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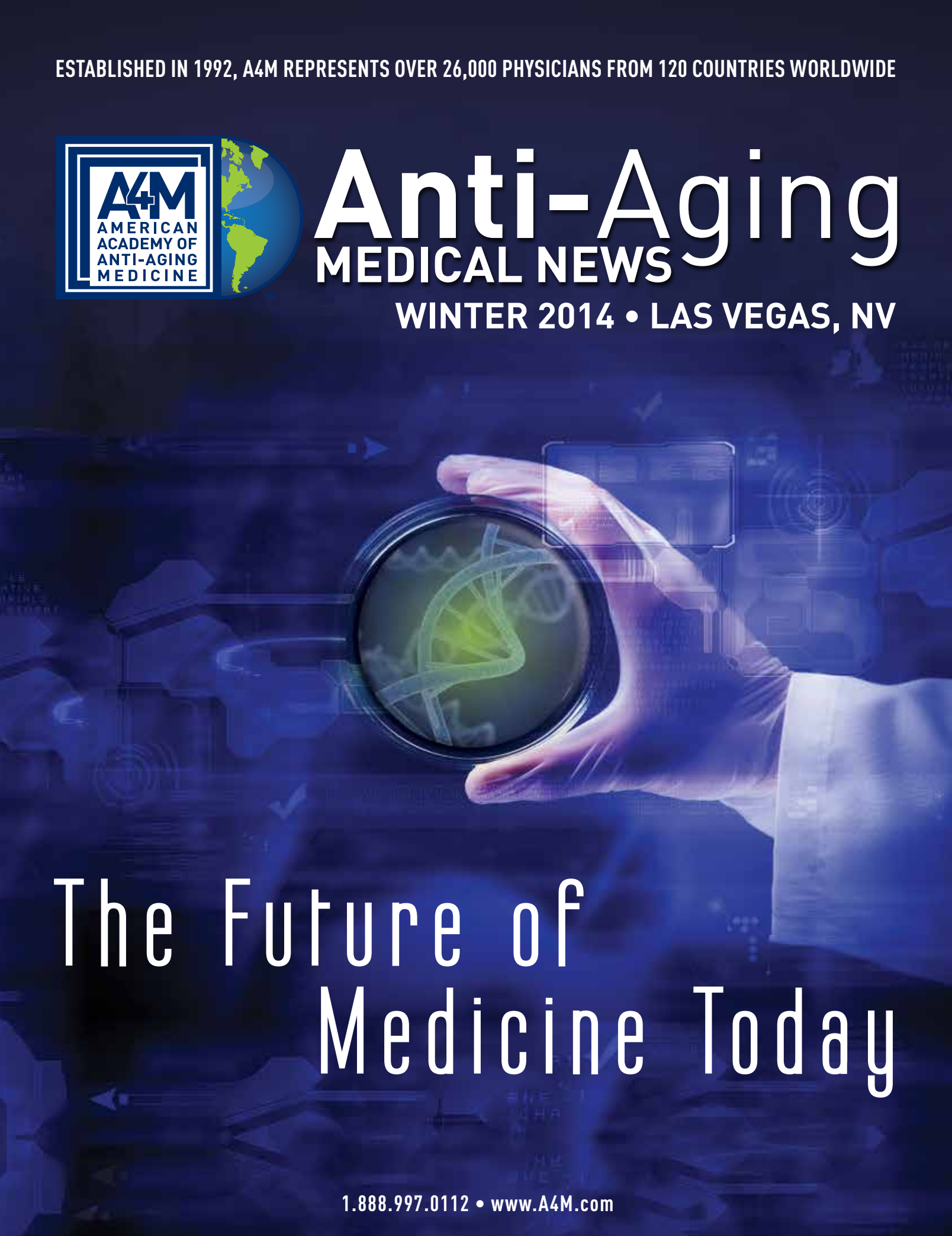
WINTER 2014 • LAS VEGAS, NV



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Distinguished Colleagues,

The American Academy of Anti-Aging Medicine (A4M) welcomes you to Las Vegas for the Winter 2014 session of the 22nd Annual World Congress on Anti-Aging Medicine, interactive workshops and advanced education training programs.

The A4M is proud that our 26,000 physicians, practitioners, and scientific members from 120 nations worldwide have made a lasting and palpable commitment that expands the availability of advanced biotechnologies and leading-edge preventive healthcare throughout the world.

In its third decade of educational service, this organization is dedicated to providing practitioners, from all specialties, with the opportunity to learn in an environment where innovation, invaluable education and scientific research meet.

As pioneers in Anti-Aging Medicine, our conferences offer a wide educational exposure to doctors not only locally and regionally, but also internationally. Anti-Aging Medicine is transforming healthcare, one practice at a time, as a way for patients to adopt a healthy lifestyle based on scientific and academic research in preventive medicine.

It is an exciting time to be involved in Anti-Aging Medicine. With your involvement, the Anti-Aging medical specialty continues to expand and become more widely accessible. By attending this event, you are part of this transformation, and we applaud you for joining this fast-growing movement.

With warm regards,

Ronald Klatz, MD, DO
President, A4M

Robert Goldman, MD, PhD, DO, FAASP
Chairman, A4M

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Schedule at a Glance

22nd Annual World Congress on Anti-Aging Medicine

Wednesday, December 10, 2014

		Presented By:
7:30am-6:00pm	Power of Hormones: An Introduction to Adult Hormone Deficiency	Thierry Hertoghe, MD; Ron Rothenberg, MD; Pam Smith, MD, MPH, MS
7:30am-6:00pm	Optimizing Sexual Function	Jennifer Landa, MD, OB-GYN, FAARFM; Michael Krychman, MD, OB-GYN; Anna Cabeca, DO, FACOG
9:00am-6:00pm	Aesthetic Treatments: Laser & Light Applications	Sharon McQuillan, MD; Denise Baker, MD; Chris Robb, MD; Edward Zimmerman, MD
8:00am-6:00pm	BHPT: Bioidentical Hormone Pellet Therapy	James Mahoney, DO
7:30am-6:00pm	Personalized Lifestyle Medicine: Relevance of Nutrition and Lifestyle Therapies	Andrew Heyman, MD; James LaValle, RPh, CCN, MS; Pam Smith, MD, MPH, MS

Saturday & Sunday, December 13-14, 2014

		Presented By:
1:00pm-5:30pm 12/13/14 8:00am-12:30pm 12/14/14	Reversing Physical Aging: Hormone & Nutritional Therapies	Thierry Hertoghe, MD

Sunday, December 14, 2014

		Presented By:
8:00am-6:30pm	Menopause/Andropause: Improving the Health and Happiness of Your Patients with Bioidentical Hormones	Jonathan Wright, MD; Daved Rosensweet, MD

SCHEDULE, SPEAKERS & TOPICS ARE SUBJECT TO CHANGE • AS OF 11/12/2014 • © A4M 2014

22nd Annual World Congress on Anti-Aging Medicine

Thursday, December 11, 2014 Conference

• Morning General Session •		Presented By:
7:00am-7:35am	Detoxification Lifestyle with Case Study	John Cline, MD
7:35am-8:10am	Effective Treatment for Fibromyalgia & Chronic Fatigue	Jacob Teitelbaum, MD
8:10am-8:50am	Effects of PRP (Platelet Rich Plasma) and Stem Cell Injections for Treatment of Musculoskeletal Conditions in an Office Setting	Joseph Purita, MD
8:50am-9:15am	Opening Remarks (Non-CME)	Ronald Klatz, MD, DO & Robert Goldman, MD, PhD, DO
9:15am-10:20am	Integrative Medicine: A Bridge over Healthcare's Troubled Waters	David Katz, MD, FACM, FACP, MPH
10:25am-11:00am	Understanding the Pivotal Role of the Endocannabinoid System and Cannabinoid Ligands in Aging, Obesity, Heart Disease, Neurodegeneration and Pain	James J. Bradstreet, MD(H), FAAFP
LUNCH (On your own) • 11:00am-1:00pm		
Exhibit Hall Hours • 10:00am-6:00pm		
• Afternoon Session 1 • The Good, the Bad, and the Ugly in the Human Microbiome •		Presented By:
1:00pm-4:00pm	You Internal Rainforest: The Good, the Bad and the Ugly in the Human Microbiome	Todd LePine, MD
• Afternoon Session 2 • Lifestyle Management/Clinical Case Protocols •		Presented By:
1:00pm-1:35pm	Telomere Measurement as a Diagnostic Test in Cardiovascular and Age-Related Disease	Sandy Chang, MD, PhD
1:35pm-2:10pm	Alternative Complementary Medicine Overview: Fight Fat with Fat	John Salerno, MD
2:10pm-2:45pm	Energy Medicine Going Main Stream	Silvia Binder, ND, PhD
2:45pm-3:20pm	The Great Cholesterol Myth	Steven Sinatra, MD
3:20pm-3:55pm	Telomere Shortening & Modulation: Case Studies from the Clinic	Harvey S. Bartnof, MD
3:55pm-4:30pm	Prevention & Treatment of the Aging Brain	Andrew Campbell, MD
• Afternoon Session 3 • 2014 Integrative Medicine Hot Topics •		Presented By:
1:00pm-1:30pm	The Immune Protocol & The Lite LDIPT Protocol: Out-Come Based Investigation 700 Patients – 52 Months	James Wm. Forsythe, MD, HMD
1:30pm-2:10pm	The Role of Bacteria in Anti-Aging Medicine: Nitric Oxide and Beyond	Nathan Bryan, PhD
2:10pm-2:50pm	Pain Management: Topical Compounding Options	Bryan Prescott, PharmD
2:50pm-3:30pm	The Critical Role of the 3-Iodothyronine Deiodinases in the Regulation of the Thyroid System	E. Denis Wilson, MD
3:30pm-4:00pm	The Pathogenesis of Systemic LPS (Metabolic and Diabetes)	Thomas Alexander, MD
• Afternoon Session 4 • Stem Cells and Reprogramming •		Presented By:
1:00pm-1:40pm	PRP: Evidence Based Regenerative Medicine	Joel Baumgartner, MD
1:40pm-2:20pm	Cryopreservation and Culture Expansion of Stem Cells	Rafael Gonzalez, PhD
2:20pm-3:00pm	Repairing Joints and Spine without Surgery: Prolotherapy/PRP/Stem Cell	Peter Fields, MD, DC
3:00pm-3:40pm	Mesenchymal Stem Cells for the Treatment of Musculoskeletal Disease	Lora Brown, MD
3:40pm-4:20pm	Use of Platelet Rich Plasma (PRP) in Penile Enhancements: Technique and Early Results	Keith Jeffords, MD
• Afternoon Session 5 • Advances in Aesthetic Medicine •		Presented By:
1:00pm-1:40pm	Managing Facial Injectable Sequelae	Sharon McQuillan, MD
1:40pm-2:20pm	Overview of Electromagnetic Spectrum Technologies for Medical and Aesthetic Treatments	Edward Zimmerman, MD
2:20pm-3:00pm	The Value of Broad Band Light Delaying Skin Aging: Report of a New Technique	Patrick H. Bitter, Jr., MD
3:00pm-3:30pm	How Menopause Changes the Biology of Skin and the Cosemeceutical Ingredients that Reverse It	Rick Rhoads, PharmD
3:30pm-4:00pm	Non-Surgical Methods for the Enhancement of Facial Beauty and Restoration of the Aging Face	Maria Angelo-Khattar, MD, PhD, MSc Dermatology

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22nd Annual World Congress on Anti-Aging Medicine

Friday, December 12, 2014 Conference

• Morning Session •		Presented By:
7:00am-7:30am	Diabetes, Alzheimer's and Heart Disease	Chris Meletis, ND
7:30am-8:10am	Dementia: Our Destiny or our Choice?	Joseph C. Maroon, MD, FACS
8:10am-8:40am	Benefits of Balance the Gut Microbiome Using Novel Prebiotic Bacteriophage Biotherapeutics	Sharon McQuillan, MD
8:40am-9:20am	Burnout: A Multiple Hormone Deficiency Syndrome	Thierry Hertoghe, MD
9:20am-10:00am	Separating Fact from Fiction: Evidence Based Answers to the Toughest Questions Patients Ask about Nutritional Support	Thomas Guilliams, PhD
10:00am-11:00am	Life at the Speed of Light	J. Craig Venter, PhD
LUNCH (On your own) • 11:00am-1:00pm		
Exhibit Hall Hours • 10:00am-6:30pm		
• Afternoon Session 1 • Moving Beyond Basics •		Presented By:
1:00pm-4:00pm	Functional Medicine Approach to Assessing and Treating Patients with Gastrointestinal and Immune Dysfunction: Moving Beyond the Basics	Jill Carnahan, MD
• Afternoon Session 2 • Lifestyle Management/Clinical Case Protocols •		Presented By:
1:00pm-1:40pm	Physiologic Vigor is determined by Biochemical Balance-Lifestyle Interventions	Shawn Talbott, PhD
1:40pm-2:15pm	Managing Glucose Toxicity and Insulin Resistance	John Troup, PhD
2:15pm-2:50pm	Personalized Genetics: Applying Genomics to General Health, Nutrition, and Lifestyle Modification	T.S. Piliszek, MD, MRCS, LRCP, FAARFM, ABAARM, CNS
2:50pm-3:25pm	Does Nutritional Supplementation with Sun Chlorella "A" Help Overcome Vitamin B12 Deficiency and Enhance IgA Levels in People with a Vegan or Vegetarian Diet?	Jay Udani, MD, CPI
3:25pm-4:00pm	Sugar Impact	JJ Virgin, CNS, CHFS
• Afternoon Session 3 • Transforming Treatments through Assessments and Evaluations •		Presented By:
1:00pm-1:35pm	Steroid Hormone Compartmentalization in Different Body Fluids	David Zava, PhD
1:35pm-2:10pm	Novel Technologies for Supporting Triads and Enhancing Patient Experience	James LaValle, RPh, CCN, MS
2:10pm-2:45pm	Inflammation Testing to Define Heart Disease Risk	Dharmesh Patel, MD, FACC, MBBS
2:45pm-3:20pm	The Impact of Toxic Mold and Mycotoxins on Human Health	Joseph H. Brewer, MD
3:20pm-3:50pm	Moving Beyond Diurnal Free Cortisol Testing: Is there a Need for a more Comprehensive Testing Model for Assessment	Mark Newman, MS
3:50pm-4:10pm	Hormone Replacement Therapy and Testing for Cancer	Emil K. Schandl, MS, PhD, MD(MA), FNACB, SC(ASCP), CC(NRCC), LNC, CLD
• Afternoon Session 4 • Hormones and the Anti-Aging Equation •		Presented By:
1:00pm-1:30pm	Women's Health and Sexuality for Anti-Aging	Anna Cabeca, DO, FACOG, ABAARM
1:30pm-2:00pm	Metabolic Syndrome and Menopause	Erin Lommen, ND
2:00pm-2:30pm	Is it Menopause or Wireless Radiation Sensitivity?	Elizabeth Plourde, CLS, NCMP, PhD
2:30pm-3:00pm	Silicon: A Review of Its Potential Role in the Prevention & Treatment of Postmenopausal Osteoporosis	Charles T. Price, MD
3:00pm-3:30pm	Man Boobs to Metabolic Syndrome	Nathan Goodyear, MD, ABAARM, FAARFM
3:30pm-4:00pm	Peptides and Hormonal Replacement	Jose Vazquez Tanus, MD, ABAARM, BCIM, FAARM, CCRP
• Afternoon Session 5 • Advances in Aesthetics Medicine •		Presented By:
1:00pm-1:30pm	Hair Loss and Hormone Replacement: Stratagems for Managing Androgenetic Alopecia in the Presence of Androgen Replacement	Alan J. Bauman, MD
1:30pm-2:00pm	An Evaluation of Efficacy and Tolerability of Novel Enzyme Exfoliation vs Glycolic Acid in Photodamage Treatment	Anne Chapas, MD
2:00pm-2:30pm	Mild Acidity Promotes Healthy Skin Microflora & Dermal Longevity	Karen Sinclair Drake
2:30pm-3:00pm	Combining New Hybrid Fractional Laser Technology with IPL/BBL Treatments	Rebecca Gelber, MD
3:00pm-3:30pm	Introduction to Global Skin Tones 3 to 6	Pamela Springer, LE
3:30pm-4:00pm	Innovations in Scar Prevention and Treatment	Jerra Banwarth, RPh

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22nd Annual World Congress on Anti-Aging Medicine

Saturday, December 13, 2014 Conference

• Morning Session •		Presented By:
7:00am-7:35am	Metabolic Control and the Retina: It's More than What Meets the Eye	Shalesh Kaushal, MD, PhD
7:35am-8:10am	Weight Loss in Patients with Diabetes: Keys to Success	Osama Hamdy, MD, PhD
8:10am-8:55am	Methylation's Role in Neurological Health, Aging & Recovery: Beyond MTHFR	Kendal Stewart, MD
8:55am-9:20am	How to Maintain Memory at Any Age	Pam Smith, MD, MPH, MS
9:20am-10:00am	From Wellness and Prevention to Testosterone Replacement Therapy	Joel Heidelbaugh, MD
10:05am-11:00am	Solving World's Problems through Regenerative Medicine: Accelerate or Perish	Alexander Zhavoronkov, PhD
LUNCH (On your own) • 11:00am-1:00pm		
Exhibit Hall Hours • 10:00am-2:00pm		
• Afternoon Session 1 • Early Detection/Prevention of Aging Disorders •		Presented By:
1:00pm-1:30pm	Protecting the Aging Brain: Functional Neurology for Better Balance, Memory and Cognition	Ellie Campbell, DO, MS
1:30pm-2:00pm	Unlocking the Secrets of the Telomere: The First Step to Reversing Aging	Al Sears, MD
2:00pm-2:30pm	Death by Calcium: A common Denominator to Premature Aging & Chronic Degenerative Disease	Thomas Levy, MD, JD
2:30pm-3:00pm	Alternative Approaches to Evaluate Insomnia	Bradley Bush, ND
3:00pm-3:30pm	Beyond Lipids: Advanced Inflammatory & Cardiometabolic Biomarkers in Clinical Practice	Robert Megna, DO, ABAARM, FAARFM
3:30pm-4:00pm	Understanding the Link Between Complex Chronic Disease, Dyslipidemia, Inflammation & Food	Jamie Wright, DO
• Afternoon Session 2 • 2014 Integrative Medicine Hot Topics •		Presented By:
1:00pm-1:40pm	Healing is Voltage	Jerry Tennant, MD
1:40pm-2:10pm	Effects of Pulsed Electromagnetic Frequency with Mark II Coil on Diabetes Neuropathy	Norman Shealy, MD, PhD
2:10pm-2:50pm	New Breakthroughs in Anti-Inflammatory Protocols	Martin Gallagher, MD, DC
2:50pm-3:20pm	Mechanism of Food Immune Reactivity & Autoimmunity	Aristo Vojdani, PhD, MSc, CLS
3:20pm-4:00pm	Nutrigenomics	George Rozakis, MD
• Afternoon Session 3 • Hormones and the Anti-Aging Equation •		Presented By:
1:00pm-1:40pm	Tame the Flame of PCOS	Felice Gersh, MD
1:40pm-2:15pm	Functional Adrenal Insufficiency	Deborah Matthew, MD, FAARMFM
2:15pm-2:50pm	Topical Hormone Case Studies	Jim Paoletti, Consulting Pharmacist, FAARMFM
2:50pm-3:20pm	Low Libido: Causes, Implications and Treatment Modalities	Shelena Lalji, MD, FACOG
3:20pm-4:00pm	The Hormone Secret: Discover the Missing Link to a Better Body, Brain and Life	Tami Meraglia, MD
• Afternoon Session 4 • Med Spa and Aesthetic Medicine •		Presented By:
1:00pm-1:40pm	Help Your Patients Look Years Younger Without Surgery	Tess Mauricio, MD
1:40pm-2:15pm	Top 20 Marketing Strategies that Will Boost Your Bottom Line	Manon Pilon
2:15pm-2:50pm	The Independent Science Behind Anti-Aging Vitamin & Nutrient Cosemeceuticals and Nutracosmetics for Youthful Skin	Jeanette Jacknin, MD
2:50pm-3:25pm	The Fusion of Wellness in the Medical Spa	Patti Biro, MS, Med.
3:25pm-4:00pm	Tapping into New Revenue Streams	Dori Soukup

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Hyaluronic Acid (HA)

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vitreous humor (hyaluronic acid)

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connective tissue (hyaluronic acid and collagen)

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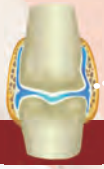

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- **Sponsored Evening Workshops** (NON-CME)
  Wednesday, December 10th | Thursday, December 11th

- **Exhibit hall**

Thursday, December 11th	10:00am – 6:00pm
Friday, December 12th	10:00am – 6:30pm
Saturday, December 13th	10:00am – 2:00pm

- **Product Showcase** (NON-CME)

Thursday, December 11th	12:00pm -1:00pm; 4:30pm-6:00pm
Friday, December 12th	12:00pm – 1:00pm; 4:00pm – 6:00pm

  Visit the product showcase inside the exhibit hall where participating exhibiting companies will present their products & services. (NON-CME)

- **Networking Reception**
  Friday, December 12th 4:00pm-6:30pm
  Mix & Mingle with other distinguished medical professionals & exhibitors while enjoying refreshments in the exhibit hall.

- **Car Giveaway**
  Play the exhibit hall game & enter to win a FREE car! Drawing will take place Saturday, December 13th at 12:00pm in the exhibit hall.

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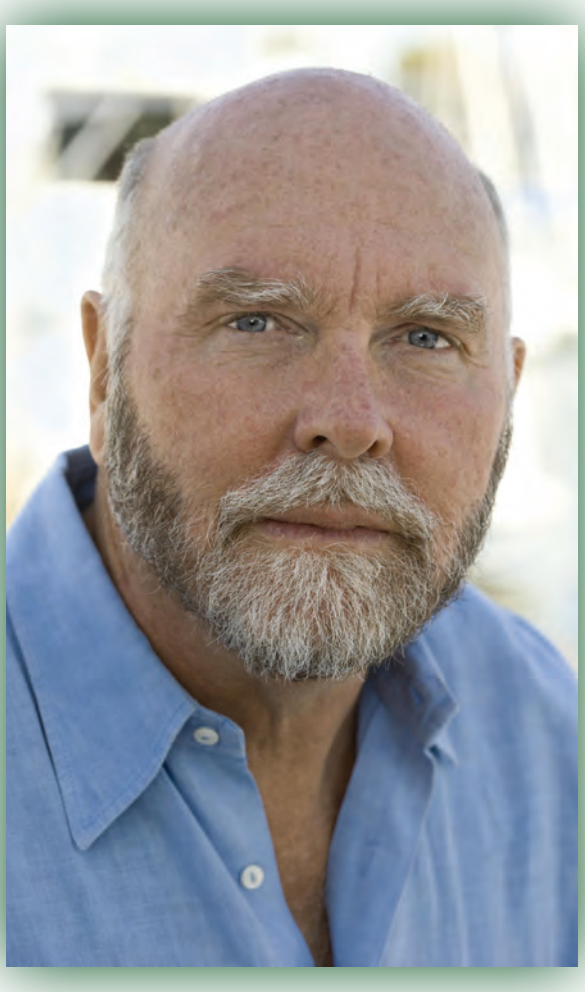

J. Craig Venter, PhD

J. Craig Venter, Ph.D., is regarded as one of the leading scientists of the 21st century for his numerous invaluable contributions to genomic research. He is Founder, Chairman, and CEO of the J. Craig Venter Institute (JCVI), a not-for-profit, research organization with approximately 300 scientists and staff dedicated to human, microbial, plant, synthetic and environmental genomic research, and the exploration of social and ethical issues in genomics.

Dr. Venter is Founder and CEO of Synthetic Genomics Inc (SGI), a privately held company dedicated to commercializing genomic-driven solutions to address global needs such as new sources of energy, new food and nutritional products, and next generation vaccines. Dr. Venter is also a co-founder and CEO of Human Longevity Inc (HLI), a San Diego-based genomics and cell therapy-based diagnostic and therapeutic company focused on extending the healthy, high performance human life span.

Dr. Venter began his formal education after a tour of duty as a Navy Corpsman in Vietnam from 1967 to 1968. After earning both a Bachelor's degree in Biochemistry and a Ph.D. in Physiology and Pharmacology from the University of California at San Diego, he was appointed professor at the State University of New York at Buffalo and the Roswell Park Cancer Institute. In 1984, he moved to the National Institutes of Health campus where he developed Expressed Sequence Tags or ESTs, a revolutionary new strategy for rapid gene discovery. In 1992, Dr. Venter founded The Institute for Genomic Research (TIGR, now part of JCVI), a not-for-profit research institute, where in 1995 he and his team decoded the genome of the first free-living organism, the bacterium Haemophilus influenza, using his new whole genome shotgun technique.

In 1998, Dr. Venter founded Celera Genomics to sequence the human genome using new tools and techniques he and his team developed. This research culminated with the February 2001 publication of the human genome in the journal, Science. He and his team at Celera also sequenced the fruit fly, mouse and rat genomes.

Dr. Venter and his team at JCVI continue to blaze new trails in genomics. They have sequenced and analyzed hundreds of genomes, and have published numerous important papers covering such areas as environmental genomics, the first complete diploid human genome, and the groundbreaking advance in creating the first self- replicating bacterial cell constructed entirely with synthetic DNA.

Dr. Venter is one of the most frequently cited scientists, and the author of more than 280 research articles. He is also the recipient of numerous honorary degrees, public honors, and scientific awards, including the 2008 United States National Medal of Science, the 2002 Gairdner Foundation International Award, the 2001 Paul Ehrlich and Ludwig Darmstaedter Prize and the King Faisal International Award for Science. Dr. Venter is a member of numerous prestigious scientific organizations including the National Academy of Sciences, the American Academy of Arts and Sciences, and the American Society for Microbiology.

David Katz, MD, MPH, FACM, FACP

Dr. Katz is the founding (1998) director of Yale University's Prevention Research Center. He received his BA from Dartmouth College in three years (1984; Magna Cum Laude); his MD from the Albert Einstein College of Medicine (1988); and his MPH from the Yale University School of Public Health (1993). He is a two-time diplomate of the American Board of Internal Medicine, a board-certified specialist in Preventive Medicine/Public Health, and a clinical instructor in medicine at the Yale School of Medicine.

Dr. Katz is the Editor-in-Chief of the journal Childhood Obesity, President-Elect of the American College of Lifestyle Medicine, founder and President of the non-profit Turn the Tide Foundation, and medical director for the Integrative Medicine Center at Griffin Hospital in Derby, CT. He is the principal inventor of the NuVal nutritional guidance system, currently in roughly 1700 US supermarkets in more than 30 states, coast to coast. He holds 5 U.S. patents on other inventions, with several patents currently pending.

Dr. Katz has published nearly 200 scientific articles and textbook chapters; innumerable blogs and columns; nearly 1,000 newspaper articles; and authored or co-authored 15 books to date, including multiple editions of textbooks in both Nutrition and Preventive Medicine.

Dr. Katz has been extensively involved in medical education. He was a founding director of one of the nation's first combined residency training programs in Internal Medicine and Preventive Medicine (Griffin Hospital, Derby, CT); and served as Director of Medical Studies in Public Health at the Yale University School of Medicine for a span of 8 years. He has led classes and given lectures for Yale students in medicine, public health, nursing, the physician assistants program, and undergraduates, as well as medical residents and faculty.

He is the recipient of many awards for his contributions to public health and medical education. He has been named one of America's Top Physicians in Preventive Medicine three times by the Consumer's Research Council of America, and serves as a judge of best diets for the annual ranking published by US News & World Report. In 2009, he was a widely supported nominee for the position U.S. Surgeon General. He was named one of the 25 most influential people in the lives of children by Children's Health Magazine. In 2012 he was cited by Greatist.com as one of the 100 most influential people in Health and Fitness (#19: http://greatist.com/health/most-influential-health-fitness-people/). He has been honored for career accomplishments by numerous organizations including the Association of Yale Alumni in Public Health; the Academy of Nutrition and Dietetics; and the American College of Preventive Medicine.

Dr. Katz has an extensive media portfolio, having worked for ABC News/Good Morning America as an on-air contributor, a writer for the New York Times syndicate, and a columnist to O, the Oprah Magazine. Currently, he is a blogger/medical review board member for the Huffington Post, a health contributor to US News & World Report, one of the original 150 'thought leader' Influencer bloggers for LinkedIn; and a health writer for Everyday Health.

Dr. Katz speaks routinely at conferences and meetings throughout the United States, and around the world, and has delivered addresses in at least 7 countries. He is a recognized thought leader in nutrition, chronic disease prevention/health promotion, weight management, and integrative medicine. In 2013, he received an honorary Doctoral Degree from the University of Bridgeport, division of Health Sciences, for his contributions to the fields of health and medicine. Widely recognized as a gifted public speaker, Katz has been acclaimed by peers as the "poet laureate of health promotion." Dr. Katz and his wife Catherine live in CT; they have 5 children.

Alex Zhavoronkov, PhD

Alex Zhavoronkov, PhD, is the director and a trustee of the Biogerontology Research Foundation, a UK-based think tank supporting aging research worldwide and is the founder of the International Aging Research Portfolio, a curated knowledge management system for aging research. He heads the laboratory of regenerative medicine at the Clinical Research Center for Pediatric Hematology, Oncology and Immunology where his research interests include Hutchinson-Gilford Syndrome, new methods of cellular reprogramming, molecular mechanisms of skin and cartilage regeneration and personalized medicine in oncology. He is also the international adjunct professor at the Moscow Institute of Physics and Technology.

He is the CEO and co-founder of InSilico Medicine, Inc a Baltimore-based company utilizing Big Data analysis for aging research and drug discovery. Prior to InSilico Medicine, he co-founded the First Oncology Research and Advisory Center, a personalized medicine organization providing contract research services to oncologists interested in gene expression and activated signaling pathway analysis and predicted effectiveness of targeted drugs to improve clinical decision making. He is also the head of research at NeuroG Neuroinformatics, a neuroinformatics company developing algorithms for cost-effective EEG devices to recognize imagined visual images and delay the onset of age-related neurodegenerative diseases.

Dr. Zhavoronkov is the author of multiple peer-reviewed scientific as well as popular papers and books on the many aspects of biogerontology, aging research and aging economics including "The Ageless Generation: how biomedical advances will transform the global economy" published by Palgrave Macmillan.

He holds two bachelor degrees from Queen's University, a masters in biotechnology from Johns Hopkins University and a PhD in physics and mathematics from the Moscow State University.

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Booth #6001 | Booth #5078 | Booth #6032 | Booth #6033 | Booth #6034 | Booth #6038

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Product Showcase
(NON-CME)

Thursday, December 11th

12:30 pm
Topic: Understanding Advanced CardioMetabolic Risk and Nutritional Correlation
Presented by: Kirstin Keilty, MD, CNS
Sponsored by: SpectraCell Labs

4:30 pm
Topic: How I Stayed Alive: The 10 Commandments
Presented by: Major General Bernard "Burn" Loeffke, PhD (USA, Ret.)

5:00 pm
Topic: GI Effects® Comprehensive Stool Profile
Presented by: Todd LePine, MD
Sponsored by: Genova Diagnostics

5:30 pm
Topic: Emerging Aesthetic Trends for 2015
Presented by: Anil Rajani, MD
Sponsored by: Viora

Friday, December 12th

12:00 pm
Topic: Leaky Gut Syndrome: The Toxic Epidemic in Aging
Presented by: Douglas A. Wyatt
Sponsored by: Sovereign Labs

12:30 pm
Topic: Obesity Epidemic, Hormonal Imbalance and Restoration
Presented by: Kimball Lundahl, DC, ND
Sponsored by: Healthy Habits Medical Business Consultants

4:00 pm
Topic: Martial Arts Longevity Secrets: Learn from the Grand Masters of Martial Arts movie star the secrets of amazing life peak performance levels and longevity.
Presented by: Alan Goldberg , Ron Van Clief, Cynthia Rothrock , Silvio Simac, Carey Tagawa, Michael Jai White, Don Wilson

5:00 pm
Topic: Protandim and Nrf2 Triggering - a Fundamentally Different Approach to Cellular Protection
Presented by: Shawn Talbott, PhD
Sponsored by: LifeVantage

5:30 pm
Topic: Clinical Applications of Stem Cells
Presented by: Clemente Humberto Zúñiga Gil
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Sponsored Workshops
(NON-CME)

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Wednesday, December 10th

6:15PM **Topic:** Leading the Evolution in Gut Health Assessment: Advances in Stool Diagnostics
Presented by: Kathy O'Neil-Smith, MD
Location: Veronese Ballroom
Sponsored by: Genova Diagnostics

6:15PM **Topic:** Using Biomarkers to Age Well
Presented by: Tara Dall, MD
Location: Titian Ballroom
Sponsored by: Health Diagnostic Laboratory, Inc

6:15PM **Topic:** Hormone Dosing with Dr. Pamela Smith and PCCA
Presented by: Pamela Smith, MD, MPH, MS; Jerra Banwarth, RP
Location: Titian Ballroom
Sponsored by: PCCA

6:15PM **Topic:** The Cannabis Revolution
Presented by: Stephen Holt, MD, DSc, PhD, LLD, ND
Location: Titian Ballroom
Sponsored by: Vitacan

Thursday, December 11th

6:15PM **Topic:** Validated In-Office Stem Cell Therapy: The Next Step in Anti-Aging
Presented by: Sharon McQuillan, MD
Location: Titian Ballroom
Sponsored by: Ageless Regenerative Institute

6:15PM **Topic:** Cannabis, Cannainoids, and CBD
Presented by: Rob Streisfeld, NMD
Location: Veronese Ballroom
Sponsored by: HempMeds

6:15PM **Topic:** Cellular Detoxification with ONDAMED
Presented by: Silvia Binder, ND, PhD; Dr. John Cline, MD, BSc
Location: Titian Ballroom
Sponsored by: ONDAMED

6:15PM **Topic:** 3T MRI & Imaging
Presented by: Eric Braverman, MD
Location: Titian Ballroom
Sponsored by: Path Medical

6:15PM **Topic:** A Personalized Approach to Patient Care
Presented by: Pamela Smith, MD, MPH, MS
Location: Bassano Ballroom
Sponsored by: SpectraCell Laboratories

6:15PM **Topic:** Telomere Modulation with TA-65: Human Case Studies
Presented by: Harvey S. Bartnof, MD
Location: Veronese Ballroom
Sponsored by: T.A. Sciences

Friday, December 12th

7:00PM **Reception by invitation only**
Location: Veronese Ballroom
Hosted by: Forever Health & BioTe

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Beyond Personalized Medicine: Personalized Lifestyle

By Maciek Sasinowski, MD, PhD

Introduction – Live Long, Live Well?

Over the last two centuries, life expectancy in developed countries has been increasing at a rate of approximately 3 months per year, with no plateau in sight. By 2050, the average life expectancy is forecast to be 96.4 years.[1,2] However, this increase in longevity or "lifespan"—which is generally credited to advances in hygiene and sanitation, infection control, improved medications, nutrition, and education—does not necessarily correlate with a person's ability to live a healthier life, or "healthspan." For example, between 2000 and 2012, global life expectancy increased by 4.1 years, but the corresponding increase in healthy years was only 2.7.[3]

Many theories about aging have emerged and faded, but—despite tremendous advances in technology and our understanding of physiology, genetics, and biochemistry—the true nature of the aging process remains uncertain.[4] Aging is inevitable, part of everyone's life. However, while most agree that getting older does beat the proverbial alternative, it is now understood that although some aging processes are natural, many phenomena previously thought to be part of natural aging actually represent signs of pathology. Two major conclusions drawn from the National Institute of Aging's landmark effort, the Baltimore Longitudinal Study of Aging (BLSA), were that 1) "normal" aging can be distinguished from disease, and that 2) no single, chronological timetable of human aging exists—we all age differently according to our genetics, lifestyle, and individual disease processes.[5]

The first conclusion from the BLSA study indicates that aging involves processes that are completely natural. Some include harmless cosmetic changes, but others may comprise more daunting transformations, such as decreased brain volume and lung capacity, immunosenescence, atherosclerosis, and a reduced ability to maintain nutritional balance. This may seem discouraging, but does it necessarily follow that we should simply resign to die at 75, as suggested by Ezekiel Emanuel, one of the architects of Obamacare?[6] Perhaps not. We now understand that many of the changes commonly attributed to aging, such as altered personality or increased risk for cardiovascular or cardiometabolic disease, are signs of pathology and are, in many cases, preventable with timely identification and appropriately tailored intervention.

The first conclusion reached by the BLSA investigators was crucial to our understanding of aging and our ability to differentiate physiological aging from pathological processes. The second conclusion, however, carries a potentially even greater promise for our future pursuit of the "healthy aging phenotype."[1] It is now understood that aging is a vastly complex and multifactorial process that affects everyone differently. While an individual's genotype is thought to explain roughly 25% of the variation in life expectancy, other factors such as environment and lifestyle (e.g., pollution, stress, infections, access to health care, financial security) provide the remaining balance. Furthermore, the aging process occurs at varying rates in different tissues and appears to also include a significant stochastic element.[1,7-9] Given the complexity and diverse nature of aging, a complete understanding of this intricate network of mechanisms can only be achieved through a melding of insights provided by comprehensive and individualized exploration at the cardiovascular, metabolic, hormonal, and neuroendocrine levels.

Healthy Aging – One Size Does Not Fit All

The last two decades have seen an increased emphasis on system-level, integrated science as clinical researchers have recognized that the characterization of single genes and proteins has provided only limited insight and benefits toward early diagnoses, improved subtyping and prognoses, and treatment of diseases. This integrative approach is critical for our ability to elucidate the network of structural, regulatory, and dynamic interactions, thereby providing a comprehensive understanding of the physiology and pathophysiology that ultimately leads to effective intervention strategies. In particular, laboratory tests have been used to stratify risk and guide medical decision support for decades. In recent years, however, novel biomarkers and comprehensive biomarker panels provided by some clinical laboratories have brought these tests into the health care delivery process and are changing the face of medicine. Comprehensive testing provides insight into the individual patient's pathophysiology, allowing clinicians and other health care professionals to tailor a specific lifestyle intervention that includes nutrition, exercise, dietary supplementation, and medication, as necessary.

Consider cardiovascular (CV) and cardiometabolic (CM) diseases, for example. Both are complex and multifactorial, and represent the main age-related diseases.[10] Thus, controlling risk factors for these conditions will help to reduce their incidence, leading to a healthy lifespan. In CV disease (CVD), it is crucial to appreciate that lipids and lipoproteins represent only the tip of the iceberg underlying the disease process. Factors such as inflammation, oxidation, myocardial stress, genetics, and many others must be considered to gain a full understanding of the pathophysiology involved. In CM disease, traditional risk stratification metrics, such as glycemic control determined by blood glucose measurements, are the last to become abnormal. Advanced markers that detect insulin resistance and pancreatic beta-cell dysfunction provide much earlier warning signs of pathology, allowing clinicians to identify and engage at-risk patients at a time when intervention is most effective.[10] The following sections describe four panels of markers used in comprehensive risk assessment that provide a methodology for personalized lifestyle intervention.

Markers of Cardiovascular Health

Age is a key risk factor for coronary artery disease (CAD). Age-related changes in the CV system can lead to increased risk of CVD, such as atherosclerosis, hypertension, myocardial infarction, and stroke.[11] Aging men and women experience hormonal changes, inducing weight gain and unfavorable lipid profiles, coupled with increasing risk for CVD.[12,13] Conversely, favorable lipid metabolism and lower CVD prevalence are associated with longevity and healthy aging. Although traditional lipid concentrations, i.e., total cholesterol, and low- and high-density lipoprotein cholesterol (LDL-C and HDL-C) are often used as surrogates for lipoprotein particle number, comprehensive testing of lipids and lipoproteins provides a more accurate assessment of CVD risk.[14,15] In addition, comprehensive biomarkers are associated with longevity and cognitive function, and some have been proposed as biomarkers for the rate of biological aging.[16-19] For example, centenarians and their offspring have significantly larger particles of LDL and HDL, which are associated with lower prevalence of hypertension, CVD, and metabolic syndrome.[16-18] In the elderly, cognitive decline is associated with lower plasma HDL and apolipoprotein A-I (apoA-I) concentrations, and increased levels of triglycerides and apolipoprotein B (apoB).[16-18] Thus, the preservation of CV health, through maintaining optimal levels of lipids and lipoproteins, is essential for augmenting both lifespan and healthspan.

Cardiometabolic Markers

Insulin resistance and diabetes are widely recognized risk factors for CVD. Adults with diabetes are 2- to 4-fold more likely to have heart disease or stroke than those without diabetes, and at least 65% of individuals with diabetes die from CVD.[20] In addition to predicting CVD and mortality, insulin resistance amplifies chronic inflammation—a major risk factor for aging—and is an important effector of morbidity during the aging process, substantially increasing the risk of cognitive impairment, neurodegenerative disease, and physical disability.[21-23] Early identification and treatment of CM disorders are thus vital to healthy aging. In addition to traditional markers such as hemoglobin A1c (HbA1c) and insulin, newer markers such as adiponectin, leptin, and alpha-hydroxybutyrate (a-HB), have been implicated as risk factors for bone loss, cognitive decline, and neurodegenerative disorders, as well as increased mortality in older individuals.[24-31]

Markers of Inflammation

Although acute inflammation provides a protective physiological response to stimuli such as traumatic injury and infection, chronic inflammation can cause substantial tissue damage and is widely accepted as a risk factor for aging. Low-grade, systemic inflammation is integrally involved in the pathogenesis of major age-related diseases such as CVD, diabetes, cancer, and Alzheimer's disease, and contributes to many conditions that reduce quality of life as we age, including sarcopenia, degenerative arthritis, osteoporosis, and frailty.[32-34] Inflammation may also induce oxidative stress, which augments tissue damage and further amplifies the inflammatory response—creating a vicious feedback loop that greatly increases the risk of poor health outcomes during the aging process.[35] Circulating markers of inflammation, such as high-sensitivity C-reactive protein (hs-CRP) and fibrinogen, are strong predictors of age-related morbidity and mortality[33,36-38]; moreover, they may help identify individuals with early-stage vascular inflammation and/or subclinical CVD, which is associated with premature aging.[39]

Hormone Markers

The hormones of the hypothalamus, pituitary gland, and gonads cooperatively regulate a range of important physiological functions, including development, reproduction, and aging. Measurement of circulating hormone levels can aid in the assessment and diagnosis of a variety of conditions, as proper hormonal balance and homeostasis is vital for overall health and wellbeing. Hormone deficiencies are also integrally related to the general health decline that often accompanies normal aging. For example, loss of testosterone and estrogen in older men and women is associated with signs and symptoms such as physical weakness, decreased muscle mass and bone mineral density, obesity, loss of libido, and depression.[40,41] Men and women with low testosterone levels are also at increased risk for CV events, CV-related mortality, and all-cause mortality.[42-45] Several reports have provided provocative evidence that decreased physical activity and increased obesity can cause declining testosterone levels in middle-aged and older men. However, it remains unclear whether healthy lifestyle behaviors and maintenance of optimal weight are sufficient for the preservation of testosterone levels during aging and improved health outcomes.[46-48] Other aging-related hormone markers include dehydroepiandrosterone sulfate (DHEA-S), the common precursor for most steroid hormones, which has been shown to have anti-inflammatory and anti-oxidative activity, and is thought to have regenerative effects. In particular, DHEA-S deficiency has been associated with prolonged psychosocial stress, providing a possible mechanistic link between chronic stress and accelerated aging.[49] In general, the identification and treatment of hormone imbalances can help maintain good health, independence, and physical and emotional wellbeing during the aging process.

The Future – Well Beyond Medicine

Despite the lack of a single mechanism that underlies healthy aging, the increase in human lifespan and, to a lesser degree, healthspan demonstrates that the process has been, and can continue to be, affected to some degree. Even though some common pathways in the aging process have been identified, pursuit of the "healthy aging phenotype" requires a multifactorial and individualized approach that takes advantage of system-level, personalized insights provided by technologies such as comprehensive biomarker testing. We have come to understand that population-based approaches to health care, resulting in guidelines and suggestions such as the Polypill (comprising a statin, three antihypertensives, an aspirin, and folic acid), aimed to reduce CVD by over 80%,[50] do not constitute long-term, strategic solutions. Rather, they represent short-term, reactive measures intended to counteract the multitude of unhealthy lifestyle choices we make that put us at risk. It is crucial to leverage tools that can identify the multifactorial nature of CVD risk factors and stratify at-risk individuals, and then to intervene appropriately with treatments tailored to each specific individual and the etiology of their particular pathology. These approaches have been shown to result not only in improved patient health, but also reduced health care expenses.[51] Therefore, an integrative, preventive, and tailored approach that combines lifestyle and appropriate pharmaceutical intervention cannot remain the exception in our health care, but rather must become a rule and integral component of clinical practice and patient care.

"The doctor of the future will give no medicine but will interest his patients in the care of the human frame, in diet and in the cause and prevention of disease."
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presenting
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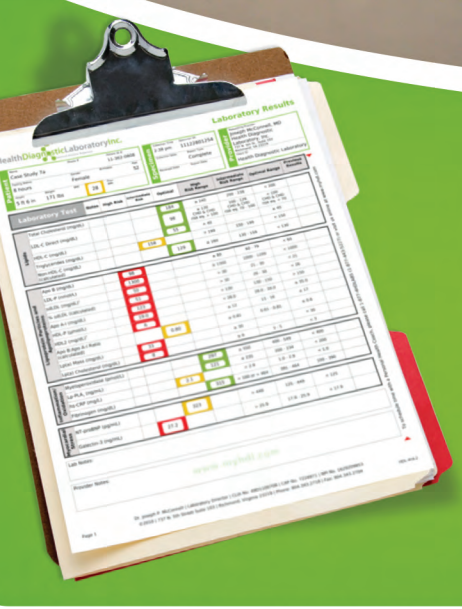

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1. Summarized from data published by ADA. Testing in Asymptomatic Patients. *Diabetes Care*. 2012;35 (Supplement 1):S14. Table 4.
2. Joslin Diabetes Center. Joslin Clinical Guidelines for Overweight and Obese Adults. 8/07/2011.
3. Hayes KC. Macronutrient ratios and insulin response for improved glucose control. Publication in process.

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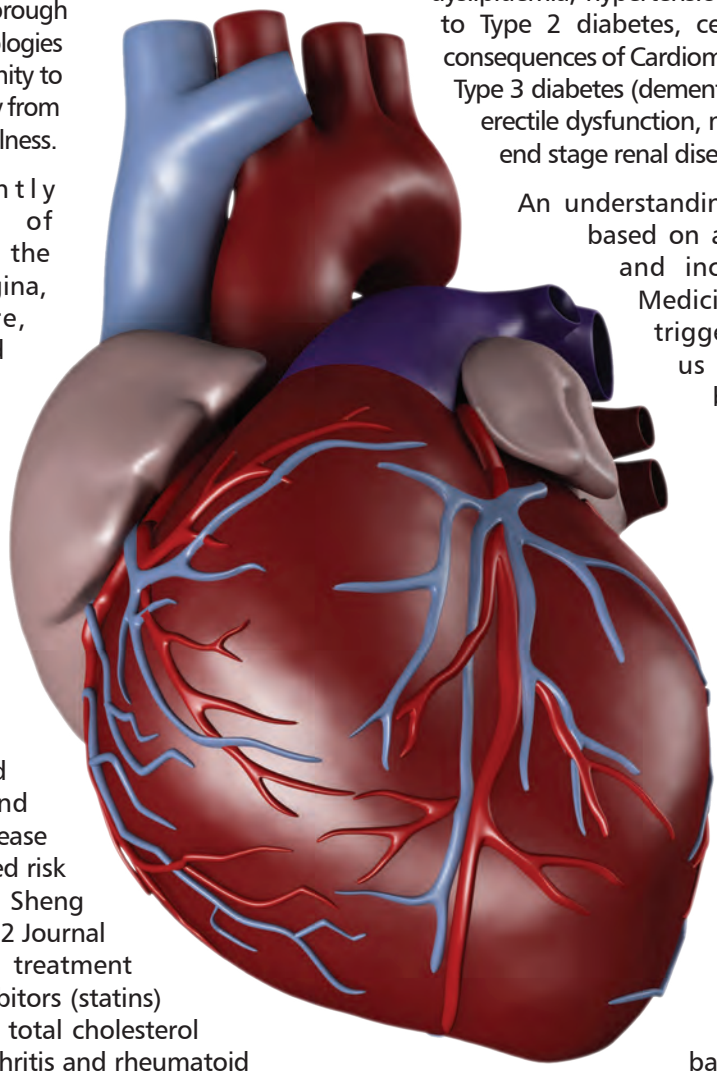
The Heart and Medicine:
Recent Advances in Fatty Acid Metabolism

ABSTRACT: Patients do not just wake up one morning with cardiac disease. Instead there is an extended pre-clinical phase during which lifestyle choices determine the outcome. Recent advances in our understanding of oxidative stress, endocrine signaling, immune/inflammatory balance and energy production illuminate opportunities for efficacious intervention. A thorough exploration of these pathophysiologies will allow physicians the opportunity to offer their patients a journey away from illness and disease to optimal wellness.

By Joseph Lamb, MD:
Director of Intramural Clinical Research, Metagenics

INTRODUCTION: Currently accepted understanding of cardiovascular disease views the signs and symptoms of angina, congestive heart failure, myocardial infarction and sudden death as acute episodes marking a disease process. Instead, systems biology approaches focus upon pathophysiological changes including oxidative stress and immune/inflammatory dysregulation that contribute to the development of disease with closely linked co-morbidities. It has been noted that rheumatoid arthritis and autoimmune inflammatory disease are associated with an increased risk for cardiovascular disease.[1] Sheng and Murphy[2] note in their 2012 Journal of Rheumatology paper that treatment with HMG CoA reductase inhibitors (statins) were associated with reduced total cholesterol levels in patients with osteoarthritis and rheumatoid arthritis. Additionally, statins were associated with reduced cardiovascular events and mortality in rheumatoid arthritis patients and with reduced all-cause mortality in osteoarthritis. Many now view cardiovascular disease not as an isolated condition but rather as the grouping of pathophysiologies that underlie Cardiometabolic Syndrome. Cardiometabolic Syndrome is defined by signs and symptoms including abdominal obesity, insulin resistance, elevated blood pressure, atherogenic dyslipidemia, a pro-inflammatory state and a pro-thrombotic state.[3] Beyond dyslipidemia, hypertension, atherosclerosis, progression to Type 2 diabetes, cerebro-vascular disease, the consequences of Cardiometabolic Syndrome also include Type 3 diabetes (dementia), sleep apnea, malignancies, erectile dysfunction, non-alcoholic fatty liver disease, end stage renal disease and osteoporosis.



An understanding of cardiovascular disease based on a systems biology approach, and incorporating the Functional Medicine model of antecedents, triggers, and mediators allows us to see the connectedness between events and the connectedness between organ systems. Recognizing this interconnectedness allows us to effectively harness the latest science in a holistic model of care so that we can offer new opportunities for intervention.

Classically, we think about many conditions as being separate, both in their signs and symptoms as well as their etiology. Yet, we now recognize the connectedness of the mechanisms of oxidative stress, endocrine signaling, immune/inflammatory balance and energy production as underlying the pathophysiology of the named disease entity. Though some of our colleagues may be puzzled by the fact that inflammation and insulin signaling seem to overlap, these signaling pathways share a common developmental history in the teleologic expansion from the fat body of the Drosophila, the common fruit fly, to the unique organs and their metabolically active adipocytes, hepatocytes and leukocytes that we see in mammals.[4] Zhang et al[5] have gone even further and suggested that though

"near instantaneous signaling of hypothalamic IKKß and NFkß might once have been critical for survival in a pathogen-filled environment by helping innate immunity, this signaling might now be very responsive [overly] and truly detrimental in today's near constant calorie-rich environment."

Atherosclerosis has been traditionally characterized as the progressive deposition of cholesterol with subsequent narrowing of lumen leading to restricted blood flow. However, Steinberg has very aptly noted that despite the key role that hypercholesterolemia plays as a major causative factor in atherogenesis, it is equally clear from the very beginning that atherogenesis has a strong inflammatory component.[6] Our current model proposes that hypertensive stimuli and oxidized lipids lead to pro-inflammatory changes in the endothelial cells that separate the lumen from the intima and media of the vessel well. The anatomical location of this single cell layer becomes crucial in our understanding of the dance that takes place between the consequences of hyperlipidemia and hyperglycemia and the consequences of inflammatory and oxidative stress secondary to toxicity.

Endothelial injury is the initiating event that leads to the migration of low density lipoprotein particles through the endothelia layer into the intima and subsequent oxidation to oxidized low density lipoprotein (OxLDL). Hypertensive stimuli such as a high salt diet and increased production of angiotensin II promote the production of reactive oxygen species and these species contribute to hypertension and to the sequelae of this disease.[7] OxLDL increases lipoprotein phospholipase activity and upregulates the generation of inflammatory mediators that promote the recruitment and migration of monocytes through the endothelial layers and their activation into macrophages. These macrophages recruit additional monocytes and scavenge the deposited cholesterol. Lipid laden macrophages transform into foam cells. These changes upregulate the secretion of inflammatory cytokines, cellular adhesion molecules, matrix myeloperoxidases and generation of reactive oxygen and nitrogen species. Ultimately, necrotic cell death of the macrophage is a final step in the formation of an inflamed cholesterol laden atherosclerotic plaque. This plaque is vulnerable to the accumulation of calcium and also vulnerable to plaque rupture with the subsequent pro-thrombotic state leading to development of flow limiting thrombi.

As we consider the biology of the endothelial layer, the role of lipids remains a significant one and indeed an evolving one. Close attention still needs to be made to the concentrations of low-density lipoprotein (LDL), high-density lipoprotein (HDL) and the very low density lipoprotein fractions. Yet, fatty acids are more than just building blocks for obstructive plaque. Fatty acids play integral roles in inflammatory processes and cell signaling. It has been demonstrated in the GISSI study that low dose fish oil significantly reduced the cumulative rate of all-cause death, nonfatal MI, and nonfatal stoke.[8] The individual components of fish oil docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) have been evaluated for their individual properties. DHA is the principal omega-3 fatty acid in fish and fish oils responsible for their blood pressure and heart rate lowering effects in humans.[9] EPA and DHA seem to be equally effective in lowering triglycerides with the caveat that while DHA leads to significant increases in HDL cholesterol, it may also lead to increases in LDL cholesterol not seen with EPA containing supplements.[10] Traditionally, supplementation of omega-3 fatty acids was conducted to restore balance between the pro-inflammatory omega-6 prostaglandins and the anti-inflammatory omega-3 prostaglandins. Recent research demonstrates additional pathways for modulation of physiology by omega-3 fatty acids as it has been shown that DHA, by modulating MAP kinases, regulates the expression of transcription factors involved in t-cell differentiation in disease and health.[11] Beyond this regulatory kinase role, omega-3 fatty acids bind to the G-coupled protein receptors (GPCR) 120 and 140. When upregulated, these receptors increase insulin sensitivity by increasing glucose-dependent, glucose-like, peptide-1 secretion and by regulating adipocyte differentiation while down regulating inflammation.[12,13] GPR-120 functions as a receptor for unsaturated long chain fatty acids and plays a critical role in modulating adipogensis and regulation of appetite and food preferences. Ichimura et al[14] have shown that GPR-120 deficient mice fed a high fat diet developed many of the signs and symptoms we associate with cardiometabolic syndrome, including obesity, glucose intolerance and fatty liver disease. And it has been found that intravenous infusion of free fatty acids influences proliferation of the beta cell in type 2 diabetic subjects.[15]

With an expanding data base suggesting the benefits of marine oils containing omega-3 fatty acids, one would suspect that a consensus would have formed around the administration of omega-3 fatty acids in subjects with cardio metabolic syndrome and cardiovascular disease. Yet, a recent study by Kromhout et al[16] pointed out that "low dose supplementation with EPA/DHA or ALA did not significantly reduce the rates of major cardiovascular events among patients who had myocardial infarction and who were receiving state of the art antihypertensive, antithrombotic, and lipid-modifying therapy." However, there were major flaws with the design of this study and indeed the study's 2x2 factorial design was inappropriate for the evaluation of two non-independent study drugs being the fish oil and the ALA. Additionally, the study was under powered to detect differences between the four groups. One of the major physiological effects of omega-3 fatty acids supplementation is reduction in triglycerides and the relevance of the low doses administered in this study are demonstrated by no change in triglycerides in any of the treatment groups. Despite this in a post-hoc analysis, there was a significant reduction in cardiovascular risk for the diabetic subjects on fish oil in this study. An additional trial[17] and a recent meta-analysis[18] have suffered from similar methodological flaws.

Defilippis et al[19] in a conservative opinion recommend "one serving 200-400 grams of fatty fish two times per week in a diet that includes foods rich in ALA for the primary prevention of cardiovascular disease." Additionally, they recommended one serving of fatty fish or a fish oil supplement containing 900 milligrams of EPA/DHA every day as well as a diet rich

in ALA for patients with known cardiovascular disease or congestive heart failure. Two excellent reviews of omega-3 fatty acids are available.[20,21]

An increasing literature base describes an additional important fatty acid, palmitoleic acid (C16:1n7)(PA), an omega-7 fatty acid. PA is an endogenous end product of the conversion of the saturated fatty acid, palmitic acid by stearoyl-coenzyme-A desaturases (SCD). Fatty acid synthesis or de novo lipogenesis (associated with upregulated SCD1) is responsive to energy imbalance, carbohydrate intake and alcohol intake. SCD 1 is primarily found in the liver and adipose tissue. While a few preliminary studies correlated unfavorable lipid and inflammatory biomarkers to PA, the production of palmitoleic acid by the liver in response to dietary stimuli associated with poor lifestyle choices is greater than the production in adipose tissue and may confound these studies correlating serum levels of PA with biomarkers. In an elegant animal model of mice genetically knocked out for fatty acid binding protein (FABP-/-), Cao et al. demonstrated that adipose tissue uses adipose-derived PA "to communicate with distant organs and regulate system metabolic homeostasis." [22] And in further animal studies and early human work, PA has been linked to the regulation of lipid metabolism and glucose metabolism.[23,24] Mozzafarian et al. note that their "findings suggest that circulating palmitoleate may have direct regulatory benefits on some metabolic pathways, consistent with animal experiments, yet may also be a marker of other underlying lifestyle traits such as carbohydrate intake and energy imbalance (adiposity) that could confound these direct effects." [25] To address this issue of confounding, Mozzafarian et al. in a study of dietary intake of exogenous (from dairy) trans-PA, higher levels of trans-palmitoleic acid were associated with lower levels of insulin resistance, atherogenic dyslipidemia and incident diabetes.[26] In ongoing clinical work, exogenous supplementation of cis-PA in humans with dyslipidemia has been associated with favorable changes in inflammatory and lipid biomarkers. Preliminary results from a recent placebo controlled study with 60 healthy patients demonstrated improvements in blood lipids (reductions in total cholesterol [7%], triglycerides [17%], and LDL [7%] and increase in HDL [3%]) and hs-CRP (50% reduction) for the treatment group with minimal changes in the placebo group.[27]

Additionally, specifically targeting therapeutics for endothelial dysfunction allows the practitioner an opportunity to intervene early in the treatment of pre-clinical cardiovascular disease. Niacin, CoQ10, Vitamin D, and dark chocolate all have favorable impacts upon endothelial function.[28,29,30] Foods that are rich in nitrites and nitrates are beneficial for improving endothelial function. Hord notes that "approximately 80% of dietary nitrates are derived from vegetable consumption; sources of nitrites include vegetables, fruit, and processed meats. Rho-iso-alpha acids (RIAA), an extract of the hop plant, have been shown to inhibit PKCßII.[31] Given the activity of PKCßII inhibitors in restoring endothelial function, medical foods supplemented with Acacia and RIAA are excellent choices for the nutritional support of individuals with cardiometabolic syndrome.[32,33] Our preliminary work (conducted here at the Functional Medicine Research Center) with another hop extract, Tetra-hydro-alpha-acid (THIAA), and niacin has demonstrated statistically significant efficacy in improving endothelial function in humans.[34] In cell culture studies , the combination of THIAA and niacin was superior to niacin alone.

CONCLUSIONS: Exploring the interconnectedness of cardiometabolic syndrome and its associated diseases provides an opportunity to better understand the mechanisms that underlie these important conditions. Treatment directed at pathophysiological disturbances rather than diagnostic categories provide opportunities for patients to take a journey from illness to health and wellness. Our opportunity as practitioners is to help guide patients on this journey and our reward is that our patients will experience better health.

A longer version of this amended review first appeared in publication as Lamb J, Bland J. The Heart and Medicine: Exploring the Interconnectedness of Cardiometabolic-Related Concerns through a Systems Biology Approach. Global Advances in Health and Medicine Journal 2012;1(2):36-43. (David Riley editor at GAHMJ has requested this note to appear at conclusion of article in the disclosure section.)

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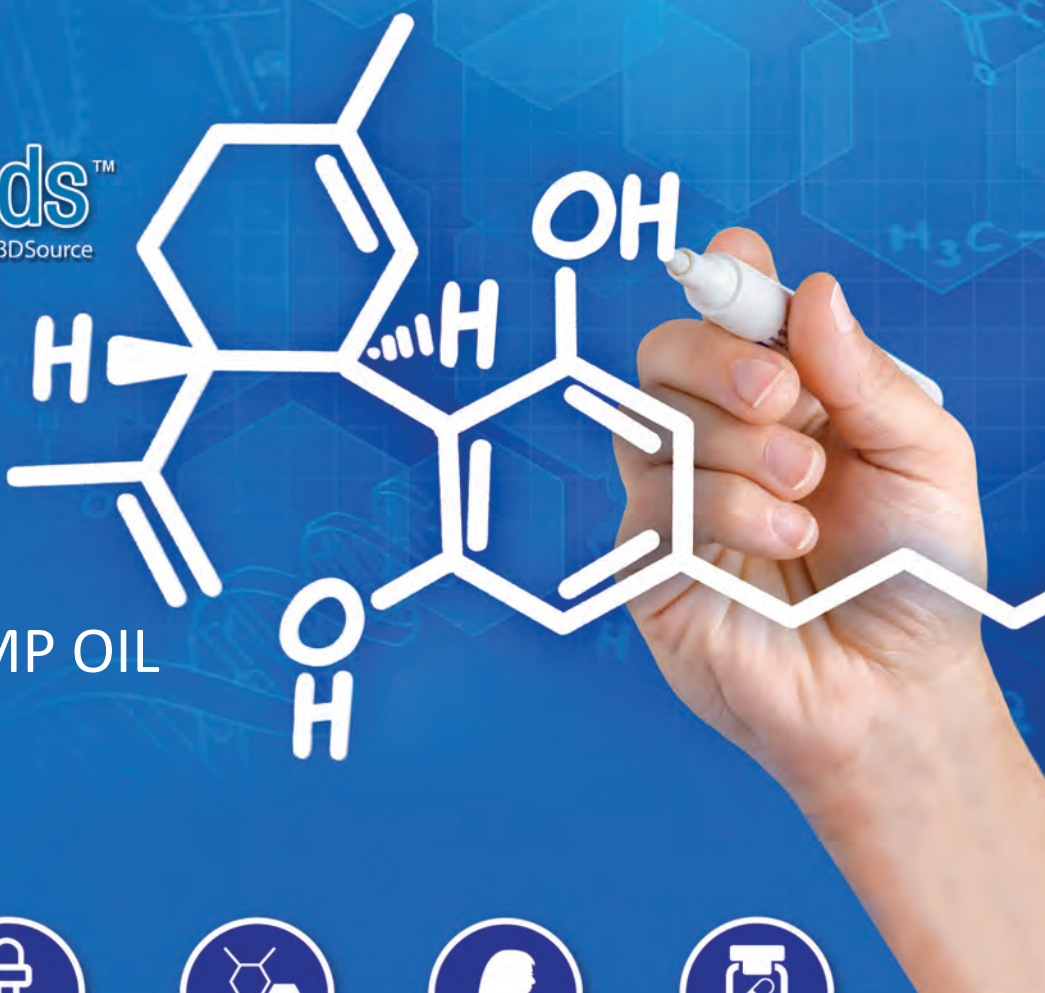

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Imbalance of the Endocannabinoid System Plays a Pivotal Role in Chronic Illnesses

By James Jeffrey Bradstreet, M.D., FAAFP
Director, Brain Treatment Center of Atlanta and Bradstreet Wellness Center, LLC
Clinical Faculty, Western University, COMP, Pamona, CA

Introduction: The endocannabinoid system (eCB), with its receptors, associated fatty acid ligands and enzymatic pathways, represents one of the most important regulatory pathways in human health and disease. Accumulating evidence connects the eCB to obesity, diabetes, heart disease, chronic pain, reproductive disorders, neurodevelopmental problems (including autism), chronic inflammatory conditions, and neurological disorders. The eCB acts as a vital surveillance system linking the diet and intestinal microbiome to adiposity, immune-regulation and neurobehavioral functions. A previously unrecognized role of vitamin D and vitamin D binding protein (VDBP) in the regulation of the eCB appears vital to brain and immune function. A variety of naturally occurring phytocannabinoids (in addition to the traditionally known cannabis-related compounds) have been identified and synthetic and natural regulators of the eCB are under investigation as pharmacological interventions. Of equal importance, secondary to a changing and more accepting regulatory and political environment, Cannabis and its oil is becoming even more widely available to both practitioners and patients. This new acceptance of the importance of the eCB has led to rapid proliferation of scientific interest and research, resulting in remarkable advances in our understanding of how the eCB regulates many vital pathways in mammalian physiology.

History of the Medical Understanding of Cannabis: Cannabis has a lengthy recorded medicinal history. While its utilitarian (fabric, paper and rope) history dating back to the earliest civilizations of China's Stone Age times[1], and it appears on some of the earliest inscriptions still in existence; ancient Chinese turtle shell texts[2]. Several references to the ancient Chinese medicinal use of Cannabis survive to this day and support its use around the time of 2500 B.C.[3]. These manuscripts reference a wide variety of pharmacological application including its use as an anesthetic for use in surgery.

Despite this lengthy history of medicinal application, the first discovery of a brain cannabinoid receptor (CB1) would have to wait until 1988[4]. Still, it's a little surprising that by 2014 we are only now beginning to appreciate the vast importance of eCB in neurological regulation. A few years later in 1993, an immunological receptor (CB2) was found to be distinct from CB1, and was observed to be expressed in splenic macrophages[5]. Early on this led to the supposition that CB1 was involved in neurological functioning and that CB2 was the immunological regulatory of the eCB. However, this view is clearly inadequate and as novel receptors beyond CB1and 2 are discovered the complexity of this system indicates a delicate and yet powerful web of interconnections and cross-talk is required for homeostasis in mammalian systems.

Interactions with Medications & Supplements: This delicate balance of homeostatic mechanism is, however, easily disrupted. Is a recent review article the authors made these conclusions:

"Evidence indicates that several classes of pharmaceuticals upregulate the eCB system, including analgesics (acetaminophen, non-steroidal anti-inflammatory drugs, opioids, glucocorticoids), antidepressants, antipsychotics, anxiolytics, and anticonvulsants. Clinical interventions characterized as ''complementary and alternative medicine'' also upregulate the eCB system: massage and manipulation, acupuncture, dietary supplements, and herbal medicines. Lifestyle modification (diet, weight control, exercise, and the use of psychoactive substances—alcohol, tobacco, coffee, cannabis) also modulate the eCB system."[6]

With this is mind it is easy to see why access to the potpourri of substances available by prescription or over-the-counter, as well as other agents easily accessible to most of us, has created physiological chaos within the eCB. This also positions eCB disruption at the center of most, if not all, chronic illnesses.

The Role of Dietary Fat: The Western – particularly the US diet – is replete with omega 6 fatty acids and in particular, arachidonic acid[7]. Arachidonic acid (AA) is the substrate from which endocannabinoid ligands are derived. These consist of the two primary endogenous ligands of the CB receptors: anandamide (N-arachidonoylethanolamide, AEA) and 2-arachidonoylglycerol (2-AG). The human body synthesizes anandamide from N-arachidonoyl phosphatidylethanolamine (NAPE), which is derived from arachidonic acid[8]. AEA appears to be an active ligand (agonist) of both CB1 and 2, whereas 2-AG is felt to act more exclusively at CB1[9]. As such, the high omega 6 diet will be associated with changes in the eCB regulation with resultant chronic pain and inflammatory conditions including: type II diabetes[10], atherosclerosis[11], obesity[12], arthritis[13] and neuro-inflammatory states including Alzheimer's dementia[14].

The Role of the Gut & the Microbiome in eCB Regulation: Perhaps the most important present day inference with normal eCB function is the widespread disruption of human microbiome by antibiotics, diet and synthetic food additives. The role of endocannabinoids in regulating leptin and obesity, perhaps the most important health issue of our time, was recognized in 2001[15]. However the linkage between the microbiome's regulation of the eCS took another decade before this critical connection was brought to light[16]. The abstract from this vital research clearly defines the importance of this issue:

"Obesity is characterised by altered gut microbiota, low-grade inflammation and increased endocannabinoid (eCB) system tone; however, a clear connection between gut

microbiota and eCB signalling has yet to be confirmed. Here, we report that gut microbiota modulate the intestinal eCB system tone, which in turn regulates gut permeability and plasma lipopolysaccharide (LPS) levels. The impact of the increased plasma LPS levels and eCB system tone found in obesity on adipose tissue metabolism (e.g. differentiation and lipogenesis) remains unknown. By interfering with the eCB system using CB(1) agonist and antagonist in lean and obese mouse models, we found that the eCB system controls gut permeability and adipogenesis. We also show that LPS acts as a master switch to control adipose tissue metabolism both in vivo and ex vivo by blocking cannabinoid-driven adipogenesis. These data indicate that gut microbiota determine adipose tissue physiology through LPS-eCB system regulatory loops and may have critical functions in adipose tissue plasticity during obesity."

Most recently in mainstream gastroenterological literature, the gut microbiotia has become an attractive therapeutic target for treating the gut-immune-brain axis in type II diabetes and obesity[17].

The Role of Vitamin D & VDBP: In 2012, researchers from the University of Pisa, Italy, observed a significantly down-regulation of circulating VDBP in multi-vessel coronary artery disease and further found an inverse relationship between VDBP serum levels and the severity of disease. Vitamin D has a well-accepted anti-inflammatory role in human physiology. Researchers in India recently observed an inverse relationship between vitamin D levels and the microvascular complications of diabetes, i.e., lower vitamin D correlated with higher levels of microvascular disease. In 2014 our team discovered the VDBP in its glycosolated form Gc-macrophage activating factor (GcMAF) regulated the transcription of eCB related receptors[18].

Summary: It is apparent the eCB plays a central regulatory role in physiological homeostasis, and no intervention for chronic illness will be successful without carefully considering the contribution of eCB tone to the individual's current health state. The microbiome-gut barrier-vascular inflammation- brain axis is a nexus for nearly all diseases of modern mankind, and it is regulated by an interaction of LPS and the eCB. By adding in the contribution of vitamin D and its associated binding proteins, a complex and interconnected system becomes apparent. These vital eCB pathways appear to be adaptive to environmental, microbiological and behavioral changes, and are regulatory in a way that attempts to assure survival and stability.

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Scientists' research was awarded the Nobel Prize in Physiology/Medicine in October 2009 for the breakthrough discovery of telomerase. Telomerase stabilizes telomere length by adding DNA repeats (nucleotides) onto the telomeric ends of the chromosomes. Shortening of these DNA sequences, known as telomeres, acts as an aging clock and explains the Hayflick Limit – the number of times cells can divide before becoming non-functioning (senescent) or dying (apoptosis).

Since telomeres act as a clock within our cells, they represent their age and how well they function. As they shorten, they signal changes in gene expression, changing the cell's phenotype to that of an older cell. Furthermore, short telomeres can lead to genetic mutations that result in serious complications associated with old age. Though good telomere health may not necessarily extend lifespan, research shows that it clearly supports a longer "healthspan" — the number of years an organism is functioning with vitality.

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  (2) human syndromes of premature aging associated with premature telomere erosion.
- Understand common human aging pathologies that are associated with telomere shortening.
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TELOMERE CONTROL IN STEM CELL THERAPIES

Pilar Najarro, PhD

Shortening telomeres are an indication of cellular aging. New technologies for fast and accurate measurement of telomeres for stem cell therapy quality control can track telomere function and dysfunction, and mark the rate of cell aging in regenerative medicine products.

BACKGROUND

Discovered in the 1930s, with their role elucidated in the 1970s, telomeres are caps of repeating sequences of nucleotides that protect the ends of chromosomes, stopping them from fusing, or from losing base pairs during replication. As part of the cell's natural aging process, every time cells replicate, the telomeres get a little shorter and once they reach a critical length, the cell stops replicating, enters senescence and eventually dies.

Abnormal shortening of the telomeres has been linked with a wide variety of age-related diseases and conditions, including cardiovascular disease, CNS disorders such as Alzheimer's disease, macular degeneration, arthritis, osteoporosis, skin damage, infertility, menopause and diabetes. In 85-90% of adult cancers the enzyme telomerase, which normally is only active in germ cells, stem cells and fetal tissues, becomes reactivated. This allows the cells to escape the aging process and become immortal.[1]

The regulation of telomere length and telomerase activity is a complex and dynamic process that is tightly linked to cell cycle regulation in human stem cells. Measuring telomere length distribution, and particularly the presence of short telomeres, informs researchers and physicians about the health of a cell, because it reflects cell viability and chromosome stability.

Telomere attrition is inherent to cell division and therefore during the ex-vivo expansion of stem cells a tight control of genome maintenance is needed. Rigorous, quantitative telomere testing can now be used as a key biomarker that correlates with the ability of human cells to proliferate and replenish tissues.

FUNCTIONAL DECLINE OF TELOMERES IN STEM CELLS

In the cells of our body, not including sperm and eggs, the activity of telomerase that elongates telomeres is usually reduced after birth so that the telomere length is gradually shortened with cell divisions. This fate does not apply strictly in the same way to stem cells.

Stem cells are found in all of us from the early stages of human development to the end of life. There are two main types of stem cells. Embryonic stem cells which can be derived form a very early stage in human development have the potential to produce all of the body´s cell types. These cells keep their telomerase activity "on" in order to reduce telomere attrition ensuring their proliferative and self-renewal capacity. Adult stem cells are found in certain tissues in fully developed humans, from babies to adult and can produce a limited type of specialized cells. The level of telomerase activity is low or absent in the majority of adult stem cells regardless of their proliferative capacity. Thus, even in stem cells, except for embryonic stem cells and cancer stem cells, telomere shortening occurs during replicative ageing (Figure 1) although possibly at a slower rate than in normal somatic cells.

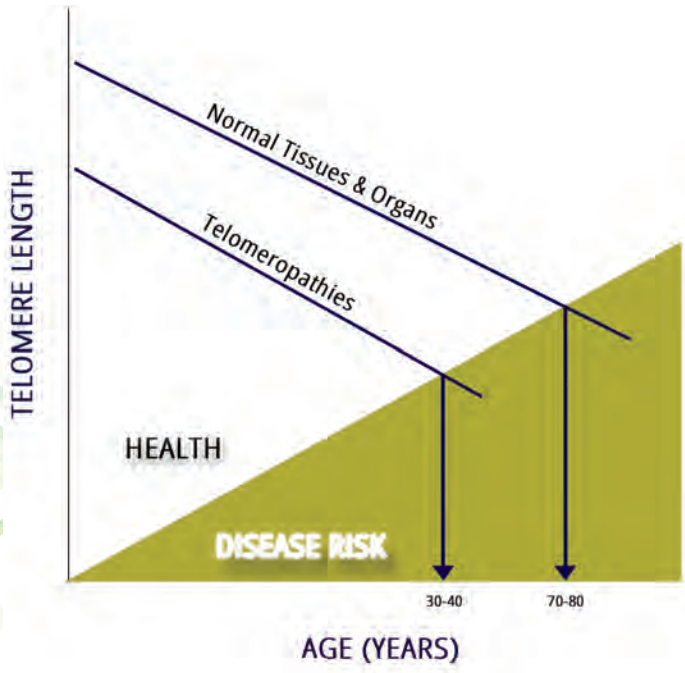


Figure 1: Telomeres shorten as we age. Telomeropathies are associated with a premature impaired capability of tissue renewal by stem cells.

Adult stem cells are hidden deep within organs, surrounded by millions of ordinary cells, and may help replenish some of the body´s cells when needed. In fact, some adult stem cells are currently being used in therapies. They have been found in several organs that need a constant supply of cells such as the blood, skin, and lining of the gut, and have also been found in surprising places like the brain, which is not known to readily replenish its cells.[2]

Recently, the importance of telomere maintenance in human stem cells has been highlighted by studies on telomere related diseases (telomeropathies) such as dyskeratosis congenita, which is a genetic disorder in the human telomerase component.[3] People with telomeropathies show decreased lifespan in addition with a premature loss of tissue renewal, suggesting that telomerase activity is rate-limiting for tissue maintenance and regulation of homeostasis. These findings are of great relevance as they indicate that telomerase activity and telomere length can directly affect the ability of stem cells to regenerate tissues.[4]

STEM CELL QUALITY CONTROL

Cell culture is a term that refers to the growth and maintenance of cells in a controlled environment outside an organism. A successful stem cell culture is one that keeps the cells healthy, dividing and unspecialized. The culturing of stem cells is the first step in establishing a stem cell line –a propagating collection of genetically identical cells. Cells lines are important because they provide a long-term supply of multiplying cells that can be used in research and cell therapy development. It is well accepted that over time all cell lines -as they are maintained in culture, change and there is no reason to expect stem cell lines to behave differently. Therefore, development and manufacturing of a therapeutic stem cell product requires extensive quality control (QC) to ensure the identity, quality, and safety of the cells.

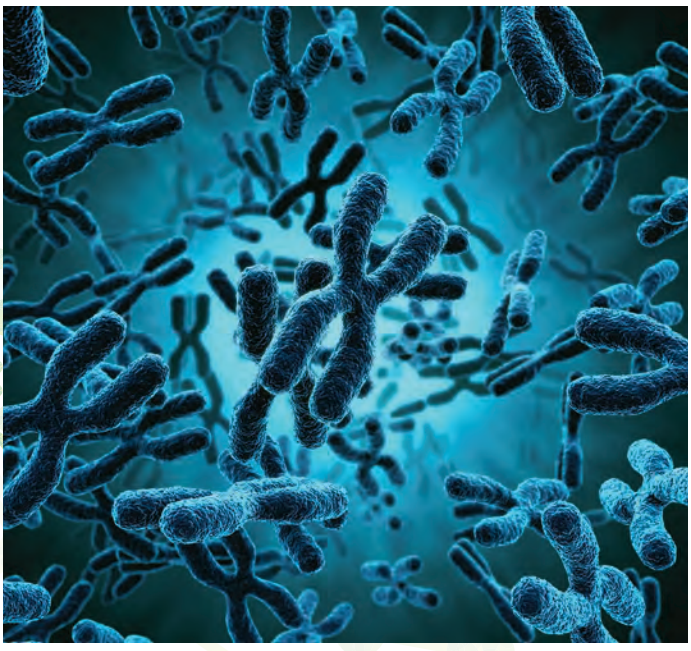
Mesenchymal stem cells (MSCs) are adult stem cells that can be isolated from most adult tissues, including bone marrow, adipose, liver, amniotic fluid, lung, skeletal muscle and kidney. The term MSC is currently being used to represent both mesenchymal stem cells and multipotent mesenchymal stromal cells. Human adult MSCs are being evaluated for the treatment of a large variety of pathologies, including traumatic lesions and cardiovascular and autoimmune diseases.[5] Although MSCs can be obtained from several tissues, they are scarce and their quantity and quality depends on a patient's clinical history, age, gender and genetic background. Most cell therapy protocols use 10–50 million MSC per treatment, requiring expansion of extracted stem cells ex vivo before implantation.

Telomere attrition is inherent to cell division and therefore during the ex-vivo expansion of stem cells changes in telomere length might arise underlying the necessity of a tight monitoring of genome maintenance. Different experimental approaches are being used to dissect the challenges faced by stem cells in their need to accurately preserve their genome integrity while maintaining their rapid proliferation and unique cell cycle characteristics.

Several companies are working in the development of culture conditions and working procedures that will reduce the prevalence of genomic aberrations without compromising cell number yields. These efforts are aimed to optimize and standardize the methodology that would result in a high quality, well characterized product safe and consistently reproducible.

Indeed, the increasing use of MSCs has led to production processes that need to be in accordance with Good Manufacturing Practice (GMP). In cellular therapy, safety remains one of the main concerns and refers to donor validation, choice of starting material, processes, and the controls used, not only at the batch release level but also during the development of processes. Implementing controls during the manufacturing of clinical-grade MSCs is essential. The controls should ensure microbiological safety but also avoid potential side effects linked to genomic instability driving transformation and senescence or decrease of cell functions (immunoregulation, differentiation potential).

Available methods for inspecting the genomic content of cells vary in their resolution, sensitivity, cost, and time.[6] Generally, they can be divided into cytogenetic methods, isolated DNA–based methods, and isolated RNA–based methods. The cytogenetic methods, such as G-band karyotyping and spectral karyotyping, are based on analyzing chromosomes at the metaphase stage of mitosis. Their resolution is relatively low but their



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sensitivity is high because the analysis is performed at the single-cell level. Also recently available is the possibility to examine telomere length distribution in MSCs samples in cells in interphase measuring telomeres individually and thus allowing to track cellular aging. In particular, the use of high-content screen such as HT Q-FISH (high throughput quantitative fluorescent in situ hybridization) allows measuring telomere length in individual cells (fig 2), in tissue and cell lines, and in blood samples, in peripheral blood mononuclear cells (PBMCs) and informs of the presence of critically short telomeres that are responsible of cellular senescence.

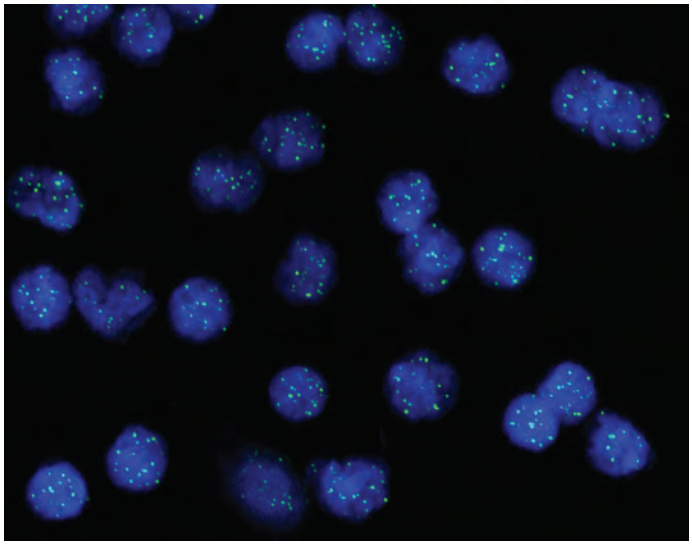

Figure 2: High content screen on human cells based on fluorescence imaging of telomeres (bright pink) from individual cells (blue nuclei). Intensity is converted to base pair length determination for each specific signal.

Growth of MSC in culture may be improved by adjusting cell culture conditions and the signals to which they are exposed by using cell culture techniques that reduce the selection for aberrant cells. Expanding MSCs in vitro is a stressful event for these cells but there are several ways to minimize genomic insults. Good cell culture practice include working with low-passage cells, applying gentle passaging techniques, and avoiding unnecessary freeze-thaw cycles that may reduce the accumulation of DNA damages. In addition providing proper environmental conditions and stimulating or inhibiting some signaling pathways can further improve the quality of stem cell during in vitro expansion. For example, low oxygen concentrations (hypoxia) has been shown to both, help to maintain the pluripotent state and to push the MSCs toward anaerobic glycolysis[7], resulting in less oxidative stress (ROS) and less DNA damage.

During cell culture optimization and on assessment of final product, master and working cell banks HT-Q-FISH technology for telomere length determination can be a companion biomarker for product development in regenerative medicine.

SUMMARY & FUTURE CHALLENGES

The clinical grade production of stem cells necessitates adhering to good manufacturing practices (GMP) to ensure the delivery of a "cell drug" that is safe, reproducible and efficient. All parts of the process must be defined: the starting material (tissue origin, separation or enrichment procedures), cell density in culture, and medium (fetal calf serum (FCS) or human serum, cytokines with serum-free medium, etc.) Analytical methods are needed to assay cells and impurities and at least a minimum, quality control of cells must consider the phenotype, functional potential, microbiological safety, and ensure the cultured cells remain untransformed.

Telomeres have been recently proposed not only as mere "replicometers" capable of counting cell division cycles but as important cellular structures that can also act as molecular switches to decide on cell cycle progression in response to a variety of stresses.[8] They are a key measuble factor on stem cell quality control and can be crucial to assess stem cell maturity and pluripotency, and to monitor proliferative capacity.

Stem cell science is extraordinarily promising. There have been great advances in treating diseases and conditions of the blood system using blood-forming stem cells, and these show us just how powerful stem cell therapies can be. Scientists all over the world are researching ways to harness stem cells and use them to learn more about, to diagnose, and to treat various diseases and conditions.

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[4]PMID: 20336134; http://www.ncbi.nlm.nih.gov/pubmed/20336134

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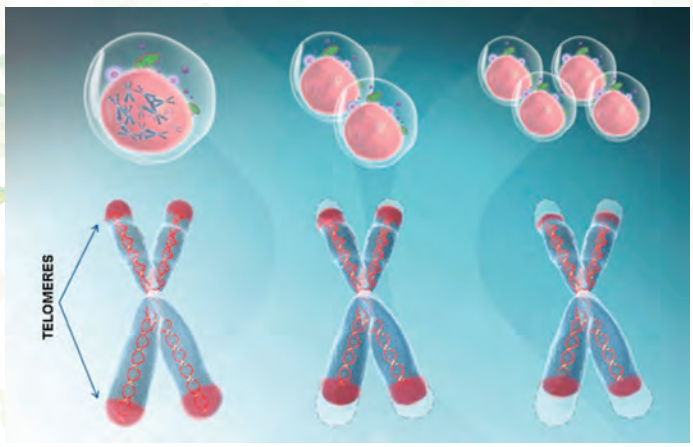
[7]PMID: 22139129; http://www.ncbi.nlm.nih.gov/pubmed/22139129

[8]PMID: 25040628; http://www.ncbi.nlm.nih.gov/pubmed/25040628



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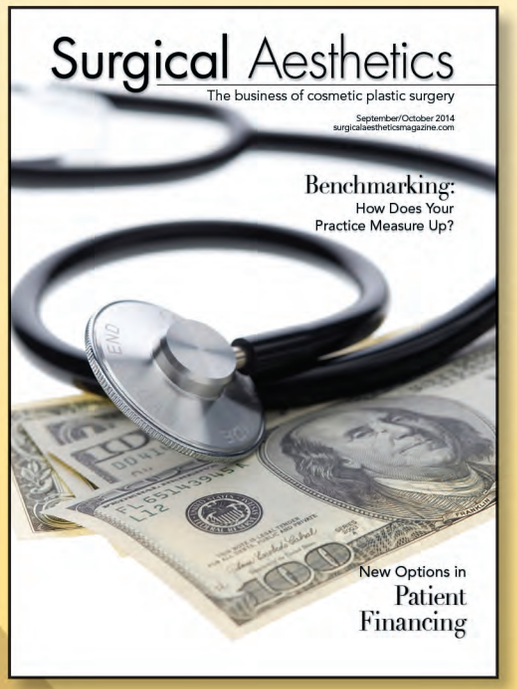

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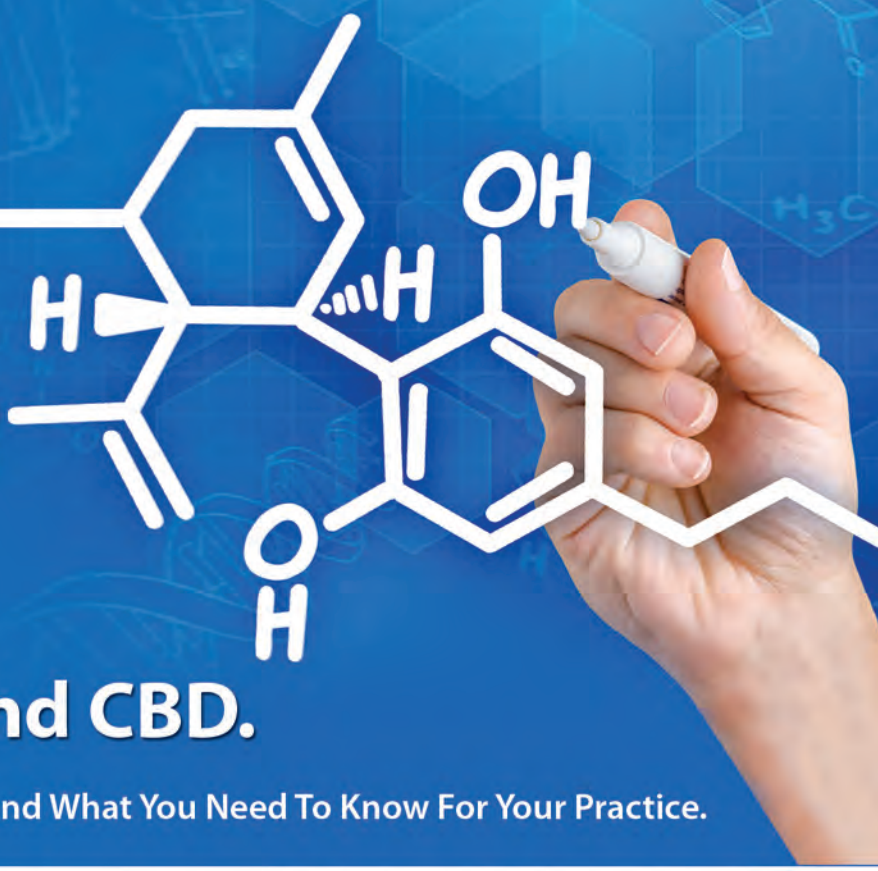

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Frequency of Abnormal Fecal Biomarkers in Irritable Bowel Syndrome

Julius Goepp, MD,• Elizabeth Fowler, PhD,• Teresa McBride, ND,• Darryl Landis, MD

Abstract: Primary Study Objective: Determine the frequency of abnormal fecal biomarker test results in patients with 13 irritable bowel syndrome (IBS)–related ICD-9 (International Statistical Classification of Diseases and Related Health Problems) codes.

Study Design: Quantitative review of de-identified records from patients in whom IBS was a possible diagnosis.

Methods: Records were selected for analysis if they included any of 13 IBS-related diagnostic codes and laboratory test results of fecal testing for all biomarkers of interest. Data collection was restricted to one 12-month period. Frequency distributions were calculated to identify rates of abnormal results for each biomarker within the total number of tests conducted in the eligible population.

Results: Two thousand, two hundred fifty-six records were included in the study, of which 1867 (82.8%) included at least one abnormal value. Quantitative stool culture for beneficial bacteria (Lactobacillus and Bifidobacterium) indicated low growth suggestive of intestinal dysbiosis in 73.1% of records, followed by abnormally elevated eosinophil protein X (suggestive of food allergy) in 14.3%, elevated calprotectin (suggestive of inflammation) in 12.1%, detection of parasites in 7.5%, and low pancreatic elastase (suggestive of exocrine pancreatic insufficiency) in 7.1%.

Conclusions: Abnormal fecal biomarkers are prevalent in patients with diagnoses suggestive of IBS. Abnormal fecal biomarker testing, if confirmed in additional independent clinical trials, could substantially reduce the economic costs associated with diagnosis and management of IBS.

Background

It is estimated that 10% to 20% of Americans in their most productive years are afflicted with irritable bowel syndrome (IBS).[1-3] IBS imposes a social burden estimated to cost approximately $20 billion a year.[4]

Despite the existence of guidelines to the contrary, many primary care physicians continue to view IBS as a "diagnosis of exclusion" and pursue costly and often invasive diagnostic studies.[5-7] The conditions to be excluded (such as inflammatory bowel disease, malignancy, and infectious colitis), while carrying potentially grave prognoses, are rarely discovered during evaluation of patients who have IBS or other functional bowel disorders.[5,8-10]

Conversely, evidence is emerging that the syndromic symptoms that define IBS according to the Rome III clinical criteria (recurrent abdominal pain or discomfort, improvement with defecation, change in frequency or in form/appearance of stool) may in fact have protean causes, often arising from one or more specific gastrointestinal (GI) conditions.[11] The advent of relatively inexpensive tests based on identification of selected fecal biomarkers now makes it possible to identify or exclude several of these underlying conditions, with the potential for a positive clinical and economic impact.[12]

GI conditions capable of producing manifestations of IBS include exocrine pancreatic insufficiency, which has an estimated prevalence of 6.1% in subjects with IBS symptomatology, and may be suggested by low levels of fecal pancreatic elastase (PE).[13] Inflammatory disorders such as inflammatory bowel disease may be discriminated from IBS with the use of the neutrophil-derived protein calprotectin in stool.[14-17] Food allergies, which have a reported prevalence rate of about 25% in IBS patients,[18] may be suggested by the presence of elevated fecal levels of eosinophil protein X, which may also be elevated in inflammatory bowel disorders and parasitic infections.[19-25] Pathogenic infections such as Clostridium difficile and

parasites such as Giardia lamblia are reported in 5.7% and 6.5%, respectively, of people with symptoms attributable to IBS[26,27] and are readily detected on fecal specimens using established techniques such as culture and light microscopy. Blastocystis hominis, the most common human intestinal parasite, was long thought to be non-pathogenic.[28,29] Some (but not all) recent studies, however, have demonstrated a significant increased prevalence of Blastocystis hominis in IBS patients compared with controls, and at least one authority has recommended treatment with metronidazole in the face of a positive identification of the organism and a symptomatic patient.[29-35]

Even in the absence of known pathogens, close study of the microbiome reveals differences in fecal bacterial populations (dysbiosis) between IBS patients and healthy controls. While a clear-cut "IBS microbiotype" has not been identified, studies have described relative increases in detrimental groups of commensal bacteria and decreases in beneficial groups, most specifically a decrease in Bifidobacteria and an increase in enterobacteriaceae.[36-43] Additionally, IBS patients are known to have a reduced diversity and stability of populations of bacterial organisms compared with controls. [36,44] The emergence of rapid means of detecting intestinal dysbiosis (eg, through 16S ribosomal DNA polymerase chain reaction [PCR] amplification) in patients suspected of having IBS adds an additional potentially powerful biomarker to the list.[36,41,44,45]

Publications to date, however, have typically focused on the identification or exclusion of one suspected condition potentially capable of producing IBSlike symptoms, such as bile acid abnormalities, exocrine pancreatic insufficiency, or inflammatory bowel diseases.[11,13-17,46-48] Identification of these individual disorders has proved useful at containing diagnostic and therapeutic costs.[12,49]

Unlike older, invasive diagnostic tests that areused in a serial fashion as each condition is excluded, fecal biomarker testing is relatively inexpensive and suited to parallel testing on a single fecal sample. If this approach is validated, it may permit clinicians and patients to arrive at a treatable diagnosis associated with the symptoms of IBS in a rapid and cost-effective manner. It may also suggest further targeted evaluations. A comprehensive study of the use of parallel testing in the context of IBS needs to be performed.

We report here a retrospective, administrative database review study of patients in whom multiple fecal biomarker testing had been performed, seeking to produce a descriptive but quantitative account of the various conditions capable of being evaluated by such testing.

METHODS

Objectives

Determine the frequency of abnormal fecal biomarker test results in patients with 13 IBS-related ICD-9 codes. The objective of this study was to identify the frequency of abnormal fecal biomarkers in patients with diagnoses consistent with IBS. The presence of abnormal fecal biomarkers may be suggestive of a potentially treatable source of GI symptomatology.

Design

We conducted a quantitative review of administrative records from patients in whom IBS was a possible diagnosis and who had undergone fecal testing for all biomarkers of interest over a 12-month period and then generated frequency distributions of abnormal test results. For this study, all data were de-identified prior to analysis, and no protected health information was recorded. It was not possible to correlate this dataset with clinical criteria such as Rome III.

Setting

We examined the computerized database of Genova Diagnostics, Inc (Asheville, NC, www.gdx.net), the Clinical Laboratory Improvement Amendments (CLIA)–certified clinical laboratory where the biomarker testing was conducted.

Table 1 Biomarkers of Interest

Biomarker	Definition of Abnormal Result	Interpretation of Abnormal Result in Context of IBS
Stool Culture, Beneficial Bacteria: (Lactobacillus, Bifidobacterium)	Growth in 1 or fewer quadrants (Lacto)/2 or fewer (Bifido)	Reduced numbers of beneficial symbionts (dysbiosis)
Eosinophil Protein X	>7 µg/g	Suggestive of food allergy or parasites (causes of eosinophilic inflammation)
Pancreatic Elastase	<200 µg/g	Suggestive of exocrine pancreatic insufficiency
Calprotectin	>50 µg/g	Suggestive of neutrophilic inflammation, eg, IBD
Occult Blood	Present	Suggestive of inflammation, malignancy, enteric infection
H pylori	Present	Suggestive of gastritis
C difficile	Positive	Suggestive of *C difficile* colitis
Parasites	Entamoeba histolytica/dispar, *Giardia lamblia*, Cryptosporidium: EIA[a] positive *Blastocystis hominis*: present on microscopic exam All other parasites: present on microscopic exam	Evidence of parasitic infection

[a] Detection by enzyme-linked immunosorbent assay (EIA).

Table 2 Diagnostic Codes Used To Define Eligible Records

ICD-9 Code	Diagnosis	Frequency (%)
789	Abdominal pain	47.61
787.91	Diarrhea	14.14
564.1	Irritable bowel syndrome	13.92
787.3	Flatulence, eructation, and gas pain	7.89
564.01	Slow-transit constipation	6.47
564	Constipation, unspecified	4.83
579.9	Unspecified intestinal malabsorption	1.99
558.9	Other noninfectious gastroenteritis and colitis	1.33
789.07	Abdominal pain, generalized	0.93
536.8	Dyspepsia and other disorders of stomach function	0.40
789.06	Abdominal pain, epigastric	0.22
536.9	Unspecified functional disorder of stomach	0.18
564.9	Functional intestinal disorder, unspecified	0.09

Patient Population

Adult records (18 years and older) were eligible for inclusion in the study if they contained results for all of the biomarkers of interest (Table 1) and if the ordering requisition listed at least one of the 13 ICD-9 codes commonly used by clinicians when evaluating patients with functional bowel disorders including IBS (Table 2).

Performance characteristics of these biomarkers for diagnoses that may present as IBS have been published elsewhere for pancreatic elastase,[50-53] calprotectin, [54-56] eosinophil protein X,[57] Clostridium difficile,[58,59] and parasitology exam,[60] with sensitivities and specificities for such diagnoses ranging from 83% to 96% and specificities in the range of 82% to 96%. The precise relationship of gut microbiota patterns to human health and disease is not yet sufficiently clear to provide specific performance characteristics.

Intervention

The intervention in this retrospective, descriptive study was the ordering of fecal biomarker tests at the discretion of the referring physician.

Main Outcome Measure

The study's main outcome measure was a frequency distribution representing the proportion of abnormal results (as defined in Figure 2) within the total number of tests conducted in the eligible population.

Results

A total of 2256 records were associated with one of the pre-selected IBS-related ICD-9 codes and had data available for all biomarkers of interest (Table 1). ICD-9 codes 789 (abdominal pain), 787.91 (diarrhea), and 564.1 (IBS) accounted for the majority (75.5%) of records; no other code represented more than 8% of records.

The gender distribution of the 2256 records was 73% female and 27% male, a ratio consistent with published data on gender distribution in IBS.[61,62] Of that group, 1867 records (82.2%) included at least one abnormal value. A frequency distribution of records with at least one abnormal test result is shown in Figure 1.

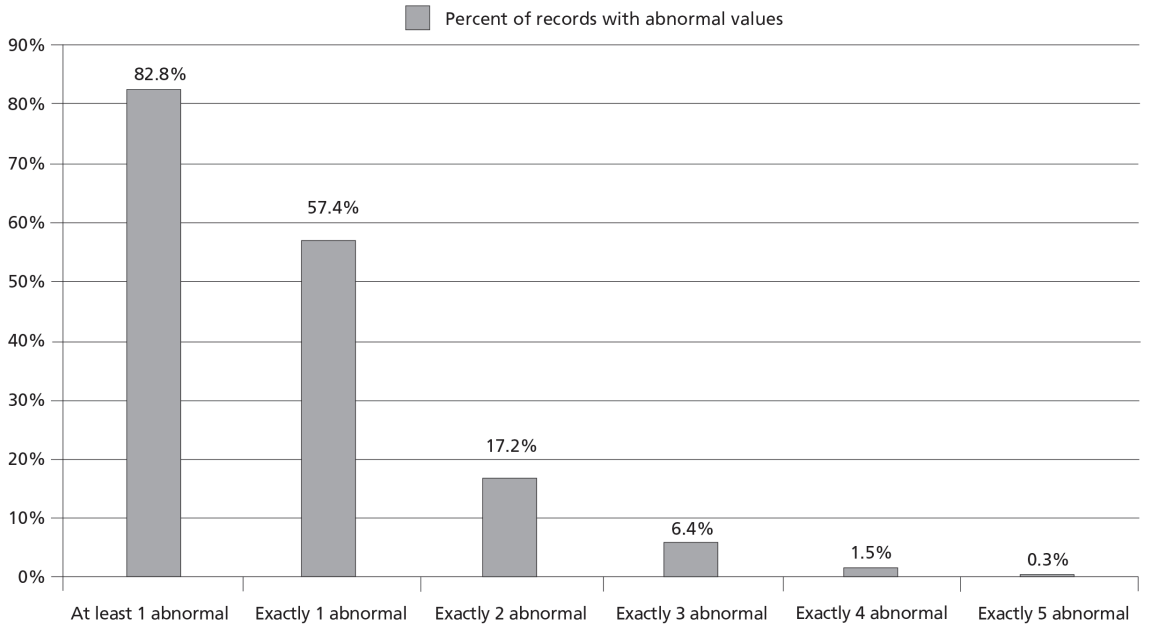

Figure 1 Distribution of records with abnormal results as a proportion of all records (N=2256).

Figure 2 shows the distribution, by fecal biomarker, of total abnormal results among the 2256 records analyzed. Several biomarkers could be divided into subcategories. The 7.5% of all abnormal labeled as "parasites" represented 73 instances (3.2%) positive for Blastocystis hominis by light microscopy, 8 each (0.4%) for Giardia lamblia and Entamoeba histolytica/dispar (by enzyme immunoassay [EIA]; similar testing for Cryptosporidium revealed no positive results), and 78 (3.5%) for all other parasites by microscopic examination.

For calprotectin, the 12.1% of results with abnormal values represents 102 specimens (4.5%) with values greater than 119 μg/g; lowering that threshold to include specimens with values in the range of 51 to 119 μg/g added an additional 171 cases (7.6%) with abnormal values.

Limitations

This study had certain limitations. This retrospective, data review study did not use clinical Rome III criteria for inclusion of records. Rather, it included patients whose ICD-9 codes suggested the presence of GI symptoms commonly manifested by patients undergoing evaluation for IBS, including abdominal discomfort associated with changes in fecal frequency or appearance. We argue, however, that these patients may in fact represent the real situation faced by practicing clinicians, namely, patients whose symptoms have no immediately obvious cause.

The population studied here is representative of primary care physicians who submitted samples for comprehensive stool profiles to one CLIA-certified clinical laboratory. These physicians and their patients may represent a unique community of providers and patients not representative

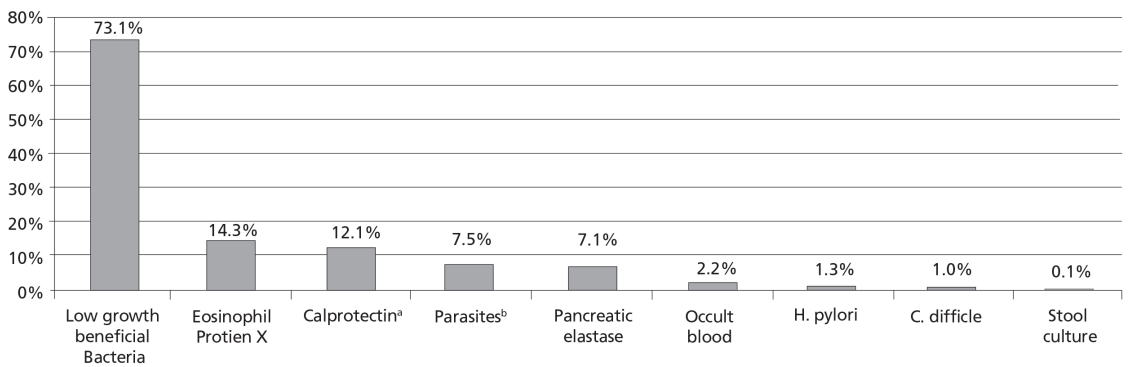

Figure 2 Frequency distribution of abnormal test results for each biomarker studied, as a proportion of all records (N = 2256). Percentages add to more than 100% because some records had more than one abnormal value.

a Calprotectin value = 4.5% greater than 119 + 7.6% in range 51-119 μg/g;
b Parasites value = Blastocystis hominis at 3.2% + entamoeba 0.4% + giardia 0.4% + other parasites 3.5%.

of the general population. Study requisition forms to capture diagnoses from ICD-9 codes are unlikely to be precise and clinicians use variable codes. These figures may represent an over-or-under-estimations of the true prevalence of these conditions in this popultation and the general population.

We believe, nonetheless, that this study provides valuable descriptive information about the potential occurrence of treatable conditions within a pool of subjects in whom IBS may have been a consideration. This study is hypothesis-generating, and additional rigorously designed studies will enhance our understanding of the role of fecal biomarker testing in evaluation of patients who have symptoms consistent with IBS.

Discussion

IBS has recently been proposed to be an "umbrella diagnosis," representing a collection of different clinical conditions capable of causing symptomatology associated with the syndrome.[11] Individual studies have been published focused on one of the many conditions that may produce IBS symptoms,[11,13,63] but no study, to our knowledge, has yet attempted to characterize the frequency with which such multiple conditions may occur within a single population. Such information would be of use in developing cost-effective screening strategies aimed at suggesting the presence or absence of treatable conditions in patients manifesting IBS symptoms.

We chose the biomarkers for this preliminary study based on their known utility in establishing or excluding the more common disease processes that can produce symptoms consistent with IBS. Several of the biomarkers, eg, calprotectin and C difficile EIA, are also FDA cleared. Newer fecal biomarkers of relevance, such as 16S ribosomal DNA PCR amplification, may emerge as practical additions to the biomarker toolkit for evaluation of patients with IBS-like symptoms.

We studied the frequency of abnormal test results on fecal biomarker testing conducted on a group of patients whose ICD-9 codes indicated the potential for IBS. We identified at least one abnormality among the biomarkers tested in more than 80% of cases. While this may appear to be a large proportion, it is consistent with previous work by Habba et al,[11] who found that 98% of patients with initial presentation of diarrhea-predominant IBS had a different diagnosis after testing and that 68% had conditions related to a treatable condition; of that group, 98% had a favorable response to therapy.

In our study, five biomarkers (beneficial bacteria, eosinophil protein X [EPX], calprotectin, parasites, and pancreatic elastase) accounted for the bulk of abnormal results. Each of these biomarker abnormalities is potentially useful as a screening test, suggesting the possible presence of a treatable condition whose eradication would reduce or eliminate symptoms compatible with IBS.

A low growth of beneficial bacteria (lactobacillus or bifidobacteria) was found in 73.1% of our samples. This is consistent with the type of beneficial bacteria insufficiency, or dysbiosis, that has been associated with IBS symptomatology.[43] Dysbiosis of this kind in IBS patients has been found to respond favorably to probiotic therapy.[64,65]

An elevation in fecal EPX was identified in approximately 14% of fecal samples. Elevated fecal EPX has been reported in patients with inflammatory bowel disorders (including Crohn's disease, ulcerative colitis, and microscopic colitis), in which concentra tions in stool are especially high, in those with parasitic infections, and also in patients with known food allergies; EPX levels fall significantly when specific treatment is provided.[19,21,22,24,25,66,67] Patients who present with IBS symptoms have been found to have EPX levels that do not differ from healthy controls when such patients do not have an associated eosinophilia-mediated condition.[67]

Fecal calprotectin is known to be present in stool in neutrophil-mediated inflammation of the intestinal mucosa.[68] Conversely, in functional disorders such as IBS, calprotectin levels are typically much lower than those found in inflammatory bowel disease (IBD) and not significantly different from those found in healthy controls.[69,70] Van Rheenen et al, in a meta-analysis of 13 studies from the primary literature, found that in adults being evaluated for IBD, screening by measuring calprotectin levels would produce a 67% reduction in the number of adults undergoing endoscopy, while only 3 of 33 adults in every 100 who do undergo endoscopy will not have IBD (but would likely have a different condition for which endoscopy is nonetheless inevitable).[14] Conversely, 6% of adults would have a delay in diagnosis of IBD because of a false negative result.

In the present study, fecal calprotectin levels were elevated in 12.1% of all specimens with use of a screening cutoff of >50 μg/g of stool. This figure represents 4.5% with values greater than 119 μg/g and an additional 7.6% of the total data set with values in the range 51 to 119 μg/g. In most clinical studies, cutoff levels of 50 or 100 μg/g are used[71,72]; however, when values from healthy controls are reported, levels of fecal calprotectin are well below 50 μg/g, typically in the range less than 10 to 20 μg/g.[73-75]

The clinical implication of using the lower cutoff level is clearly that more potential cases of inflammatory conditions will be identified, with a secondary increase in false-positive results. In a previous study aimed at

predicting relapse in IBD patients, however, the 50 μg/g cutoff produced a sensitivity and specificity of 90% and 83%, respectively, indicating an acceptable false-positive rate.[73] While the higher cutoff level of 100 μg/g has been shown to produce greater overall diagnostic accuracy,[76] in a test aimed at screening for treatable conditions in IBS, the lower cutoff (and resulting higher false-positive rate) may be preferable.

In the present study, parasites as a whole accounted for 7.5% of abnormal values. The single most commonly-identified organism was Blastocystis hominis, which until recently was regarded as a non-pathogenic organism.[28,35] Several recent studies, however, point to a moderately strong association between B hominis and symptomatic IBS, with some variation between geographic areas.[29,31,35] Certain genotype 1 of the organism shows the closest correlation with IBS.[32,77] In light of growing evidence for an etiologic role for the organism, [28] it appears reasonable to include B hominis in a screening test seeking treatable underlying conditions capable of producing IBS symptoms, particularly because treatment with metronidazole is curative.[34]

In the present study, abnormally low levels of pancreatic elastase (<200 μg/g of stool) were identified in 7.1% of all specimens; this figure represents the sum of the 2.2% of records with a value <100 μg/g and the 4.9% in the range 100 to 199 μg/g. Low pancreatic elastase is a reliable indicator of exocrine pancreatic insufficiency, comparing favorably with the secretin-cerulein test (the "gold standard"),[78] as well as several other commonly used tests for detecting pancreatic exocrine impairment. Various estimates of sensitivity, specificity, and negative and positive predictive values of fecal pancreatic elastase have been published, depending on the test used and the specific pancreatic pathology detected. A cutoff of 200 μg/g is generally accepted as the lower limit of normal[79-82]; using this value, Loser et al[78] found fecal PE1 to correlate well with the secretincerulein test, to outperform fecal chymotrypsin, and to have an overall sensitivity and specificity of 93% for diagnosing exocrine pancreatic insufficiency. When patients in that study were classified by disease severity, the sensitivity was 100% in moderate-to-severe cases but only 63% in mild cases. The lower cutoff value (<100 μg/g) may assist in identifying patients with more severe pancreatic exocrine insufficiency.

In conclusion, in this retrospective database review study of subjects with common GI disorders compatible with manifestations of IBS, a large proportion (more than 80%) were found to have evidence of a potentially treatable condition capable of producing IBS-like symptoms. Abnormal values suggesting intestinal dysbiosis, food allergy, parasite infection, exocrine pancreatic insufficiency, or inflammatory processes in the gastrointestinal tract were the most common findings. In clinical practice, these patients might then have undergone further, focused evaluation in order to arrive at a firm organic diagnosis and an effective treatment regimen; in short, these individuals might prove to have diagnoses other than "IBS." A structured, parallel fecal biomarker testing panel may represent a relatively inexpensive screening method for underlying, treatable causes of IBS symptoms. Future prospective studies focusing on patients meeting current clinical criteria such as the Rome III should be conducted, including a rigorous follow-up of all abnormal findings to evaluate the utility of a structured fecal biomarker testing panel in patients with IBS symptomatology.

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Dr Landis owns stock in Genova Diagnostics.
Dr Goepp received consultant's fees from Genova Diagnostics, Inc.

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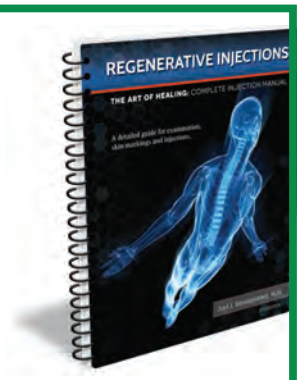

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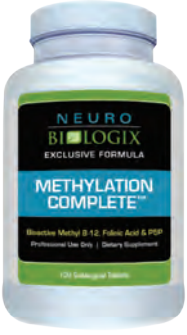

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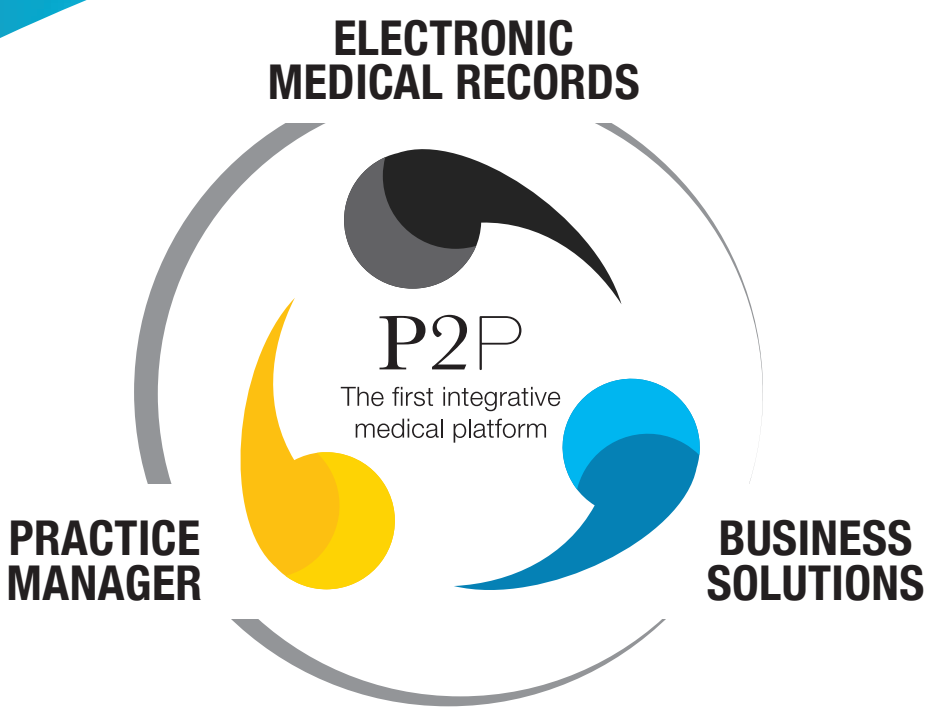

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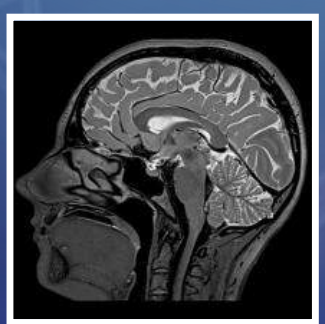
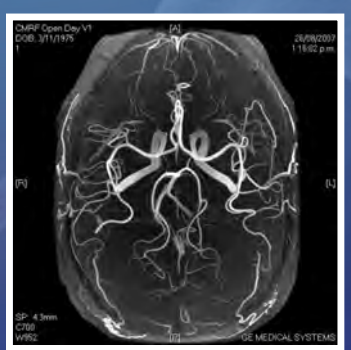
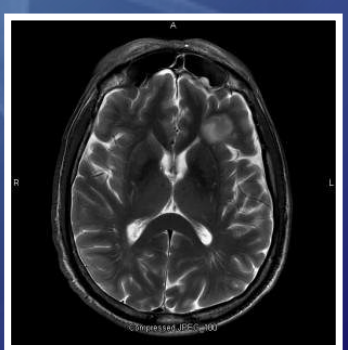






PATH TO LIFE EXTENSION

Lifestyle Medicine: More Life, Less Medicine

By Thomas Guilliams, PhD

The human body has an amazing ability to maintain its own health when provided with the right amounts and types of healthy signals. When harmful signals are removed and appropriate signals are enhanced, cells and organ systems are capable of creating a healthy outcome. Lifestyle-based interventions (i.e., Lifestyle Medicine) provide the tools and incentives to help individuals assess and prioritize the signals in their own lives that require changing, offering a bridge of support through the journey to a healthier outcome.[1] Sounds simple, right?

For the clinician, implementing lifestyle medicine requires understanding chronic disease prevention and intervention from a different perspective, one that fully integrates the relationship between lifestyle inputs ("signals") and the patient's ability to convert those signals into healthy outcomes. As Hippocrates understood it nearly 2,400 years ago, "Everyone has a physician inside him or her, we just have to help it in its work. The natural healing force within each one of us is the greatest force in getting well." Voltaire put this more bluntly in the 18th century when he said, "The art of medicine consists of amusing the patient while nature cures the disease."

Lifestyle-based therapy mandates a patient-centered philosophy; it requires assessing all the signals that influence the health of each individual and recognizes that each individual has a different capacity to translate those signals and change behaviors. Clinical success is dependent, in this model, on an ongoing relationship between an engaged clinician and an empowered patient. It is also dependent on new paradigms with new (or revised) nomenclature to describe those paradigms. Here we describe a few of those newer terms that help a clinician transition into the new paradigm of "Life as Medicine."

Physiological Resilience & Metabolic Reserve

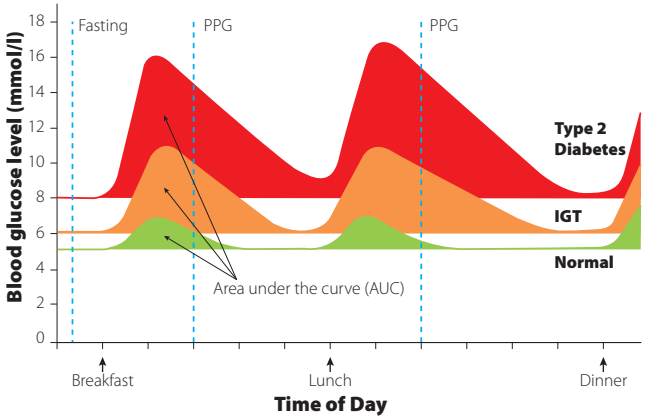
While the name we give to chronic diseases might depend on the organ system or tissues involved, such as atherosclerosis, osteoarthritis, Alzheimer's disease, type 2 diabetes or cancer; the chronic dysfunction involved in each of these conditions is ultimately caused either by the presence of harmful signals that overwhelm a tissues' ability to maintain proper structure or function, or the absence of healthy signals that promote tissue recovery. Lifestyle medicine, at its core, is simply a way to promote those signals designed to protect all tissues and organs. Two important layers of resistance against poor lifestyle signals are built within each system; they are Physiological Resilience and Metabolic Reserve.

Physiological resilience describes the capacity of each cell or organ system to withstand the necessary (and immediate) changes that create the rhythm of a healthy organism. The ability of arteries to dilate when blood flow increases, the ability to quickly dispose of excess glucose after a meal, or the ability to increase and decrease cortisol when a stressful situation arises and resolves are just a few examples of physiological resilience. Each of these systems, and thousands more, are like coiled springs or rubber bands capable of being stretched for a particular physiological purpose, then designed to snap back to their original status, ready for the next physiological challenge. Some systems are stretched and snap back in an instant, such as nerve conductivity, others in hours, like blood-glucose control. Some follow a circadian cycle, such as the HPA axis, and others a monthly cycle, menstruation for example. Homeostasis is really not a "steady-state" but really the controlled rhythmic fluctuation of thousands of interrelated systems that oscillate from physiological challenges.

When inappropriate or overwhelming signals begin to overpower our physiological resilience, the stretching of that system does not immediately resolve. Perhaps the best example of this is the slow progression of impaired glucose tolerance caused by insulin resistance, which is sometimes referred to as the "metabolic continuum." Note in Figure 1 the "stretching" of the post-prandial glucose affects the area under the curve (AUC) of glucose much more than the change in the eventual fasting state. By the time a patient is deemed to be a "type 2 diabetic,"

their ability to reach normal fasting levels is compromised by changes in peripheral insulin sensitivity and pancreatic insulin capacity; the rubber band has been stretched too far. If physiological resilience defines the immediate capacity of cells, tissues and organ systems to respond to changes in physiological need, metabolic reserve defines the long-term capacity of tissues and organ systems to withstand

Blood Glucose Excursions in People with Type 2 Diabetes, Impaired (IGT) and Normal Glucose Tolerance



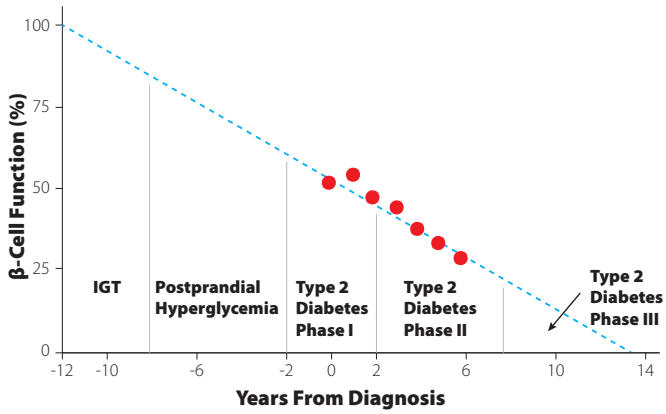
(Figure 1)

repeated (chronic) changes to physiological needs. It is, in essence, the stored-up "reserve" available for each metabolic and organ system to maintain and rebuild its physiological resilience. As with any reserve, its capacity is vulnerable to depletion; but also capable of being resupplied and strengthened.

For instance, related to the day-to-day physiological resilience that permits glucose disposal is the long-term metabolic reserve within pancreatic beta cells. In fact, by the time a person is diagnosed with type 2 diabetes, up to half of their pancreatic beta cell function is already depleted. Peripheral insulin sensitivity and beta cell function are critical long-term metabolic reserve functions that can influence meal-to-meal physiological function and, as most clinicians now know, alter the metabolic functions of nearly all tissues. The reason nearly every chronic disease is made worse with type 2 diabetes is because the same metabolic and oxidative stresses triggered by insulin resistance and hyperglycemia that destroy beta cell function are at work in all other tissues as well.

Another well-known system that shows the principles of physiological resilience and metabolic reserve is bone mineral density. The constant back- and- forth of bone remodeling (osteoclast/osteoblast function) is designed to respond to the immediate circumstances of the individual; such as nutrient availability, hormone status and weight-bearing activities (physiological resilience). During menopause, internal and external signals favor changes that lead to a loss in bone mineral density. While the eventual outcome for the individual is partly determined by the immediate changes in bone mineral density and strength, the outcome is greatly influenced by the level of bone mass built during the previous 50 years (i.e., peak bone mass). The level of bone mineral (metabolic) reserve available when menopause starts will greatly impact the likelihood of osteoporosis and, while building some bone mineral reserve can be done after menopause, the ability to build the reserve in the years between puberty and 30 is far greater.

Building metabolic reserve, then, is one of the core concepts for preventing all chronic diseases. Think of these reserves: antioxidant reserve, detoxification



capacity, insulin sensitivity, cellular/mitochondrial energy, bone mineral storage, neuronal plasticity and even gut microbial reserve. Assessing and supporting these core reserves should be the ultimate goal of lifestyle-based chronic disease prevention. Therapies should be designed to assess these reserves and prioritizing therapies known to specifically support the tissues and organ systems for which the individual shows weakness.

Using Lifestyle Therapies as Intervention

Today, any lifestyle-based approach must recognize that many patients are well down the road of manifesting outcomes of organ dysfunction, where the prevention of chronic disease seems to be too late. It should be noted, however, that the same processes (building metabolic reserve and supporting physiological resilience) that protect tissues from the onset of chronic damage are even more vital when these tissues are metabolically stressed to the point they manifest dysfunction and result in the diagnosis of a clinically defined "disease." While clinicians and patients may need to augment their lifestyle interventions with targeted therapies or even rescue medication, the promotion of the healthy signals designed to elicit healthy outcomes (lifestyle signals) should always remain the foundation of programs designed to support long-term health outcomes.

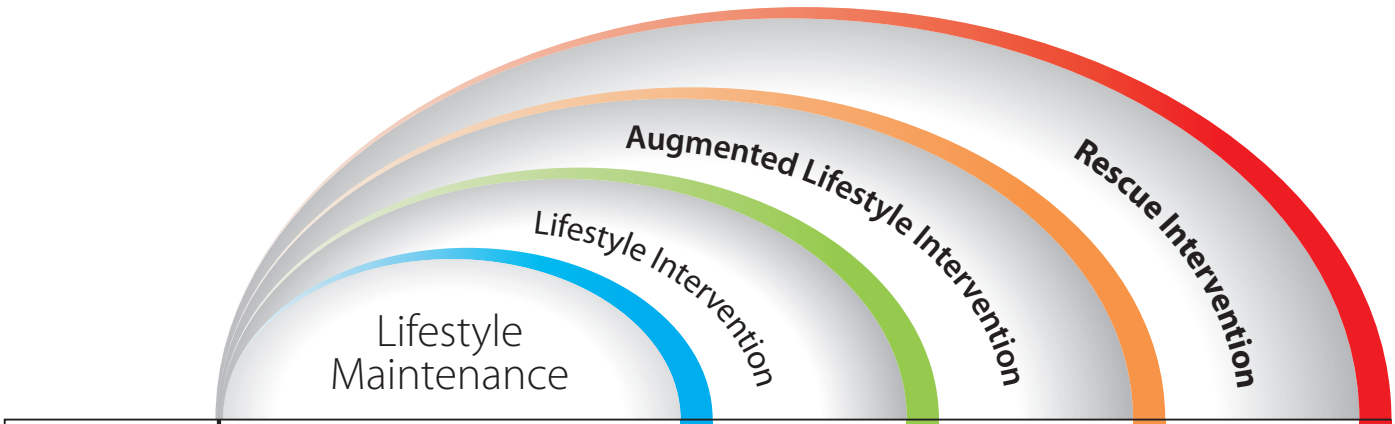
Therefore, implementation of lifestyle medicine within a wide range of clinical starting points requires a hierarchy of therapies that span early prevention to late intervention. Here, we describe a Prevention to Intervention Hierarchy,

that is defined by 4 points along a continuum: Lifestyle Maintenance, Lifestyle Intervention, Augmented Lifestyle Intervention and Rescue Intervention. Here is a brief description of each:

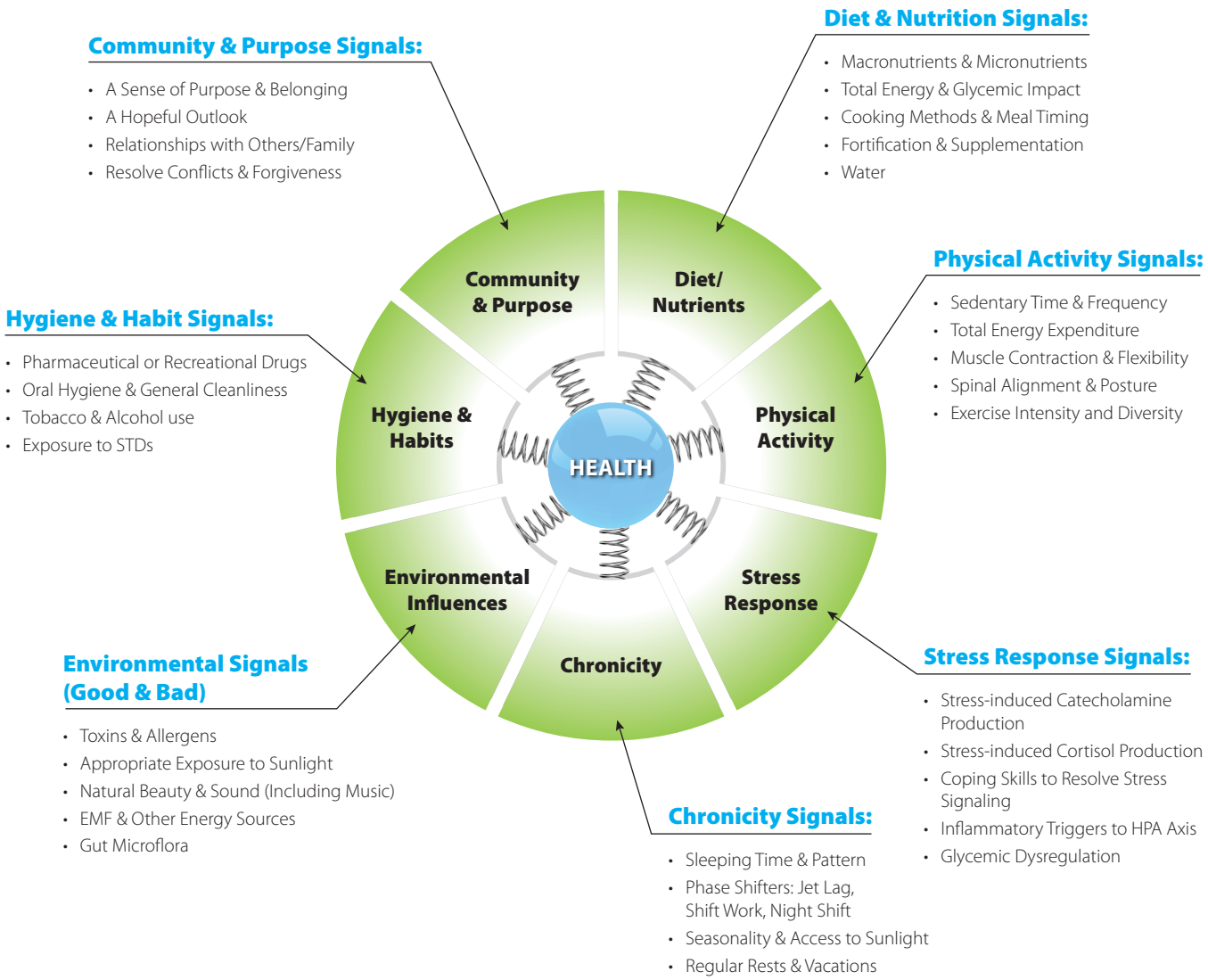
Lifestyle Maintenance: This is simply the use of healthy life signals that maintain our metabolic reserve: eating right, keeping fit, sleeping well and avoiding signals that sap our vitality. The longer we can rely on a prudent life maintaining our health, the more reserve we build against chronic disease. Essentially, this is "Life as Medicine."

Lifestyle Intervention: This is what most clinicians and patients think about when they describe lifestyle medicine or therapy. Using the same signals that maintain our health, we are now more intentional about the therapy, increasing both the specificity and dose of the lifestyle signal. For instance, we are much more specific about dietary choices or changes, becoming fit, modulating stress and kicking bad habits known to be harmful to our health. There is still a strong reliance on the body's capacity to build or rebuild metabolic reserve, but there is a greater focus on risk prevention based on a person's specific vulnerability.

Augmented Lifestyle Intervention: Having discovered many of the specific mechanism(s) that link our lifestyle decisions to our health, we are now capable of designing therapies to target those mechanisms. These therapies are the heart of integrative medicine—things like nutritional and nutraceutical therapy, osteopathic or chiropractic manipulation, detoxification protocols and many more. For instance, recognizing omega-3 fatty acid consumption as one of the key signals within a healthy diet allows us to augment, through supplementation, the consumption of concentrated omega-3 fatty acids. Clinicians may find the majority of their patients will require augmented therapies for a long time, perhaps indefinitely, depending on the severity and history of the chronic diseases experienced by the patient.



	Lifestyle Maintenance	Lifestyle Intervention	Augmented Lifestyle Intervention	Rescue Intervention
Vitamin D & Bones	Sunlight: Work and Play	Sun Therapy/ Diet	Vitamin D Supplement	Osteoporosis Drugs
Antioxidant Defense	Wholesome Diverse Diet	↑ Fruits & Veggies	Supplement Vitamins C and E/ Quercetin etc.	N/A
Back Health	Normal Physical Activity	Exercise and Flex Training	Chiropractic/ Physical Therapy	Surgery/Pain Medications
Joint Health	Physical Activity/Hydration	Weight Loss/ Flexibility	Glucosamine/ Chondroitin Sulfate	Drugs, Knee Replacement
Cancer Prevention	Wholesome Diverse Diet	↑ Fruits & Veggies (Cruciferous)	Supplement Sulforophane or I3C	Antineoplastic Drugs
Detoxification	Wholesome Living	Avoidance/ Organic Focus	Supplement Agents that ↑Detoxification	Drugs to Combat Toxin Symptoms



Rescue Intervention: These are interventions now commonplace in our modern medical system, drugs and surgery, used to rescue the patient and to prevent organ damage, major debility or death. However, when the same paradigm of rescue interventions is used in the attempt to ameliorate chronic conditions, a person's metabolic reserve and physiological resilience are rarely enhanced. In fact, they are often depleted. Since the pharmaceutical burden weighs heavily on most patients diagnosed with chronic diseases, wise clinicians who intend to use lifestyle therapies as the core of their intervention strategy must understand how to manage patients who have been treated with, or are currently on, these rescue therapies.

A Symphony of Signals from all Spheres of Life

If nearly every aspect of our physiology can be modulated by the lifestyle and environmental signals around us, we could easily define thousands of different signals. In a clinical setting, this vast number of potential variables can overwhelm the therapeutic decision-making process. It is helpful, therefore, to consider signals that can be defined in a limited number of categories. For this purpose, it is helpful to define seven categories, or spheres, of signals based loosely on origin. Since some lifestyle decisions are complex, they may include signals from multiple categories. As more research into the specific mechanisms of each signal is discovered, especially genomic and epigenetic signals, we now understand how many signals from diverse origins can promote a synergistic good (or bad) health outcome.

It is our hope that the leading clinicians, teachers, policy makers and parents of tomorrow will recognize the intrinsic healing capacity of the human body and will implement practices that allow future generations to reduce their risk of chronic disease by living in a way that preserves their metabolic reserve, buffering against depletion of their physiological reserve, reaching their goal of optimal health.

[1] The Core Philosophy can be found in Dr. Guilliams' book, The Original Prescription: How the Latest Scientific Discoveries Can Help You Leverage the Power of Lifestyle Medicine. Point Institute. 2012

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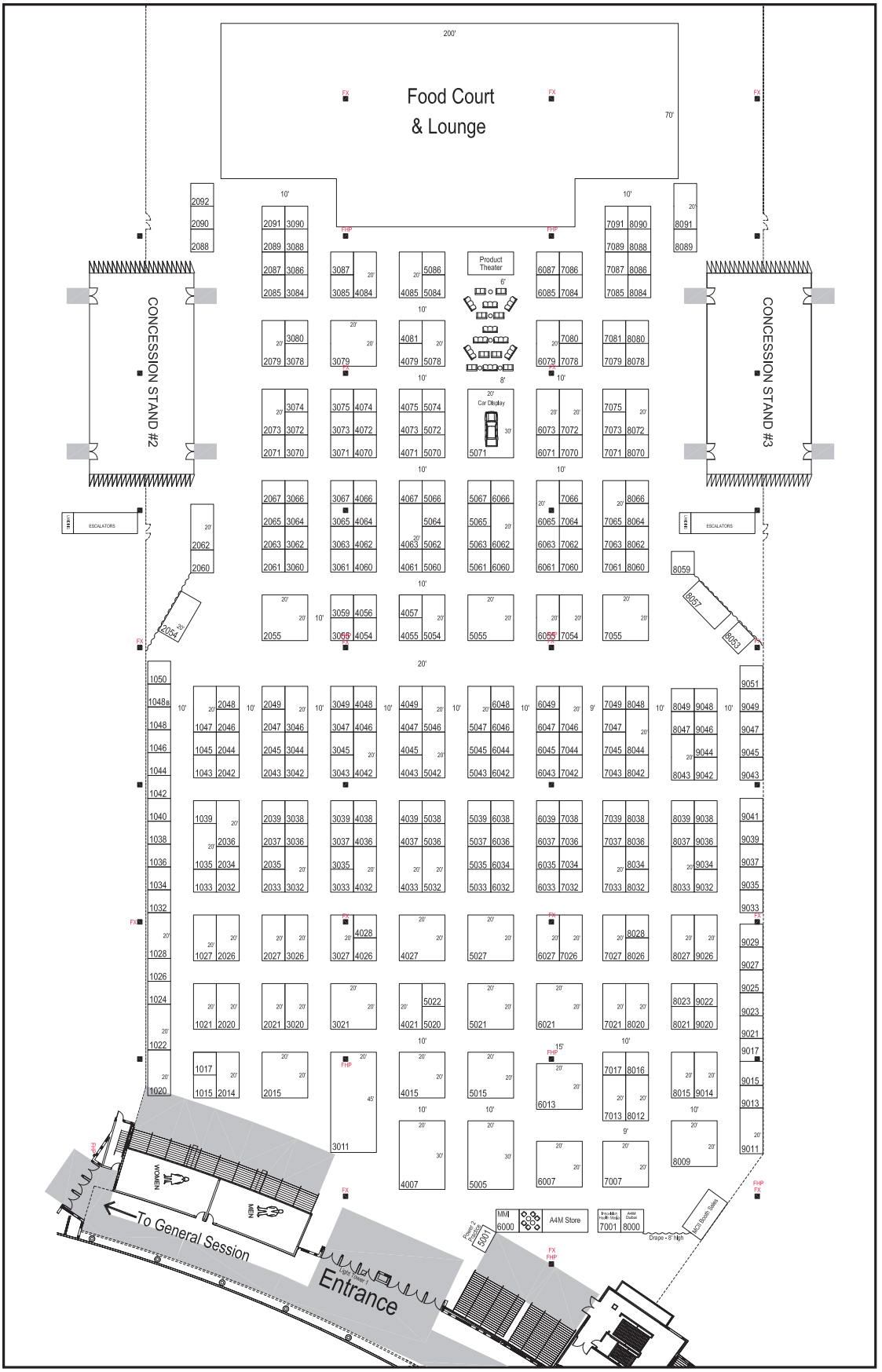

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Website: www.parkrx.com
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PARKWAY CLINICAL LABORATORIES • Booth #5078

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Email: info@parkwayclinical.com
Website: www.parkwayclinical.com
Parkway Clinical Laboratories is a full service CAP accredited, CLIA-certified laboratory providing basic & specialized tests for more than four decades with a special focus on supporting anti-aging and wellness providers in the initial diagnosis and ongoing care of patients suffering from complex chronic diseases, nutritional deficiencies and advanced cardiovascular risk. We provide all supplies to collect, store and transport specimens. Through our team of certified phlebotomists, PCL also offers on-site blood draw service. Reports are available within 24-48 hours of specimen receipt.

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Ph: 212.213.6155 • F: 212.213.6188
Email: pathmedical@aol.com
Website: www.pathmed.com
Eric Braverman, MD author bestselling books Younger You, Younger (Thinner) You, The EDGE Effect and more. As President of the PATH Foundation he has co-authored numerous studies with commissioners of health, generals, the director of NIDA, etc. He is the director of PATH Medical and THN. Vitamins, information, and more available at our booth.

PATIENT ONE • Booth #3039

120 Broadhollow Rd., Ste. 2, Farmingdale, NY 11735 USA
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Email: info@patientoneformulas.com
Website: www.patientoneformulas.com
Patient One® MediNutritionals is a premium line of science-backed dietary supplements, available exclusively through healthcare professionals. Patient One's diverse range of medinutritional formulas feature today's most advanced ingredients and are supported by extensive clinical data. Their products span health categories including cardiovascular support, immune support, digestive support, bone and joint support, cognitive support, endocrine support, healthy aging, detox and weight management, emotional wellness, eye care, men's & women's health and more.

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Email: nqader@pccarx.com
Website: www.pccarx.com
At PCCA, we help practitioners and pharmacists use compounding to create personalized medicine that makes a real difference in patients' lives. Our products give pharmacists innovative tools, while our education and support provide pharmacists and practitioners alike with invaluable resources. PCCA works with our member pharmacies to help them deliver compounding therapies for a wide range of conditions encountered in pediatrics, women's and men's health, otolaryngology, pain management, dermatology (wound and scar) and palliative care.

PERIGEE MEDICAL • Booth #3087

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Website: www.gomegaspore.com

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Email: scocilova@physicianslab.com
Website: www.physicianslab.com

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Email: info@power2practice.com
Website: www.power2practice.com

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Website: www.precisionhormones.com

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QOL Labs was established in 1998 with the mission of providing safe and efficacious nutraceutical products to improve the quality of life (QOL) of our customers. We believe that the only way to determine the efficacy and safety of a compound is to test it in well-designed human clinical trials. And the only way to deliver such efficacy is to use the dosages consistent with those trials. For that reason, we became the only nutritional supplement company to guarantee that 100% of the ingredients used in our formulas are backed by human studies and delivered in dosages consistent with the clinical research.

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641 Lexington Ave., 27th Floor, New York, NY 10022 USA
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Email: info@restorsea.com
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Restorsea launched in October 2012. All products are natural, clinically proven, and anchored by its proprietary formulations using Aquabeautine XL® an enzyme released by baby salmon. Restorsea products exfoliate without inflammation and are as effective as the leading brands. As such, Restorsea has received acclaim from outlets including Elle, Shape Magazine, NYTimes, Lucky, Marie Claire, Harper's Bazaar and InStyle.

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Website: www.rheinlabs.com
Rhein Consulting Laboratories is an independent, research-driven facility specializing in state-of-the-art, cost-effective assessment of sex and adrenal hormones. Assayed in urine, rather than saliva, RCL's results are clinically relevant, accurate, reproducible, and widely accepted scientifically.

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Sovereign Laboratories LLC is a Sedona-based company dedicated to developing natural products that provides the public with the best solutions for optimal health. Our company founder Douglas Wyatt, was the founder and owner of Symbiotics who introduced scolostrum for world wide human consumption.

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As a globally organized company with swiss origin, we develop, manufacture and distribute innovative products worldwide in the field of complementary and alternative medicine, particularly in bionic solutions. Presently the world's first and only android based Comprehensive Health Interface, the Omnium1 -, the world wide Nr.1 iMRS PEMF systems for home use, which have been proven over the past 2 decades, and a wide range of brand-new options from light and sound –research (brain wave entrainment).

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Email: info@tasciences.com
Website: www.tasciences.com
T.A. Sciences is a consumer health and wellness company dedicated to discovering and creating groundbreaking products through telomerase activation. Based on the science of telomere biology, the company is solely focused on research-based solutions to rejuvenating the aging process at the cellular level.

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1311 Church St., San Francisco, CA 94114 USA
Ph: 866.778.2646 • F: 866.778.2646
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is a magna cum laude graduate of Boston University Medical School. She completed an internship in pathology at Massachusetts General Hospital followed by an internship and residency in internal medicine at the Brigham and Women's Hospital in Boston. She is trained in hormonal rebalancing and targeted amino acid therapies (TAAT) with neuroscience and has an extensive background in nutrition, applied physiology and sports medicine. Dr. O'Neil-Smith is currently collaborating on a book on optimum health and performance and nutrigenomics with Harper Collins.

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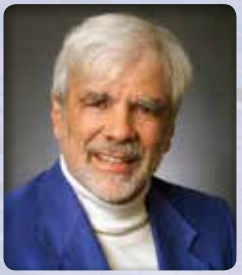

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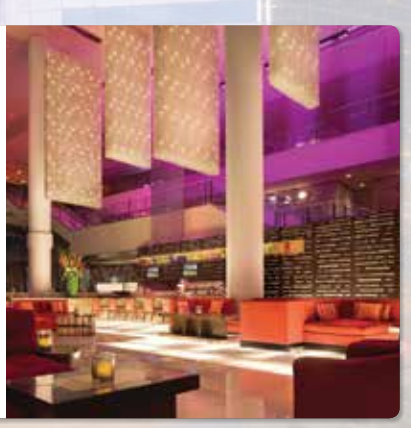

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