical Scholar in Medicine at the Massachusetts General Hospital and Hutter Family Professor of Medicine at Harvard Medical School. He is also a faculty member at the Harvard Clinical Research Institute. Dr. Januzzi’s work has contributed greatly to the understanding of cardiac biomarker testing, where his work with several markers has set international standards for use in diagnosis, prognosis, and management of patients suffering from acutely decompensated heart failure, chronic heart failure as well as those with acute coronary syndromes. He has published more than 450 manuscripts, book chapters, and review articles, and has edited two text books on cardiac biomarker testing as well as the Massachusetts General Hospital Cardiology Review Book. He is on the editorial board of numerous scientific journals, including current service as an Associate Editor at the Journal of the American College of Cardiology: Heart Failure. He was the chairman of the NT-proBNP and ST2 Consensus Panels, and lead author of the Heart Failure Section for the Universal Definition of MI Biomarker Task Force. He is currently the Chair of the ACC Task Force on Consensus Statements and was a section editor and member of the working group for the 2013 ACC/AHA Clinical Practice Guidelines for Heart Failure. Since 2005, Dr. Januzzi has also served as a team physician for the Boston Red Sox baseball club.

**Disclosure:** Speaker has disclosed grant/research support from Abbott Diagnostics, Beckman Coulter, Ortho-Clinical Diagnostics, Roche Diagnostics, and Siemens; Consultant fees from Abbott Diagnostics, Beckman Coulter, CADTH, Roche Diagnostics and Siemens; honorarium/expenses from Abbott Diagnostics, Beckman Coulter, Randox and Roche Diagnostics.

### Allan Jaffe, MD
**Mayo Clinic, Rochester, Minnesota**

Dr. Jaffe is a graduate of the University of Maryland School of Medicine. He received his house staff and Cardiology training at Washington University and continued there for 22 years rising to the rank of Professor of Medicine and the Director of the Coronary Care Unit. He subsequently moved to the State University of New York where he was Chair of the Cardiovascular Division, Associate Chair of Medicine for Academic Affairs, and Professor of Medicine. After four years he moved to the Mayo Clinic where he is presently Professor of Medicine in the Cardiovascular Division and Chair of the Division of Core Clinical Laboratory Services within the Department of Laboratory Medicine and Pathology. He is a noted authority on the use of biomarkers of cardiac injury, inflammation, hemodynamic disturbance, and coagulation and particularly in regard to their clinical utility. He has published a large number of original manuscripts, book chapters, reviews, and sits on most of the prestigious editorial boards and guideline committees in the Cardiology community.

**Disclosure:** Speaker has disclosed receiving consultant fees from Novartis, Beckman Coulter, Critical Care Diagnostics, Amgen, Alere, and Radiometer as well as honorarium/expenses from Abbott Diagnostics and Roche Diagnostics.

### Peter Kavsak, PhD
**McMaster University, Hamilton, Ontario, Canada**

Dr. Kavsak has a proportional publication bias on cardiac biomarkers, in particular the analytical and clinical evaluation of these laboratory tests. He is precisely interested in exploring the utility of cardiac biomarkers in and outside the acute coronary syndrome setting. He has received quality funding from North American public granting agencies, foundations and industry. His menu of achievements include the AACC Outstanding Scientific Achievements by a Young Investigator Award and the Canadian Society of Clinical Chemists Research Excellence Award. He is a member of the newest NACB/AACC LMPG platform on Cardiac Markers. Perhaps the only metric exceeding his proportional bias is his systematic bias for scientific literature, where he is currently the Editor-in-Chief for Clinical Biochemistry.

**Disclosure:** Speaker has disclosed grant/research support from Abbott Diagnostics, Beckman Coulter, Ortho-Clinical Diagnostics, and Roche Diagnostics; consultant fees from Abbott Diagnostics, Beckman Coulter, CADTH, Roche Diagnostics and Siemens; honorarium/expenses from Abbott Diagnostics, Beckman Coulter, Randox and Roche Diagnostics.

### David Morrow, MD, MPH
**Brigham and Women’s Hospital, Boston, MA**

Dr. Morrow is the Director of the Samuel A. Levine Cardiac Unit in the Division of Cardiovascular Medicine at Brigham and Women’s Hospital and a Professor of Medicine at Harvard Medical School. Dr. Morrow is a Senior Investigator in the Thrombolysis in Myocardial Infarction Study Group at Brigham and Women’s Hospital with a research focus in the management of unstable and stable coronary artery disease, and he directs the TIMI Biomarker Program.

He is an internationally recognized expert in risk stratification in patients with ischemic heart disease. He has served on the 2007 National Academy of Clinical Biochemistry (NACB) Laboratory Medicine Practice Guidelines Committee on Biochemical Cardiac Markers for which he led the clinical section on acute coronary syndromes, and on the Program Committee for the American Heart Association Council on Clinical Cardiology. He is a member of the Global Task Force for a Universal Definition of Myocardial Infarction and of the American College of Cardiology/American Heart Association Guidelines Committee for Management of ST-elevation Myocardial Infarction. He sits on the editorial boards of the American Heart Journal, Circulation, Clinical Chemistry, and the Journal of the American College of Cardiology. He is a special section Co-Editor for Continuing Medical Education at Circulation. He has been the recipient of the Lerner Young Investigator Award, the William W. Parmley Young Author Achievement Award, and the Eugene Braunwald Teaching Award. Dr. Morrow has more than 200 original scientific reports, reviews, editorials, book chapters and electronic publications in his areas of expertise.

**Disclosure:** Speaker has disclosed grant/research support from Abbott Diagnostics, Amgen, Astra-Zeneca, Beckman Coulter,
BG Medicine, Daiichi Sankyo, Eli Lilly and Co, Esai, Glaxo Smith Kline, Johnson & Johnson, Merck and Company, Novartis Pharmaceuticals, Ortho-Clinical Diagnostics, Randax, Roche Diagnostics and Singulex; consultant/advisory board with Abbott Laboratories, AstraZeneca, diaDexus, Eli Lilly, Gilead, GlaxoSmithKline, Merck, Novartis, Radiometer, and Roche.

L. Kristin Newby, MD
Duke University, Durham, NC

Dr. Newby is a Professor of Medicine in the Division of Cardiology and also serves as Co-Director of the Cardiac Care Unit at DUMC. Dr. Newby received her MD from the Indiana University School of Medicine and completed a residency in internal medicine and a fellowship in cardiology at DUMC. Dr. Newby serves as co-Principal Investigator of the MURDOCK Study Community Registry and Biorepository and Principal Investigator of the Horizon 1 MURDOCK Study cardiovascular disease project.

Dr. Newby's general research interests include risk stratification and treatment of patients with acute and chronic coronary artery disease and systems issues for delivery of care to patients with these illnesses. She has led the DCRI coordinating center for several randomized clinical trials of new therapies and treatment strategies for acute coronary syndromes. She also has been the principal investigator of multiple studies assessing the use of novel protein biomarkers to enhance risk stratification and guide treatment selection in cardiovascular disease, and she is pursuing interests in the application of genomics for this purpose. She was co-investigator in the GeneQuest study of genetic associations with risk for early onset coronary disease, and she is a co-investigator in several ongoing projects exploring the use of RNA expression profiling, proteomics and metabolomics for risk stratification for coronary events. She is a member of the Steering Committee for the Duke CATHGEN biorepository collecting DNA, RNA and plasma from patients undergoing cardiac catheterization at Duke Hospital for use in ongoing and future genomic studies in cardiovascular disease.

Disclosure: Speaker has disclosed grant/research support from Bristol Myers, Glaxo Smith Kline; consultant fees from AstraZeneca, BioKier, Bristol Myers, CardioDX, Merck, Philips, Roche; board/committee membership/advisory board for Merck, Roche Diagnostics, AstraZeneca and JAH; honorarium/expenses from AstraZeneca, BioKier, CardioDX, Cubist Pharmaceuticals, Eli Lilly, Genentech, INC Research, Janssen Pharmaceuticals, Medscape LLC, Merck, NIH, Philips and Roche.

Dr. Newby will discuss, in generic terms, specific products/services from the companies listed above.

W. Frank Peacock, MD
Baylor College of Medicine, Houston, TX

Dr. Peacock is a Professor of Emergency Medicine, Associate Chair, and Research Director for Emergency Medicine at the Baylor College of Medicine, in Houston, Texas. He is currently on the Board of Directors, Chair of the publication committee, and has served as past President of the Society of Cardiovascular Patient Care (formerly known as the Society for Chest Pain Centers).

With over 400 peer reviewed publications on heart failure and acute coronary syndromes, Dr. Peacock is also the co-editor of the textbooks Cardiac Emergencies, Short Stay Management of Heart Failure, and Short Stay Management of Chest Pain. He is the 2004 and 2010 winner of the Best Research Paper Award from the American College of Emergency Physicians, and is the Codman Award recipient from the American Association of Group Practice.

Dr. Peacock has and continues to serve as PI or co-PI for many high profile national and international clinical trials such as PRONTO, ADHERE-EM, IMPACT, CHOPIN, CLUE, FASTTRACK, and TRUE-HF. Dr. Peacock's research focus is that of cardiovascular emergency medicine, and includes acute coronary syndrome, acute heart failure, venous thromboembolic disease, atrial fibrillation, biomarkers, improvements in emergency medical care and more rapid patient disposition. Dr. Peacock received his medical degree from Wayne State University Medical School and completed his Emergency Medicine training at William Beaumont Hospital, Detroit, Michigan.

Disclosure: Speaker has disclosed grant/research support from Alere, Cardiorentis, Janssen, research grants from Abbott, Alere, Banyan, Cardiorentis, Janssen, Portola, Pfizer, Roche, The Medicine's Company, ZS Pharma; consulting fees from Beckman, Boehringer-Ingelheim, Instrument Labs, Phillips, Portola, Prevencio, Singulex, The Medicine's Company, Alere, Cardiorentis, Janssen, ZS Pharma; Board Membership with Comprehensive Research Associates LLC, Emergencies in Medicine LLC

Alan Wu, PhD
University of California, San Francisco, CA

Dr. Wu is Chief of Clinical Chemistry and Toxicology at San Francisco General Hospital and Professor of Laboratory Medicine, University of California, San Francisco. He received B.S. degrees in chemistry and biology at Purdue University, West Lafayette, Indiana, and a Ph.D. degree in analytical chemistry at the University of Illinois, Champaign-Urbana, Illinois. He completed a postdoctoral fellowship in clinical chemistry at Hartford Hospital. He is certified by the American Board of Clinical Chemistry in Clinical Chemistry and Toxicological Chemistry.

Disclosure: Disclosed no relevant financial relationships.

Information about faculty disclosures:

All individuals involved in planning and developing the content of the presentations were required to submit a disclosure form.

All disclosure information was reviewed during the planning of this program. If any conflicts of interests were identified, they were resolved prior to this event.
Clinical Study to Validate the Use of a New Point of Care BNP Test as an Aid in the Diagnosis of Heart Failure

M. M. Murakami1, K. M. Smith1, P. R. Stack1, B. J. Kilburn1, R. Ler1, B. Amundson1, L. Lilja2, J. Melin2, P. S. Apple1. 1Hennepin County Medical Center, Minneapolis, MN, 2Fiomi Diagnostics, Uppsala, Sweden

Background: The Trinity Biotech Meritas BNP single-epitope assay for detecting BNP is a Point of Care test used in conjunction with the Meritas Analyzer for quantitative determination of BNP in whole blood or plasma to aid in the diagnosis of heart failure (HF).

Objective: The objective of this study was to validate the clinical performance of the Meritas BNP test (Trinity Biotech) for the quantitative determination of BNP for use as an aid in the diagnosis of heart failure (HF).

Methods: The study was designed as a retrospective study of banked EDTA plasma from 665 eligible adult subjects (281 females, 384 males) with a diagnosis of HF. The diagnosis was based on the NYHA classification I-IV. The normal range was determined using banked EDTA-plasma from 1424 non-HF patients (822 females, 602 males), including individuals with comorbidities such as diabetes, hypertension, chronic obstructive pulmonary disease (COPD) and renal disease.

Results: A box and whiskers plot of the clinical study population, classified according to NYHA, is presented in Figure A. A progressive increase in BNP concentrations with increasing NYHA classifications shows a relationship between the severity of the clinical signs and symptoms of HF and the median BNP concentrations of each NYHA class. The diagnostic sensitivity and specificity using a decision threshold of 100 pg/mL (ng/L) for various age groups (< 45, 45-54, 55-64, 65-74, 75+ years) within each gender were as follows: male: sensitivity, range 64 to 79%, specificity, range 81 to 100%; female, sensitivity, range 63 to 79%, specificity, range 81 to 100%; The ROC curve area for HF based on BNP was 0.938 (95%CI 0.927-0.949).

Conclusion: The data indicate that BNP measurements provide objective information for use in the diagnosis of heart failure. The sensitivities and specificities were determined to be acceptable according to the performance claims.

Endothelin Assay in Development for the Sgx Clarity™ Single Molecule Counting Platform Detects ET in Plasma and Discriminates Chronic Heart Failure from Healthy Donor Samples


Background: The Sgx Clarity System, a fully-automated in vitro diagnostics (IVD) platform that utilizes Singulex’s Single Molecule Counting (SMCTM) technology, is in development. The new system will be a clinical diagnostics instrument which uses the same detection concept as the widely used and accepted Research Use Only ERENNA® instrument. The ERENNA has been shown to identify low-abundance biomarkers with unparalleled sensitivity, precision, and accuracy. The Sgx Clarity System utilizes a second generation scanning-based detection system which maintains the exquisite sensitivity of the ERENNA while improving upon the throughput, dynamic range, and usability of that system. Endothelin (ET) is a low abundance biomarker in plasma, and since its discovery in 1989, plasma ET has been studied as a biomarker for CVD risk stratification and for developing heart failure (HF). However, the very low endogenous concentration of the biologically active ET peptide in plasma has made such studies difficult, and to date, there is no IVD platform that tests for ET in plasma.

Objectives: To assess the preliminary performance characteristics of the ET assay in development for the Sgx Clarity System, and to compare ET values obtained from the plasma of chronic heart failure (CHF) patients compared to those obtained from the plasma of apparently healthy donors (normals).

Methods: 44 CHF vs. normal donor plasma samples were tested with the Sgx Clarity ET assay on an Sgx Clarity prototype instrument. Protocols based on CLSI guidelines were followed for analytical performance assessment. Results were compared for statistical significance using the Wilcoxon rank sum test and were displayed in Box-and-Whiskers plots.

Results: The ET assay had an LoB of 0.25 pg/mL and an LoD of 0.44 pg/mL as calculated using StatsProTM software. The 20% functional sensitivity was determined to be 1.0 pg/mL and the 10% functional sensitivity was 1.8 pg/mL as determined using precision profile analysis. Both Within-Run and Total Precision were ≤ 8% CV at concentrations of 1.2 pg/mL and above. The reportable range up to 25 ng/mL demonstrated > 5 logs of dynamic range, and the assay was linear down to the lowest concentration tested (0.3 pg/mL). When tested for hook effect, none was observed up to 500 ng/mL. No significant impact from common endogenous interferences was observed when tested at concentrations recommended in EP-7A2. ET was detected in 100% of normal and CHF plasma samples tested and a significant difference (p<0.0001) was observed between normal and CHF samples.

Conclusion: The ET assay in development for the Sgx Clarity System demonstrates >5 logs of dynamic range, has sufficient sensitivity to detect ET in 100% of samples from apparently healthy donors, and effectively discriminates CHF from normals.
Heart-type Fatty Acid Binding Protein as a Marker of Ischaemia in Patients Undergoing Percutaneous Coronary Intervention

Objectives: To evaluate the potential of heart-fatty acid binding protein (HFABP) as a marker of myocardial ischaemia.

Methods: Percutaneous coronary intervention (PCI) was used as a model for myocardial ischaemia. Fifty patients undergoing planned or elective PCI were prospectively enrolled. Blood samples were taken pre and post balloon inflation and 3 hours after the procedure. Samples were allowed to clot, the serum separated and stored at -20° C until analysis as a batch. Heart-fatty acid binding protein was measured using the Advia 2400 analyser (Siemens healthcare diagnostics). The assay range is 0.747 to 120 ng/mL with a 6.85% CV at 5.47 ng/mL and a 99th percentile of 6.32 ng/mL. Cardiac troponin I was measured on the Advia Centaur with an analytical range of 1.7 to 50000 ng/L. The CV is stated as 10% at 30 ng/L with a 99th percentile of 50 ng/L.

Results: There was no statistical difference in HFABP pre and post balloon inflation, but HFABP was significantly higher at 3 hours (p<0.05). HFABP was significantly higher in patients who were troponin positive (defined as troponin >50 ng/L or showing a dynamic change) versus those who were troponin negative. There was no significant difference in HFABP in men versus women or those that have had previous PCI procedures. There was no correlation between HFABP and age of patient, number of balloon inflations or total inflation time.

Conclusion: HFABP is not a marker of ischaemia. However, HFABP results were consistent with paired troponin results indicating that it is a marker of cardiac necrosis. This study indicates the ongoing need for cardiac markers for ischaemia and non-MI events.

Evidence-based diagnostic decision limits for cardiac troponin for the biochemical diagnosis of acute myocardial infarction in routine clinical practice.

Objective: To assess current use of evidence-based decision limits for cardiac troponin for the diagnosis of acute myocardial infarction (AMI) in Europe.

Methods. And of current practice in 2013-14 was performed using a web-based questionnaire by distribution to European biochemical societies for circulation to their membership. Questions covered cardiac biomarkers measured, analytical methods used, decision thresholds and their derivations. Results were collated using a central database and analysed using comparative and descriptive nonparametric statistics.

Results. Returns were obtained from 442 hospitals, 50% Central or University hospitals and 39% from District (Community) hospitals from 35 countries. 395/442 (89%) provided an acute service 11% were non acute laboratories. In 98.6% troponin measurement was the preferred biomarker for diagnosis of AMI.

The decision limit for diagnosis was based on assay imprecision in 71/441 (16.1%) with the 10% CV in 61 (13.8) and the 20% CV in 10 (2.3%). The 99th percentile was used in 196 (44.3%) on optimised decision thresholds from receiver operating characteristic curve analysis in 5 (1.1%) and a local decision in 104 (23.5%). No data was available for 66 (15%). The choice of value for the decision limit was derived from the manufacturers package insert in 244 (55.2%) from peer-reviewed literature, national or international recommendations in 68 (15.5%) and from locally-based consensus review in 80 (18.1) %. No data was available for 42 (9.5%) and 3 laboratories reported they did not use a decision limit.

A detailed analysis of the decision limits used was performed for the Roche diagnostics high sensitivity troponin T (n = 183) and the largest single troponin I group, the Abbott diagnostics standard assay (n = 84). For troponin T the 99th percentile is 14 ng/L, 10% CV of 13. Only 92 (50.3%) of laboratories were using the 99th percentile for cardiac troponin T recommended. The decision limit used varied from 2 ng/L to 700 ng/L with peaks of utilisation at14 ng/L, 30 ng/L, 50 ng/L and 100 ng/L. For the 10% CV a value of 14-100 ng/L was reported and for the 99% percentile 14-400 ng/L. For the Abbott assay (99th percentile 28 ng/L) 10% CV 32 ng/L 7 (8.3%) used 28 and 3 (3.6%) 32. The range used was from 25 ng/L to 500 ng/L with peaks at30 ng/L, 40 ng/L and 300 ng/L. The 10% CV was reported as 28-500 ng/L and the 99th percentile 28-300 ng/L.

Conclusion: There is currently a lack of understanding of the decision thresholds and their derivation which should be in routine clinical use for they diagnosis of acute myocardial infarction using cardiac troponin measurement. Recent publications show that this lack of understanding will result in under diagnosis of preventable disease.

Relative contribution of high sensitivity cardiac Troponins I and T in cardiovascular risk stratification in patients with OSA after treatment with CPAP

Background: Obstructive sleep apnea (OSA) is a common condition caused by intermittent airway collapse during sleep that results in repetitive hypoxia, arousal, poor quality of sleep and excessive daytime sleepiness. OSA is a risk factor for various cardiovascular conditions and in recent decades, OSA was associated with increased cardiovascular mortality in patients with the severe form of the disease without treatment. Therefore, adequate treatment with CPAP (Continuous Positive Airway Pressure) may improve survival. With the advent of new high-sensitivity markers, there was an increase in the sensitivity of the method consequently increased interest from professionals in the use these high-sensitivity cardiac troponin T and I (hs cTnT and hs cTnI) in risk stratification of cardiovascular diseases since they are released in the cardiac lesions. The aim of our study was to evaluate the (hs cTnT and hs cTnI) methods and evaluate of cTnI conventional by two methods in patients with severe OSA before and after one year of effective treatment with CPAP.

Methods: 36 patients of the Sleep Institute in Sao Paulo, with moderate and severe OSA, 22 men, with mean BMI = 30.20 ± 9.12
Kg/m² and age = 65.4 ± 5.8 years, without other diseases were randomized and treated effectively with a year with CPAP and using average of 5 hours each night, non-smoking and sedentary. Patients collected 10 ml of venous blood before treatment and after treatment with CPAP which were frozen at -80°C and was thawed on laboratory measurements. Between several biochemical parameters will be analyzed the hs cTnT by two methods and cTnI conventional by two methods too, before and after one year treatment to apnea with CPAP. The hs cTnT was quantified with an Electrochemiluminescence (hs cTnT) Elecsys® Roche/Elecsys® third generation method. The detection limit is 0.5 pg/mL. Other method was used hs cTnI immunoassay (Abbott/ARCHITECT system). It was measured cTnI ES conventional by chemiluminescence (Vitros® - Ortho Clinical Diagnostics) 12 pg/mL is the limits of detection to this methods and cTnI by Access cTnI Dxs (Access/Beckman Coulter) with detection limits of 10 pg/mL for this method. Paired samples statistics were performed for comparisons and evaluations of results between methods before and after treatment with Wilcoxon test was performed to methods with significance differences.

Results: Based on the results presented there was a significant effective treatment with CPAP on the hs cTnT presented in the nonparametric statistical test Wilcoxon (Z=1.955, p= 0.05 and Z=1.634, p=0.04). However there was not significant difference of treatment with CPAP on the values presented by cTnI conventional dosages to both methods.

Conclusion: The hs cTnT and hs cTnI methods showed significance differences between before and after treatment with one year of CPAP in patients with apnea. This method use monoclonal antibodies with high sensitivity and specificity for cardiac injury. However there is a need of definition about the real importance of these low levels found in the condition of obstructive sleep apnea for cardiac injuries. The cTnI ES and cTnI Dx did not show sufficient sensitivity on condition of OSA.

Poster Abstract 6

Increased Incidence of Cardiac Troponin I Abnormalities in Women Utilizing a High Sensitivity Assay


Objective: To determine if sex-specific 99th percentiles with a high sensitivity cardiac troponin I (hs-cTnI) assay versus a single non-sex cut-off using a contemporary cTnI assay leads to more frequent increases in cTnI levels indicative of acute myocardial infarction (MI)

Methods: The data presented are the first results from our ‘clinical trials.gov identifier: NCT02060760’ study. Patients, 18+ years of age, presenting to the emergency department where providers used cTnI to rule-in and rule-out MI, were included in the study. Serial cTnI measurements were obtained on clinical indication between February 4 and March 13, 2014. Clinical decisions were based on the contemporary cTnI results, with hs-cTnI measured simultaneously (both on the Abbott ARCHITECT i1000® or i2000®). 99th percentiles were as follows: contemporary cTnI 30 ng/L (0.030 μg/L); hs-cTnI 16 ng/L for females and 34 ng/L for males.

Results: 792 patients presenting for MI rule-in or rule-out were enrolled, of which 45% were female. Over the course of serial cTnI measurements (0.3.6.9h) baseline and maximum values were examined by gender. At presentation mean (95% CI) values were: cTnI assay 128 (0-266) ng/L for males and 60 (31-89) ng/L for females; hs-cTnI assay 90 (0-190) ng/L for males and 45 (18-72) ng/L for females. Maximum values were also examined by gender: cTnI male 532 (126-938) ng/L female 241 (52-430) ng/L; hs-cTnI male 457 (135-781) ng/L; 236 (29-444). The hs-cTnI assay resulted in a 15% decrease (p=0.01) in patients with at least one value greater than the sex-specific cut-off. The number of women with an increase above the 99th percentile cut-off was significantly different (p=0.003) vs. males. Further, the hs-cTnI assay sex-specific cut-offs resulted in a 29% decrease in males with an increased value and a 5% increase for females with an increased value.

Conclusion: Based on sex-specific hs-cTnI assay 99th percentiles we observed a significant decrease in the incidence of cTnI increases and a significant difference in increased rates between sexes. The increased incidence of cTnI increases for women using an hs-cTnI assay could have important implications for improving treatment and outcomes for women presenting with symptoms of acute coronary syndromes.

Validation and Correlation study of the Values for the Beckman cTnI and I+3 Assay on the DxI 800 and Access-2 Analyzers.


Background: Cardiac troponin (cTn) assays have been available in clinical laboratories for nearly two decades and considered a highly sensitive marker for myocardial damage. An elevation of cTn I is used, together with other diagnostic criteria, to rule in/out a myocardial infarction (MI). Laboratories measure either cTnI or cTnT isoforms of troponin. Following a recall of cTnI reagents from the Dxl Immunoassay analyzer in October 2010, Beckman Coulter (BC) recently re-introduced a Troponin-I (Access-I+3) assay for the DxI 800 and Access-2 analyzers.

Design: Method validation studies (correlations, linearity, intra- and inter-precision studies) were performed by comparing the following cTnI results (new reagent vs old reagent) on the following analyzers: 1. The Access-2 analyzers in the Emergency Department (ED) and the Main Laboratory (ML); 2. Access-2 in the ED and DxI 800 in the ML; 3. Access-2 ML and Dxl 800 ML. A total of 115 patient specimens, presenting to our Emergency Department (ED) with history or evidence of cardiac disease or cTnI ordered following a review of patient’s chart, were used for correlation studies. Specimens were spun, aliquoted, frozen within 24 hours at -20°C, and analyzed within 30 days of collection. Precision was performed using BioRad Cardiac Marker Plus Quality Control (Levels 1, 2, 3). Linearity was performed using BC calibrators.

Results:

Table 1

<table>
<thead>
<tr>
<th>Analyzers</th>
<th>No.</th>
<th>Mean x/y</th>
<th>Slope</th>
<th>Intercept</th>
<th>Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Access-2 ED/ML</td>
<td>109</td>
<td>0.117/0.108</td>
<td>0.981</td>
<td>-0.008</td>
<td>0.9939</td>
</tr>
<tr>
<td>Access-2 ED/DxI800</td>
<td>115</td>
<td>0.12/0.111</td>
<td>0.791</td>
<td>0.015</td>
<td>0.9776</td>
</tr>
<tr>
<td>Access-2 ML/DxI800</td>
<td>115</td>
<td>0.131/0.111</td>
<td>0.775</td>
<td>0.009</td>
<td>0.9589</td>
</tr>
</tbody>
</table>

Conclusions: The validation studies showed that the new assay demonstrated good precision and correlation between the old and new reagents. The results also showed an extended reportable range (previously reported), allowing our laboratory to report results as low as 0.04 ng/mL (previously reported using the old reagents only down to 0.4 ng/mL). By increasing the sensitivity of the assay, earlier detection of an MI may be potentially achieved.
**Reference Range Study Using High Sensitivity cTnI Assay in Development for the Sgx Clarity™ Single Molecule Counting Platform**

L. Shephard, S. Florey, Y. Cheung, R. Livingston, L. Monsalve, J. Todd, J. Bishop, J. Felberg, Singulex, Alameda, CA

**Background:** The Research Use Only (RUO) cardiac Troponin I (cTnI) assay developed by Singulex for the ERENNA® instrument provided the first evidence that cTnI is measurable in almost all healthy subjects. With that knowledge, it has become clear that cTnI is not only a valuable marker for diagnosing acute myocardial infarction, but it may also be useful as a prognostic indicator for underlying heart disease. Singulex is now developing the Sgx Clarity System, a fully-automated in vitro diagnostics platform that, similar to the ERENNA system, utilizes Single Molecule Counting technology. The new system will be a clinical diagnostics instrument utilizing a second generation scanning-based detection system which has equal sensitivity to the ERENNA with an improved throughput and an extended dynamic range. The cTnI assay in development for the Sgx Clarity System is based on the 2 + 2 antibody concept introduced by HyTest, with two capture and two detection antibodies. One antibody of each pair recognizes an epitope in the stable central portion of the cTnI molecule, while the second recognizes an epitope in the N- or C-terminal region, thus conferring overall robustness to known cTnI-specific interferences.

**Objectives:** To estimate the 99th percentile of cTnI values in apparently healthy subjects on the Sgx Clarity system using a set of reference range samples and to assess preliminary performance characteristics of the assay.

**Methods:** For the reference range study 120 male and 120 female EDTA plasma samples from self-declared healthy donors were collected from five states across the US. The 99th percentile was calculated using a nonparametric method using StatisPro™ software and gender specific results were compared. Additional performance characteristics were assessed according to protocols based on CLSI guidelines where available.

**Results:** The 99th percentiles were 13.38 pg/mL and 4.90 pg/mL for male and female donors, respectively. The 99th percentile for the overall population was 11.11 pg/mL. Based on this analysis, the recommendation for gender specific reference ranges should be considered. A precision profile was generated from the duplicate samples less than 1 pg/mL in the reference range study, and the within-run 10% and 20% functional sensitivities were calculated at 0.31 and 0.13 pg/mL, respectively. Troponin values were quantifiable above the 20% functional sensitivity in 100% of samples tested. The reportable range goes up to 25 ng/mL with no high dose hook effect up to 1000 ng/mL, thus giving the assay a 5 log linear dynamic range. No significant impact from common endogenous interferences was observed and the assay formulation was shown to be robust against common cTnI-specific interferences such as heparin, proteolytic cleavage, phosphorylation, and cTnI-C complex. Furthermore, minimal cross-reactivity was observed when tested with 1000 ng/mL of sTnI (0.019%), cTnT (0.03%) and TnC (0.00005%).

**Conclusion:** The cTnI assay in development for the Sgx Clarity System demonstrates sensitivity and precision that is equivalent to the ERENNA RUO assay, sufficient to quantify cTnI in 100% of apparently healthy donor samples. This sensitivity allows for the determination of gender specific differences in the 99th percentile of Normal cTnI values.

---

**BNP Assay in Development for the Sgx Clarity™ Single Molecule Counting Platform Demonstrates Increased Clinical Sensitivity Relative to a Conventional BNP Immunoassay**

X. Wang, S. Tjon-Kon-Sang, V. Torres, R. Livingston, L. Monsalve, J. Todd, J. Felberg, J. Bishop, Singulex, Alameda, CA

**Background:** Singulex is developing the Sgx Clarity System, a fully-automated in vitro diagnostics platform that uses Singulex’s Single Molecule Counting technology and has sensitivity equivalent to the widely used and accepted Research Use Only ERENNA® instrument while improving upon the throughput, dynamic range, and usability of that system. The BNP assay in development for the Sgx Clarity System uses the single epitope sandwich (SES) concept introduced by HyTest - where a capture antibody recognizing an epitope in the stable ring structure is paired with a detection antibody that binds only to the complex of the detection antibody-bound BNP molecule. The SES concept is hypothesized to confer a higher apparent stability of BNP in patient plasma, since much of the circulating BNP is truncated at the N- and C-termini, which are the epitope targets of most conventional commercially available BNP immunoassays.

**Objectives:** To assess the preliminary performance characteristics of the Sgx Clarity BNP assay and to compare the clinical performance to that of a conventional BNP immunoassay.

**Methods:** Forty plasma samples spanning the reportable range were tested on the Sgx Clarity BNP assay as well as the Siemens ADVIA Centaur BNP assay. Analytical performance studies followed protocols based on CLSI guidelines.

**Results:** An LoB of 0.6 pg/mL, an LoD of 1.4 pg/mL, and a 10% functional sensitivity (LoQ) of 3.4 pg/mL were obtained. Within-Run Precision was 3 - 8% and Total Precision 4 - 10% with plasma samples from 4.5 to 1130 pg/mL BNP. The assay was linear throughout the reportable range, which extends to 5000 pg/mL, and there is no high dose hook effect up to 100,000 pg/mL. No notable impact from common endogenous interferences and minimal cross reactivity was observed when other natriuretic peptides were tested. Passing-Bablok regression demonstrated good agreement between Sgx and Siemens methods (slope 1.09; Pearson correlation 0.97). Two samples, which had reported BNP concentrations of 220 and 297 pg/mL by the Siemens assay, had much higher results (1343 and 926 pg/mL) with the Sgx assay. A similar finding was observed in a subsequent study, with a small but significant percentage of discordant samples being observed. In all cases, the discrepant results were higher on the Sgx Clarity BNP assay. To further investigate this discrepancy, all samples were assayed for NT-proBNP (Roche). Those results showed clinical agreement with the Sgx Clarity BNP results relative to the established clinical cutoffs for the two molecules. This observation supports the hypothesis that some percentage of circulating BNP is not detected by conventional immunoassays whose antibodies bind to epitopes near the unstable termini of the BNP peptide. If true, the clinical relevance of this hypothesis requires further investigation.

**Conclusion:** These results support the hypothesis that the Sgx Clarity BNP assay using the SES antibody concept may be more clinically sensitive than conventional BNP assays to circulating forms of BNP in patient plasmas.
High-sensitivity cardiac troponin I in a large community-based population at risk for cardiovascular disease

A.H. Wu1, J. Estis1, P. Helestine1, K. Bu2, J. Todd2, P. Kavask3. 1University of California, San Francisco, San Francisco, CA, 2Singulex, Inc., Alameda, CA, 3McMaster University, Hamilton, ON, Canada

Background: With the development of high-sensitivity cardiac troponin assays and the ability to measure cardiac troponin in essentially all healthy individuals, comes the possibility to use this assay for risk stratification for future cardiovascular disease (CVD).

Objective: To determine plasma levels of hs-cTnI and identify the impact of co-morbidities on cTnI concentrations in a large at CVD risk population.

Methods: We previously reported the development of a high-sensitivity Single Molecule Counting assay to quantify plasma cTnI. This assay was further developed into a laboratory developed test and offered in a CLIA licensed, CAP accredited laboratory with a LLOQ of 0.4 pg/mL and the (95%tile upper limit of normal (ULN) of 4.6 ng/L. Blood samples were measured for hs-cTnI, LDL, HDL and HbA1c in 23,832 (56% female) community-based patients at risk for CVD. Parametric and non-parametric analyses were performed in de-identified data using SAS V9.3.

Results: The distributions of cTnI in the study population are shown in Table. cTnI was quantifiable in 88% patients. Seven percent of the population was above the ULN cutoff. cTnI was significantly higher in males > 50 yrs, males, as well as those with CVD risk factors of pre-diabetes, diabetes and HDL dyslipidemia, with those patients having HbA1c >5.9% having the highest concentrations.

Conclusions: This is the largest community cohort study assessing hs-cTnI results

<table>
<thead>
<tr>
<th>hs-troponin results</th>
<th>Median, ng/L</th>
<th>95th%, ng/L</th>
<th>99th%, ng/L</th>
<th>%&gt;99th%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Entire cohort</td>
<td>0.9</td>
<td>6.0</td>
<td>21.5</td>
<td>7.0</td>
</tr>
<tr>
<td>Females</td>
<td>0.8</td>
<td>4.6</td>
<td>15.5</td>
<td>4.9</td>
</tr>
<tr>
<td>Males</td>
<td>1.2</td>
<td>7.6</td>
<td>27.0</td>
<td>9.4+</td>
</tr>
<tr>
<td></td>
<td>0.6</td>
<td>3.4</td>
<td>10.9</td>
<td>3.1</td>
</tr>
<tr>
<td>&gt;=50 y</td>
<td>1.1</td>
<td>7.0</td>
<td>24.4</td>
<td>8.5+</td>
</tr>
<tr>
<td>HbA1c &lt;=5.9%</td>
<td>0.9</td>
<td>5.1</td>
<td>17.6</td>
<td>5.8</td>
</tr>
<tr>
<td>HbA1c &gt;5.9%</td>
<td>1.3</td>
<td>9.1</td>
<td>34.6</td>
<td>11.3+</td>
</tr>
<tr>
<td>LDL &lt;=129 mg/dL</td>
<td>1.0</td>
<td>6.4</td>
<td>22.3</td>
<td>7.6</td>
</tr>
<tr>
<td>LDL &gt;129 mg/dL</td>
<td>0.9</td>
<td>5.8</td>
<td>20.1</td>
<td>6.7</td>
</tr>
<tr>
<td>HDL at target*</td>
<td>0.9</td>
<td>5.8</td>
<td>20.1</td>
<td>6.8</td>
</tr>
<tr>
<td>HDL less than target*</td>
<td>1.0</td>
<td>7.5</td>
<td>25.4</td>
<td>8.6+</td>
</tr>
<tr>
<td>HDL target</td>
<td>&gt;=45 mg/dL females</td>
<td>&gt;=35 mg/dL males</td>
<td>+P&lt;0.0001</td>
<td></td>
</tr>
</tbody>
</table>

Automated approaches to wrangling wayward troponins

D.S. Herman, G. S. Baird, D. N. Greene. University of Washington, Seattle, WA

Background: Quantification of cardiac troponin I (cTnI) serum concentration is a critical part of evaluating patients for acute myocardial infarction. Errors in this quantification can lead to significant clinical mismanagement. To mitigate this risk, we sought to quantify the frequency of false-positive cTnI results and develop strategies to prevent them.

Methods: We reviewed clinical cTnI results (N=22,976; 1/21/2014 - 11/20/2014) performed using the Beckman Coulter AccuTNI+3 assay across two academic medical centers. cTnI results for individual patients were grouped and then serial tests were compared based on their resulted concentrations and collection times.

Results: Retrospective analyses of the temporal patterns of cTnI concentrations for individual patients revealed decay rates that were faster than physiologically expected and not explained by other clinical factors. Focusing on results > 0.04 ng/mL with follow-up testing within 24 hours (N=6,205; 27%), we developed specific criteria (t1/2 < 3.7 hours; %Δ[cTnI] ≤ -25%; 1st [cTnI] > 0.05 ng/mL; 2nd [cTnI] 0.03 ng/mL; (B) rapid concentration increases (t2 < 4.7 hours; %Δ[cTnI] ≥ 35%), and (C) unexpectedly rapid concentration decreases (t1/2 < 5 hours; %Δ[cTnI] ≤ -20%). Retrospective analyses indicate that the application of these rules to our study data would have triggered repeat assessment in 15% of cTnI tests and identified all of our suspected false elevations. Additionally, we expect this intervention to identify false elevations that our retrospective analyses could not flag because there was no timely follow-up testing.

Conclusions: Retrospective analyses of serial cTnI concentrations have identified a set of potentially falsely elevated results (~1 in 200) with important clinical ramifications. We are leveraging the power of our middleware system to prospectively identify these potential errors in real-time and automatically trigger repeat evaluation. Ongoing studies, including cross-institution and cross-instrument comparisons of the temporal patterns of cardiac troponin concentrations, will help to understand the sources of these potential false-elevations and enable the refinement of strategies to prevent patient harm.
## Instructions for Obtaining Continuing Education Credit for Acute Cardiac Biomarkers Update: Laboratory Guidelines Focused on Clinical Need — A Virtual Conference — Thursday, November 12, 2015

**ACCENT® credit hours**

AACC designates this live webinar activity for a maximum of 5.5 ACCENT® credit hours towards the AACC Clinical Chemist’s Recognition Award. AACC is an approved provider of continuing education (CE) for clinical laboratory scientists licensed in states that require documentation of CE, including California, Florida, Louisiana, Montana, Nevada, North Dakota, Rhode Island, Tennessee, and West Virginia. ACCENT® credit is also recognized by several organizations: AAB, ABCC, ACS, AMT, ASCLS, ASCP, ASM, CAP, IFCC, and NRCC.

**AMA PRA Category 1 Credit™**

The AACC is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians. The AACC designates this live continuing medical education activity for a maximum of 5.5 AMA PRA Category 1 Credits™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

1. **Record the Presentations You View and CE Codes on this Form**
   - As you view each presentation, complete the information requested on this form below, including the CE code, which will be displayed at the end of each presentation.
   - Each presentation is accredited for 0.5 credit.
   - Continuing education credits must be obtained by December 31, 2015

2. **Obtain Your Continuing Education Credits**
   - After you have finished viewing all of the presentations, go to: [https://www.surveymonkey.com/r/acutecardiacmarkers](https://www.surveymonkey.com/r/acutecardiacmarkers)
   - Complete the evaluation survey and after submitting your responses, you will be directed to the AACC website to enter the CE codes and obtain your continuing education certificate.
   - Read through the steps and click on “Next Screen” at the bottom of the screen to proceed to the next screen.
   - Enter each CE code you recorded. The final screen will be your certificate, which you may print out and/or save to your computer.

Questions? If you have questions about continuing education credit, contact the AACC Professional Education department at education@aacc.org.

### Presentation Title
(CE Credit not available for the poster presentations or exhibits.)

<table>
<thead>
<tr>
<th>Presentation Title</th>
<th>CE Code (displayed at the end of each presentation) – be sure to write down this code.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>