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# Anti-Aging

## MEDICAL NEWS

Winter 2015



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# 24<sup>TH</sup> ANNUAL WORLD CONGRESS ON ANTI-AGING MEDICINE



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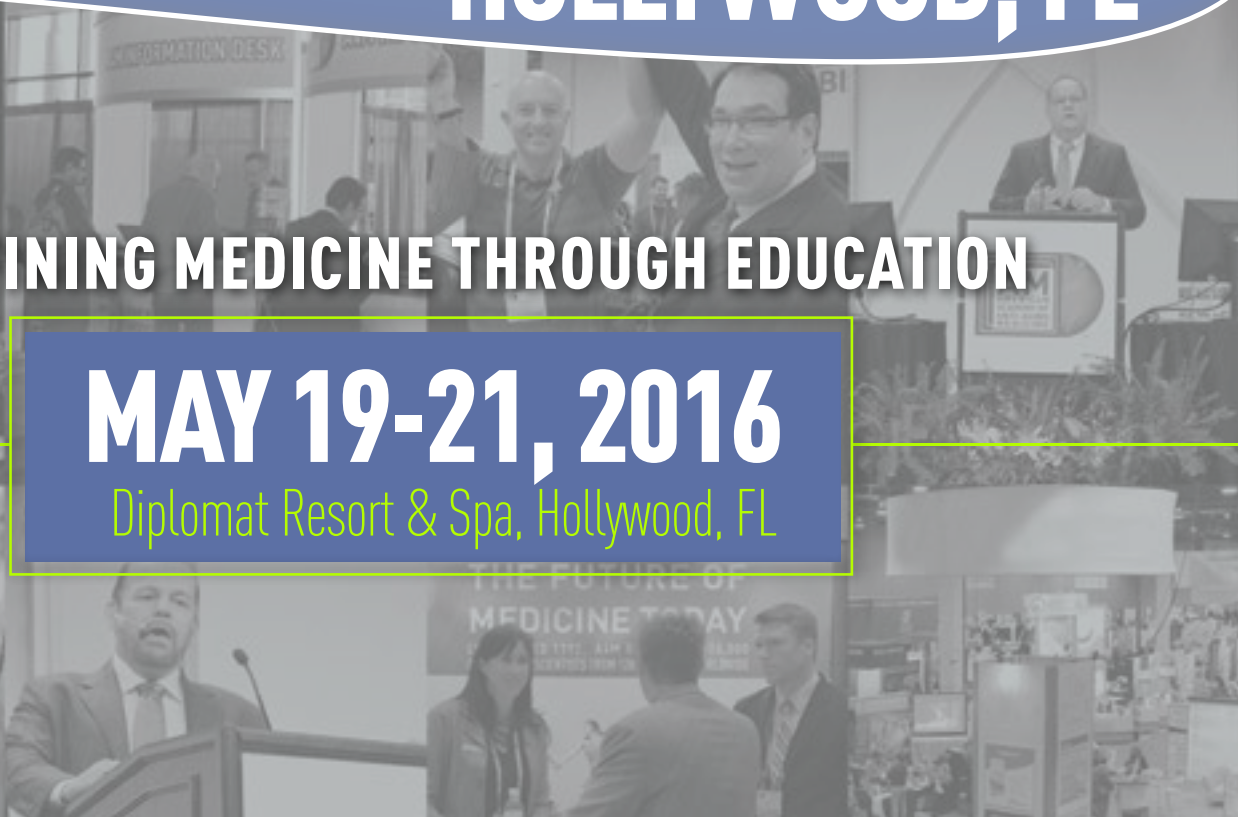
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Throughout this dynamic 4-day event, you have the opportunity to immerse yourself in interactive discussions, connect with like-minded medical professionals and most importantly, experience the evidence-based research presented by the leading minds in preventative medicine. This particular event offers an elite medical education platform where medical practitioners of all specialties are redefining medicine. You will leave this conference with new clinical protocols and skills, and the ability to immediately implement them into your practice, improving patient care and maximizing your practice's potential.

Anti-Aging Medicine is synonymous with the terms Healthy Aging or Personalized Medicine. Regardless of the term used, it can be defined as a medical approach that utilizes cutting-edge science and personalized care to prevent and reverse the effects of age-related diseases. It is the occurrence when healthcare practitioners treat the actual patient, and not just the symptoms that appear on the surface. This concept is of heightened attention as the global Anti-Aging market is estimated to reach over \$400 billion by 2030.

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 transforming the future of medicine and we salute you for leading the evolution of the healthcare industry.

With warm regards,



Ronald Klatz,  
MD, DO  
President, A4M



Robert Goldman,  
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# PROFESSIONAL MEDICAL EDUCATION WORKSHOPS (NON-CME)

## Thursday, December 10<sup>th</sup> - 6:15pm

Understanding Rosacea & Acne Rosacea:  
NEW Medical SPA Perspective  
Location: Marco Polo Ballroom

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Manon Pilon, Author

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Micro-Needling, RF Fractional Resurfacing &  
Clearista Facial, a Revolutionary New Procedure  
Location: Casanova Ballroom

Presented by:  
Garry Lee, MD

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## Friday, December 11<sup>th</sup> - 6:15pm

Personalized Medical Nutrition Therapy  
and the Art of Methylation  
Location: Veronese Ballroom

Presented by:  
John Troup, PhD  
Bridget Briggs, MD  
Lisa Bastian, NTP

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Lyme Disease:  
What to Do if Antibiotics Fail  
Location: Titian Ballroom

Presented by:  
Silvia Binder, ND, PhD  
Bjørn Øverbye, MD

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Hormone Dosing Protocol with  
Dr. Pamela Smith and PCCA  
Location: Veronese Ballroom

Presented by:  
Pamela W. Smith,  
MD, MPH, MS

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Treat Symptoms and Reduce Cancer Risk by  
Improving Estrogen Metabolism  
Location: Titian Ballroom

Presented by:  
Jennifer Landa,  
MD, FAARFM, ABAARM

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Short Telomeres:  
Treatment Prevention Protocol  
Location: Bassano Ballroom

Presented by:  
Mark Houston, MD, MS

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## Saturday, December 12<sup>th</sup> - 6:30pm

Largest Network of Physicians Share How  
BodyLogicMD Transformed Their Careers  
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Presented by: Pamela W. Smith, MD, MPH, MS

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## Saturday, December 12<sup>th</sup>

12:30 PM

**Optimize Your Epigenetics to Look Younger, Longer**  
Presented by: Carlos Orozco, BSc, MSc, MD, ND, PhD, Adv Dip Nat Med

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**Fitness In and Out**

Presented by: Alan Goldberg, Cynthia Rothrock, Don Wilson and Carey Tagawa

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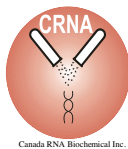
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**EXHIBIT HALL**

**December 13<sup>th</sup> • 12:00 PM**

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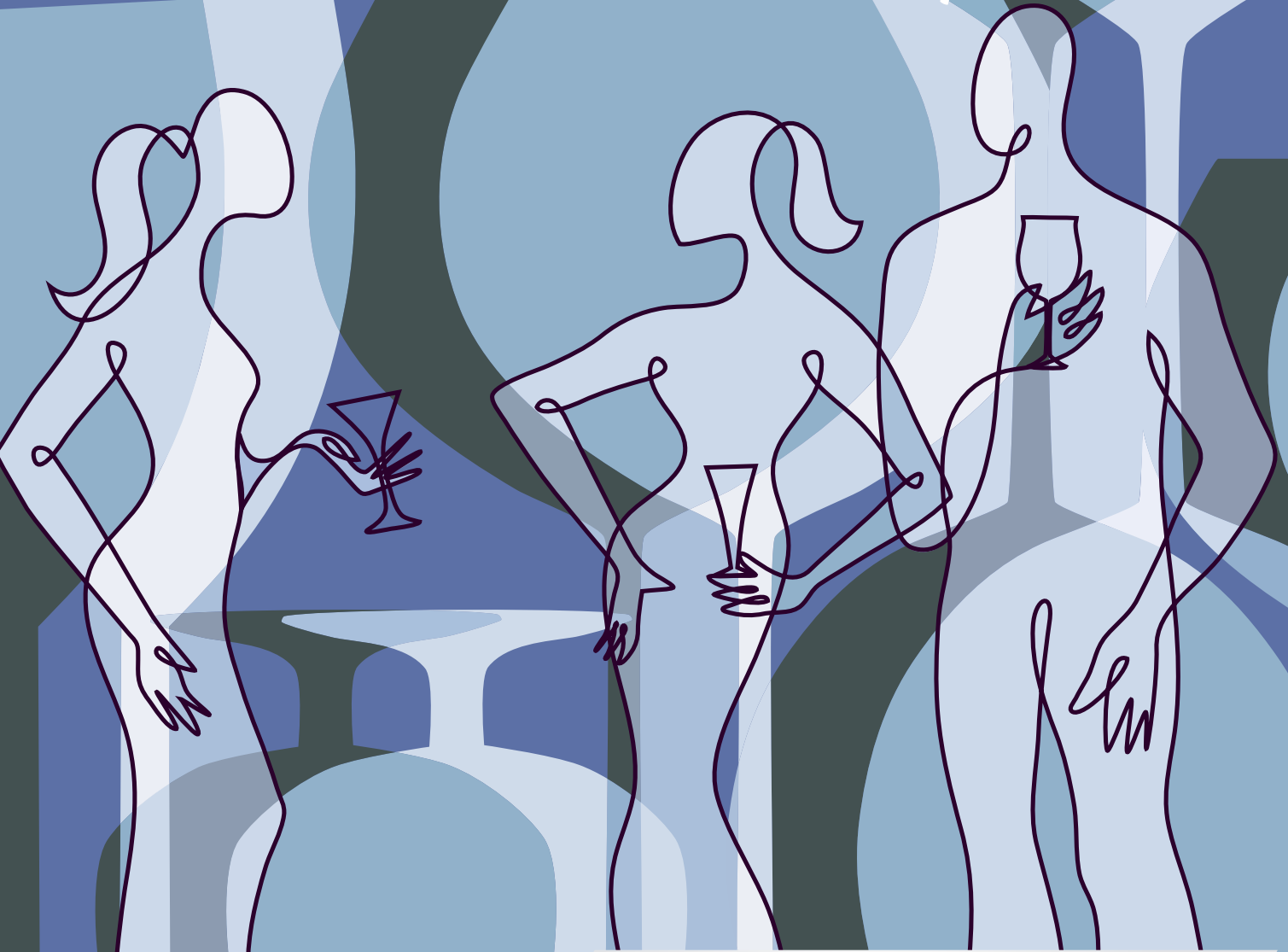
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SATURDAY, DECEMBER 12<sup>TH</sup>

4:30 - 6:30PM

LOCATION:  
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# Management of Obesity by Modulating the Gut Microbiota

By Stephen F. Olmstead, MD

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## Introduction

Obesity is a growing pandemic. The World Health Organization estimates 1.4 billion adults worldwide are overweight.<sup>1</sup> Over 10% of the world's population is obese. Obesity is an inflammatory disease associated with a cluster of disorders such as insulin resistance, type 2 diabetes mellitus, hypertension, dyslipidemia, cardiovascular disease, and metabolic syndrome.<sup>2</sup> Once simplistically considered a disorder caused by an imbalance of caloric intake versus energy expenditure, obesity is now viewed as a complex, multifactorial disorder. One factor revealed to play a key role in weight homeostasis and obesity is the composition of the intestinal microbiota.<sup>3-5</sup> Observations linking intestinal dysbiosis to the pathogenesis of obesity raise the exciting potential that probiotic and prebiotic nutritional supplements can be used to promote weight loss.

## The Gut Microbiota and Obesity

With tens of trillions of microorganisms and a collective microbial genome (microbiome) 150 times the size of the human genome,<sup>6</sup> the gut microbiota profoundly impacts human physiology and governs energy homeostasis and inflammation in ways that strongly influence the risk for obesity. It has long been known that the gut microbiota extracts energy from dietary substances indigestible by the host. Germ-free animals fail to gain weight normally.<sup>7</sup> The metabolic activities of gastrointestinal microorganisms contribute an estimated 30% of daily caloric energy.<sup>8</sup> However, gut microbial metabolism does far more than promote normal weight gain. In 2004 researchers revolutionized perceptions of obesity by reporting that intestinal microorganisms transplanted from normal mice into germ-free mice not only led to weight gain, but

increased body fat and insulin resistance despite calorie restriction.<sup>7</sup> Subsequent human studies found that the gut microbiota of obese subjects contained greater numbers of microbes in the phylum Firmicutes and fewer in the phylum Bacteroidetes than did matched lean controls.<sup>9</sup> Interestingly, after a carbohydrate- or fat-restricted low-calorie diet, the ratio of Firmicutes to Bacteroidetes in obese individuals approached that found in lean subjects. In mice, transplantation of gut microbes from genetically obese rodents into germ-free animals leads to significantly greater weight gain than microbiota from lean mice.<sup>10</sup> These results show gut microbial changes strongly contribute to obesity. Other animal studies, however, have found high-fat diets produce a marked expansion of intestinal Firmicutes populations and a decrease in Bacteroidetes suggesting the altered bacterial ratios are a secondary phenomenon.<sup>11</sup> The interplay between microbiota and diet is undoubtably complex. Microbial imbalances can both predispose to obesity and be caused, or exacerbated by, high-fat or high-sugar diets.

## Characteristics of Obesity-Associated Dysbiosis

The role of diminished Bacteroidetes numbers in obesity is controversial. While some researchers confirm reduced gut Bacteroidetes populations in obese humans,<sup>10</sup> others actually report increased numbers of Bacteroidetes in obese people.<sup>12</sup> Yet others have failed to find any association between Bacteroidetes and obesity.<sup>13,14</sup> The focus on phylum level ratios in obesity may be inappropriate. Phyla are very large taxonomic designations that may not adequately reflect





*Lactobacillus*.<sup>22</sup> Furthermore, *Lactobacillus* probiotics have been among the interventions shown to be associated with weight loss and improved glucose metabolism in both animal and human studies.<sup>25-28</sup> While it is clear that obesity, type 2 diabetes, and metabolic syndrome are associated with gastrointestinal dysbiosis, the microbiological disturbances are complex. A definitive link between obesity and relative proportions of Bacteroidetes and Firmicutes has yet to be established. The important microbiota disturbances appear to be at the genus and species levels and in reduced microbiota diversity.

## Mechanisms for Microbiota Regulation of Weight

Several plausible mechanisms whereby the intestinal microbiota can influence weight have been posited, including modulation of caloric extraction from foods. Certain bacteria, such as the Firmicutes, possess a greater array of genes encoding for enzymes to metabolize digestion-resistant dietary polysaccharides.<sup>4</sup> By increasing the energy harvest from otherwise indigestible foods, some microorganisms could contribute to weight gain. Despite an association between Firmicutes levels and obesity in animals, human studies do not provide compelling evidence of a link between particular bacterial phyla and weight or metabolic status. Studies of mono- and dizygotic twins reveal that core microbiome characteristics are shared within families and that expression of obesity-related genes can exist across a diversity of microbial assemblages.<sup>29</sup> Obese traits in humans appear more related to gene-level, functional microbiota characteristics than to any identifiable taxonomic profile.<sup>29,30</sup>

Another means by which probiotic organisms may influence body weight involves production of the bioactive fatty acid, conjugated linoleic acid (CLA). CLA is an umbrella term for a family of linoleic acid (LA) isomers containing conjugated double bonds. The most bioactive CLA isomers, *cis*-9, *trans*-11, *trans*-10, and *cis*-12, have been associated with a number of health-promoting effects including favorable modulation of blood pressure, serum lipids, immune activity, inflammation, and body composition.<sup>31</sup> Nutritional supplementation with CLA has been shown to reduce adiposity in both animal and human experiments.<sup>32-34</sup> Mechanisms postulated for the antiobesity effects of CLA include regulation of energy expenditure, adipocyte proliferation, fatty acid oxidation, lipogenesis, and lipolysis.<sup>35,36</sup> *Lactobacillus* and *Bifidobacterium* probiotics are capable of synthesizing CLA.<sup>25,37,38</sup> Administration of *Bifidobacterium breve* and LA to mice increases levels of *cis*-9, *trans*-11 CLA in the large intestine and liver by a factor of two compared to LA alone.<sup>39</sup> Likewise, strains of *Lactobacillus rhamnosus* fed to mice increase serum levels of both *cis*-9, *trans*-11 and *trans*-10, *cis*-12 CLA.<sup>37</sup> One small human study found administration of one trillion colony forming units (CFU) per day of

microbiota dysbiosis. High numbers of Prevotellaceae, a member of the phylum Bacteroidetes, are described in obese people.<sup>15</sup> Reduced numbers of Firmicutes<sup>15,16</sup> and increased numbers of *Gammaproteobacteria*,<sup>15,17</sup> are seen in weight loss after bariatric surgery. The focus on the phyla Bacteroidetes and Firmicutes ignores the potentially important role of other microbes such as *Bifidobacterium* in the phylum Actinobacteria. *Bifidobacterium* numbers are much higher during infancy in children who have a normal weight in later childhood than in obese children.<sup>18</sup> Obese children were also found to have higher fecal numbers of *Staphylococcus aureus* (Firmicutes) during infancy than did lean children.<sup>18</sup> *S. aureus* may trigger intestinal inflammation that contributes to obesity. Low numbers of bifidobacteria have also been described in overweight women compared to lean women. Low gut *Bifidobacterium* populations have been associated with excessive weight gain during pregnancy<sup>19</sup> and are depressed in people with type 2 diabetes.<sup>20</sup> Studies of weight gain with pregnancy correlate high intestinal numbers of *Bacteroides* (Bacteroidetes), *Clostridium* (Firmicutes), and *Staphylococcus* with being overweight before pregnancy and excessive weight gain during pregnancy.<sup>19</sup> Another feature common to both obesity and pregnancy is reduced overall microbial diversity.<sup>10,21</sup> Weight loss has been associated with increased levels of *Bacteroides fragilis* and total lactobacilli and decreased numbers of *Bifidobacterium longum* and *Clostridium coccoides*.<sup>22</sup> A subgroup of Firmicutes, the Erysipelotrichaceae, is significantly elevated in obese persons while another Firmicutes subgroup, the *Lachnospira*, is significantly depleted.<sup>15</sup> Low numbers of *Faecalibacterium prausnitzii* (Firmicutes) are present with type 2 diabetes in obesity while higher numbers are found in obese individuals without diabetes.<sup>23</sup> In obesity, robust populations of *F. prausnitzii* correlate with lower levels of inflammatory markers. The *Lactobacillus* genus is part of the phylum Firmicutes. Lactobacilli numbers have been observed to be higher in 8 of 20 obese adolescents compared to lean controls.<sup>24</sup> In contrast, weight loss induced by diet and exercise has been associated with an increased population of

*L. rhamnosus* increased serum levels of *cis*-9, *trans*-11 and *trans*-10, *cis*-12 CLA.<sup>40</sup> Elevation of probiotic-derived CLA has been associated with marked antiobesity effects in animals. Mice fed high-fat diets along with CLA-producing probiotics gain significantly less weight compared to mice fed high-fat diets alone.<sup>25,37</sup> The CLA produced by probiotics has also been shown to significantly blunt increases in blood glucose resulting from high-caloric intake and weight gain.<sup>25,37</sup> Probiotic CLA production may be an important mechanism to promote weight loss and mitigate the adverse metabolic effects of obesity.

One of the likeliest mechanisms by which probiotics regulate weight gain is by modulating inflammation. Obesity and common comorbidities such as diabetes, dyslipidemia, hypertension, and cardiovascular disease are associated with chronic, low-grade inflammation.<sup>41-44</sup> High-fat feeding in mice sufficiently elevates plasma levels of the pro inflammatory lipopolysaccharide (LPS) to induce significant body weight gain.<sup>45</sup> Endogenous LPS derives primarily from gram-negative bacterial cell walls. Translocation of LPS from the gut lumen into the body triggers production of inflammatory cytokines, chemokines, enzymes, eicosanoids, adhesion agents, and free radicals.<sup>46</sup> *Bifidobacteria* and *Lactobacilli* improve gut barrier function, directly bind LPS in the gut lumen, and reduce levels of gram-negative organisms in the intestinal tract through competitive exclusion, secretion of bacteriocins, and inhibition of bacterial adhesion.<sup>47</sup> Prebiotics show potential benefit in weight loss and mitigation of metabolic syndrome primarily by increasing gut *Bifidobacterium* populations. Animal studies indicate supplemental *Bifidobacterium* and *Lactobacillus* significantly reduce intestinal and plasma LPS levels.<sup>48,49</sup> In one study, rats subjected to hemorrhagic shock were pretreated with either oral *Bifidobacterium* or a saline solution. Postmortem analysis revealed plasma LPS levels were significantly lower in the *Bifidobacterium*-treated group.<sup>49</sup> LPS-lowering effects in experimental models have also been noted for *Lactobacillus* organisms such as *L. rhamnosus*.<sup>50</sup> Additional human research has confirmed a link between LPS-induced endotoxemia and impaired glycemic control. In healthy human volunteers, the rise in plasma LPS following injection of LPS produces significant elevations in plasma and adipose inflammatory cytokines and adipokines as well as a significant increase in insulin resistance.<sup>51</sup> While definitive conclusions cannot yet be reached, these data suggest manipulations of the intestinal microbiota favoring growth of *Bifidobacterium* and

*Lactobacillus* downregulates LPS-mediated inflammation that contributes to the development of obesity and metabolic syndrome.

## Prebiotic and Probiotic Support for Obesity

The emerging link between LPS-induced inflammation and obesity suggests manipulation of the intestinal microbiota to minimize LPS endotoxin exposure may be a rational approach to managing weight.

Healthy modification of the intestinal microbiota can be achieved by improving diet and/or probiotic and prebiotic supplements. Probiotics are living microorganisms that confer health benefits when consumed.<sup>52</sup> Prebiotics are nutritive materials that selectively support the growth and activity of beneficial microorganisms in the intestinal tract. Prebiotic properties have been best documented for digestion-resistant oligosaccharides such as inulin-type fructans and galactooligosaccharides.<sup>53,54</sup> Feeding inulin-type fructans to genetically obese mice significantly elevates cecal levels of *bifidobacteria* and *lactobacilli* while reducing intestinal permeability, plasma levels of LPS, inflammatory mediators, and fat deposition in visceral, epididymal, and subcutaneous adipose tissues.<sup>55</sup> Rats administered a standard diet enriched with 10% oligofructose for four weeks lose significantly more weight than rats fed the same diet without oligofructose.<sup>56</sup> There are few human intervention trials involving weight maintenance and prebiotics. In one study of adolescent males, intake of 8 g/day of inulin-type fructans was associated with less weight gain and reduced total fat mass, especially in subjects who maintained adequate calcium intake.<sup>57</sup> In another study, obese, dyslipidemic, premenopausal women were given daily doses of a syrup containing 0.14 g/kg of inulin-type fructans which led to a reduction in weight, body mass index (BMI), fasting insulin levels, and LDL-cholesterol.<sup>58</sup> In a blinded, randomized, controlled clinical trial examining the effects of prebiotics on weight loss, 21 g/day of oligofructose or an equicaloric control was administered to obese and overweight individuals. After 12 weeks, persons in the oligofructose group lost approximately 1 kg of body weight, most of it fat mass from the trunk region, while slight increases in weight and fat mass were recorded for the placebo group. Additionally, beneficial changes were observed for satiety hormones with an increase in peptide-YY and a decrease in ghrelin.<sup>59</sup> Preclinical and clinical data also support the use of probiotics for weight management.



Rats fed high-cholesterol diets along with the probiotic *Lactobacillus plantarum* experience significantly reduced gains in total body, liver, and fat pad weight.<sup>60</sup> Similar reductions in body weight gain are observed in rats administered high-fat diets along with *Bifidobacterium* compared to rats fed high-fat diets alone.<sup>61</sup> In humans, researchers have found 2 billion CFU/day of *Lactobacillus acidophilus* was associated with significantly more weight loss following Roux-en-Y gastric bypass surgery than in control subjects.<sup>28</sup> Women who received dietary counseling plus *L. rhamnosus* and *Bifidobacterium lactis* during their first trimester of pregnancy were significantly less likely to develop central adiposity after delivery than women who only received dietary counseling.<sup>62</sup> *L. rhamnosus* and *B. lactis* supplementation in pregnant women has also been shown to reduce the risk of gestational diabetes.<sup>63</sup> In a trial examining the effects of pre- and postnatal probiotics on the risk of overweight in children, 10 billion CFU/day of *L. rhamnosus* or placebo was given to pregnant women four weeks prior to delivery and, after delivery, for an additional six months to either breastfeeding mothers or to infants who were bottle feeding. The results, although falling shy of statistical significance, revealed a clear trend towards reduced weight gain in the probiotic group at four years of age.<sup>64</sup> And a randomized, double-blind, placebo-controlled study involving overweight adults evaluated the anti-obesity effects of the probiotic *Lactobacillus gasseri*. A fermented milk beverage with or without 100 billion CFU/day of *L. gasseri* was administered to 87 obese men and women. After 12 weeks, participants in the *L. gasseri* group experienced significant reductions in BMI, waist-to-hip ratio, total body fat mass, and abdominal visceral fat area compared to control subjects who experienced mild to moderate increases in each of these variables.<sup>65</sup>

## Conclusion

Mounting evidence indicates the intestinal microbiota contributes to the regulation of energy homeostasis, body weight, and glycemic control. Microbial organisms play an important role in extracting energy from the diet, producing bioactive substances that influence carbohydrate and lipid metabolism, and modulating inflammation. Efforts to link obese and lean phenotypes with particular microbial profiles have yielded conflicting data, but studies suggest obesity is associated with reduced microbiota diversity and diminished populations of *Bifidobacterium* and *Lactobacillus*. These two probiotic genera antagonize enteric gram-negative bacteria and reduce proinflammatory LPS. LPS-induced inflammation has been posited as a contributory factor to obesity, blood glucose dysregulation, and possibly other metabolic disorders. Attenuation of LPS levels may be one of the principal mechanisms by which probiotics favorably impact body weight and glycemic control. Clinical studies confirm *Lactobacillus* and *Bifidobacterium* probiotics are effective for weight management. Prebiotics exert an inhibitory effect on appetite by modulating satiety hormones and

facilitating weight loss. Future research should clarify the links between the microbiota and metabolic dysregulation and provide further insights into how manipulation of the microbiota may help stem the obesity pandemic.

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# 2016

## MARCH 3-6

San Francisco Marriott Marquis  
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- > A4M BHRT Symposium
- > MMI Module II: Cardiology
- > ABAARM/ABAAHP Written Exam



## APRIL 15-16

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# EVENT SCHEDULE

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## MAY 18-21

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Diplomat Resort  
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- > **24<sup>th</sup> Annual World Congress** (May 19-21)
- > ABAARM/ABAAHP Exams
- > Specialty Workshops (May 19)
- > MMI Module I: Endocrinology (May 19-21)
- > MMI Module VIII: Clinical Practice Protocols (May 19-21)

## JUNE 2016

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TBD

- > MMI Module V: Nutrition & Exercise

## JUNE 23-25

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San Diego Marriott Marquis  
San Diego, CA A4M

- > BHRT Symposium
- > MMI Stem Cell Module II
- > MMI Module XIV(B): Weight

## SEPTEMBER 21-24

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Hyatt Regency Dallas  
Dallas, TX

- > ABAARM/ ABAAHP Exams
- > A4M BHRT Symposium (Sept 21-24)
- > MMI Module III: Neurology (Sept 22-24)
- > MMI Module VII: Autoimmune Disease & Inflammation (Sept 22-24)

## SEPTEMBER 30 - OCTOBER 1

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Capitol Hilton  
Washington, DC

- > A4M Symposium

## OCTOBER 5-8

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Sheraton Boston Hotel  
Boston, MA

- > MMI Module IV: Gastroenterology
- > MMI Module VI: Toxicology & Detoxification

## DECEMBER 8-11

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Venetian/Palazzo Resort  
Las Vegas, NV

- > **24<sup>th</sup> Annual World Congress** (Dec 9-11)
- > ABAARM/ABAAHP Exams
- > MMI Module I: Endocrinology (Dec 9-11)
- > MMI Module VIII: Clinical Practice Protocols (Dec 9-11)
- > MMI Module XIV(C): Weight (Dec 9-11)

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## **Module II: A Metabolic & Functional Approach to Cardiovascular Disease**

The causes, mechanisms, diagnosis, and management of cardiovascular and cardiometabolic diseases including CHD, CHF, hypertension, metabolic syndrome, dyslipidemia, atherosclerosis, and renal disease are covered in this module. Topics include cardiovascular pathophysiology and biology, inflammation, oxidation, hormones, adipokines, stress, nutrition and nutrigenomics, glycemic control, environmental factors and toxicology, infections, and risk factor testing.

## **Module III: A Metabolic & Functional Approach to Neurology**

This module reviews the most recent developments in the field of neurology utilizing a metabolic approach to the prevention, management, and treatment of neurologic diseases. The course will cover pathophysiology and the role of neurotransmitters, inflammatory and degenerative disorders, neurovascular diseases, psychological and psychiatric syndromes, the gut-immune-brain connection, and healthy brain function.

## **Module IV: A Metabolic & Functional Approach to Gastroenterology**

Comprehensive metabolic, functional, and nutritional approaches to gastrointestinal dysfunction and disease are reviewed in this module. Physiology and pathophysiology, GI microbiome and dysbiosis, gut permeability, hormones, diet, inflammatory bowel diseases, celiac disease and gluten sensitivity, the gut-immune-brain connection, irritable bowel syndrome, and other digestive and glandular disorders are highlighted.

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## » **Module V: A Metabolic & Functional Approach to Nutrition & Exercise**

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## » **Module VI: A Metabolic & Functional Approach to Toxicology & Detoxification**

This module covers symptoms, disorders and diseases associated with exposures of heavy metals, pesticides, chemicals, drugs, nutrients, the natural environment, and other toxic causes of oxidative stress. This course describes the pathophysiology of toxic exposure, methods to prevent and avoid exposure including nutritional and lifestyle approaches, early detection, lab testing, and treatment protocols. Metabolic, digestive and antioxidative detoxification phases and processes are detailed.

## » **Module VII: A Metabolic & Functional Approach to Inflammation & Autoimmune Disease**

This module focuses on inflammatory disorders, autoimmune diseases, allergies, cancer, and the gut-immune-brain connection. Cellular and molecular biology of immunity, the cellular stress response, oxidation, genetic damage, inflammation, etiology of disease including environmental and lifestyle factors, and the risk for cancer development are reviewed. Clinical approaches to patient evaluations, testing, and disease management are provided.

## » **Module VIII: Clinical Practice Protocols**

This review course examines patient case histories with a range of metabolic symptoms, disorders, or diseases that are covered in Modules I-VII. This clinical intensive course provides the tools to prevent, detect, diagnose, treat, and manage a variety of patient conditions that are commonly and uncommonly observed in the clinical setting. The goal is to provide comprehensive metabolic, functional, and nutritional approaches towards disease management that enable the Fellow to practice Metabolic Medicine confidently and effectively.

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# Finding purpose in THE FUTURE OF MEDICINE

By Mary Caire, MD



I was only 19 years old, but I remember my grandfather's funeral like it was yesterday. The cathedral in New Orleans was overflowing with generations of people who literally owed their lives to Dr. Arthur Caire, III. He had been with them when they were born, taken them through broken bones and illnesses, and had even been by the side of many of their loved ones as they died. They were there to pay their respects and to say thank you.

I was deeply moved by the connection my grandfather had to each person there. That day was a pivotal moment which fueled my desire to become a fifth-generation doctor. Like him, I knew that I wanted to do my part to impact as many lives as possible.

I earned my MD from Louisiana State University (LSU) medical school, and trained at Charity Hospital, the largest public hospital in New Orleans. We took the toughest medical cases, regardless of a patient's ability to pay. The training was intense. I worked for five years as a combined Internal Medicine and Physical Medicine & Rehabilitation (PM&R) resident, achieving Chief Resident status in my final year.

I slept there. I ate there. I lived there. I often wondered if I was sacrificing too much of my personal life for one goal-- to be the best doctor

I could possibly be. My desire to help others never wavered.

I completed my residency and became Board Certified in PM&R. Within a year, I started my own business. **Medical school teaches us so much about what it takes to be a great doctor, but there is no time dedicated to showing us how to run a business.** Shortly after completing my residency, I had a crash course in business development... hiring staff, writing a business plan, buying equipment, marketing, and navigating my way through a maze of insurance contracts.

The realities of caring for people under the umbrella of managed care and insurance reimbursement forced me to see a large number of patients in a short amount of time. I was constantly frustrated that I didn't have enough time to truly know my patients on a deeper level.

I started my family at the age of 40 with the birth of my son, Andrew, and the following year, my daughter, Elizabeth. Becoming a mother gave me an even higher sense of purpose to help others and to make the world a better place. This awareness was heightened by the knowledge that my children would be raised in a more toxic world. I thought about my own long-term health in a whole new light, wanting to be

there for my children as long as possible.

This shift in my view of healthcare, paired with the changes in insurance and government regulations, made the business side of my practice more laborious and brought me back to my deep roots in medicine. **How could I have that same profound impact on my patients that my father and grandfather once had?** I asked myself over and over: What do I want my legacy to be?

Around the same time I began questioning my role in medicine, I received an enticing offer to join a hospital-owned Physical Medicine & Rehabilitation (PM&R) practice. Deep down, I knew that wasn't my answer. I began to look for other ways to help my patients. I remember one day in particular. I was at the surgery center performing interventional pain procedures, when another surgeon, Dr. Kevin Light, began describing his new training through The American Academy of Anti-Aging Medicine (A4M) and the conversion of his surgical practice into integrative medicine. I was intrigued. Could this be what I was looking for?

For weeks, I researched my options. With only hours left to sign the hospital-owned group contract, I made a bold choice – I chose to cut my salary and my clinical hours in order to work on the cutting-edge of science. I made the choice knowing that it would be virtually impossible to go back to the traditional world of medicine. I was re-invigorated by the possibilities ahead for me and for my patients. I chose to follow the path of integrative medicine.

I thought back to my father, my grandfather, and the doctors who came before them and how they built real relationships with patients. I knew this shift to integrative medicine would allow me to do the same. Ironically, embracing the future of medicine allowed me to reconnect to its past.

What happened next shocked me. My staff found other opportunities due to concerns about the future of my practice and their job security.

I was left to write a new business plan, design a new office space, hire new employees and complete a fellowship. I am as thankful for the challenges as I am for the opportunities.

Today, I am Fellowship Trained in Anti-Aging & Regenerative Medicine and Metabolic &

Nutritional Medicine, and certified by the American Board of Anti-Aging & Regenerative Medicine. I have a dedicated staff and a renewed vitality for my career. I no longer accept insurance and make a good salary focusing on the greater good of my patients. Today, my patients seek me out for my education and expertise, instead of coming to me because I am the closest doctor who accepts their insurance plan.

I practice precision medicine and embrace the role of DNA in my integrative practice. Precision medicine elevates integrative, holistic care by enabling a higher-level of patient knowledge. In addition to a patient's lifestyle, diet, health history, biomarkers, microbiome composition and metabolomics, we can now understand their unique genetics and genetic predispositions. We can pinpoint risks for specific diseases and help patients make choices that prevent the diseases from ever manifesting.

With integrative medicine's focused approach for doctors and DNA's power to motivate and inspire patients, we are poised to have more control over our health (our lives) than ever before.

As I immerse myself in the latest DNA research, I marvel at advancements that are revolutionizing health care today:

- **Individualized Medicine or Precision Medicine:** We are moving from prescribing a one-size-fits-all pill for a particular disease or disorder to creating individualized health programs based on a person's unique nutritional needs, genetics, and biochemistry.
- **Personalized Longevity Plans:** The child probably has already been born who will live to be more than 150 years old. Therefore, we need to switch our thinking from how to stop disease and refocus on how to maintain health so that people over 100 can live active, productive lives and contribute to our society's success.
- **Big Data and Predictive Analytics:** Matching a particular therapeutic plan to a specific genomic profile is a big data problem. Massive data storage and processing power is required to analyze data on therapeutic interactions with different genetic combinations. For example, just focusing on 20 genes is a calculation with  $4.3 \times 10^{67}$



possible combinations. Large, open big-data platforms can do what humans cannot do – analyze that data and take it to the next level by predicting outcomes and doing it almost instantaneously.

As doctors and medical professionals who embrace integrative medicine, we must blaze the trails for this amazing medical future.

I have already begun to reach out to genetic testing companies, big data companies, educational institutions, and policy makers to create partnerships and learning opportunities. My vision is to find the right combination of partners to create the healthy future I know we can for our patients and our families.

Moving forward into the unknown, I think doctors need to understand not only medicine, but also its role in society. We need to reach out to our policy makers, big businesses and the media to help them understand what could be if we all worked together to create a healthier nation.

As a fifth-generation physician, I proudly take my place alongside those who went before me, and am inspired by their dedication. I understand now that the real lesson they tried to teach me was not about a specific medical procedure or building a successful practice. It was about changing lives and making the world a better, healthier place.

.....

**D**r. Mary Caire is an integrative medical doctor, speaker, and author who is a leader in DNA-informed precision medicine. She is board-certified in the specialty of Physical Medicine and Rehabilitation and fellowship-trained in Regenerative and Metabolic Medicine.

Using a process called the “The Caire Method,” Dr. Caire practices personalized medicine. Her clients receive customized health restoration and preservation plans which are based on their own unique genetics, hormonal balance, and biochemistry.

Dr. Caire is founder and medical director of The Caire Institute, located in Allen, TX, where she trains other physicians and healthcare providers on her unique protocols and methodology, which promote optimal health through integrative medicine.

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# Lyme Disease:

## A MICROSCOPIST'S SEARCH FOR AN *antibiotic free solution*

**Bjørn Johan Øverbye, M.D., Norway**  
(edited by Silvia Binder, N.D., Ph.D., Germany)

The following article is not endorsed and/or supported by The American Academy of Anti-Aging Medicine. The purposes of this publication do not imply endorsement and/or support of any author, company or theme related to this article.



**W**e all know the official stories how Doctor Allan Steer identified tick-transmitted arthritis in Lyme County in 1972. We all rejoice how Steere sent ticks from the county to Swiss born entomologist Willy Burgdorfer in 1985 and asked him to search for parasites and how Burgdorfer identified *Borrelia* spirochetes in the gut of the tick and then the link was established. Lyme Disease caused by tick-borne *Borrelia*. Both became “immortalized”.

A few years prior to these glorious events that changed the way we have come to regard a number of previously unexplained diseases of organs, muscles, joints and the nervous system, I was bitten by a tick. The year was 1978 and practically no one in my country had ever heard about *Borrelia* although German microscopist Otto Obermeier had identified the spirochete as cause of disease as early as 1868. By 1908, it was baptized *Borrelia*. By 1920, it was wide spread knowledge in German speaking countries that bloodsucking ticks transmitted the spirochete and spread from furry animals to humans and between humans. By 1950, Germans identified *Borrelia* in blood form by dark field microscopy and gave Penicillium to the sick and they recovered.

Due to the ignorance of the times, I suffered untreated Neuro-Borreliosis and went through living hell for one year with no help whatsoever. Later I came to learn most Norwegian patients still do, due to reasons called the “Lyme-Borreliosis war” between antibiotic minimalists and more realistic doctors.

During the following decades, I suffered badly and tried every treatment in the book, mostly alternative therapies since the doors to hospitals were closed. In my search for help, I studied under German surgeon/microscopist/immunologist and natural therapist Wolfgang Grüger, M.D. I learned about blood symbiotic, pleomorphic microbes, blood pH and syphilis. No *Borrelia* was discussed! I later studied with Professor Henk Oswald in the Netherlands in 1994; the same story as in Germany. No *Borrelia* was discussed! Swimming spirochetes were dismissed as “something else”.

In 1999, I had a relapse of neurological symptoms and spent some years rebuilding myself with immunotherapy and electromagnetic therapy to which I was introduced in Germany.

In 2010, when taking a course at The Monroe Institute in Virginia, now 100% symptom free due to natural therapies, I “stumbled over” inventor, microscopist and electronic genius Jim Meisner to whom I am still in debt. I learned the real thing from him: *Borrelia*; when looking at live blood through his Olympus 50X Dark Field Microscope.

I also learned how he was able to kill *Borrelia* using a replicate of a machine made by French inventor George Lakhovsky and adapted later on by American microscopist genius Royal Rife. I had the honor to meet his friend whom he had cured from severe crippling Lyme disease in three months using this electro-magnetic wave equipment.

Inspired by such stories, a local businessman lent me a high quality dark field /phase contrast/immunofluorescence microscopy and I set out to do research.

Over the years, I have collected a photo library of 1,000 pictures taken from blood voluntarily donated by people who wanted to help me find an explanation for nature of things.

By 2012, I had acquired a 15,000 page library on topics such as ticks, vectors, ecology, microscope building, optics, microbiology, *Borrelia* and spirochete sciences, therapies, politics of medicine and not to forget electromagnetic science of curing sick and killing microbes! By 2014, it all materialized into the biggest book on the topic ever written in a Scandinavian language with the title: Sick from Tick Bite (Syk av Flåttbitt, Veiviseren forlag).

So what did I learn from the ill fate of suffering from the worst disease ever and bouncing back twice? The biggest evil on this planet is: Lack of true knowledge of nature of things and the consequences thereof!



But by curing my own evils, I came out as a humbler and more knowledgeable doctor.

First, I came to learn the nature of the enemy: a small spirochete of the magnitude of 1-2 RBC length (ca. 10-20  $\mu$ m). Under a Dark Field Microscope at 1,000 diameters of magnification it looks like a small, thin, white thread with the characteristic way of wiggling around. This is the "classical appearance" common to all spirochetes. I photographed

them live and with still photos from the most unexpected volunteers; even healthy ones.

Thus I came to see with my own eyes the phenomena so often not talked about: the healthy carriers! I followed the people's histories and came to see for myself how a tick bite infected them with spirochetes many years earlier, and how they stayed healthy. Yet, how later when neglecting their health or exposed to traumas or immunosuppressing drugs the spirochetes appeared from "nowhere" and caused symptoms related to Lyme disease or Neuro-Borreliosis.

I also came to see a host of other phenomena known as *Borrelia* pleomorphism: spirochetes roll up into small discoid forms, change to smaller antigen and wall-free L-forms more apt to escape the immune system. And I could see the phenomena called shedding; how spirochetes wiggle intensely and throw off immune-proteins meant to kill them.

By 2014, I visited world famous Dark Field Microscopy guru Dr. Bela Bozsik in Budapest who had photographed and filmed the blood of more than 100,000 Lyme-Borreliosis patients. His records were impeccable: He could match the best serum tests and sometimes discovered *Borrelia* in previous sero-negative sick patients. His Hungarian team had a 95% cure when steering their therapies with Microscopy and serum testing in parallel; 3-4 months of antibiotics plus immune supporting supplements was the secret.

During my second visit he offered me a job at a hospital in Hungary as a *Borrelia* physician to take care of foreign patients. I often wonder if I should have taken this job to learn more and increase my expertise and above all, help people who are sick with no answers given as I had experienced.

From Dr. Bozsik, I learned how *Borrelia* "blebs". I saw photographs of how spirochetes under attack would make small blebs, some empty as decoys from immune cells to chase in vain when a few other blebs

containing valuable DNA meant to grow into a new spirochete could escape. I saw cysts with rolled up spirochetes inside other cells, "nests of spirochetes" covered with biofilms and how spirochetes cover themselves with slime (biofilm) to make them "slippery" so that immune proteins could not stick to their surface antigens; And I saw film of spirochetes breaking in two to create two offsprings from one mother.

When asking Dr. Bozsik, he admitted somewhat unwillingly "Well, there is a chance that many therapies on the market that make people clinically healthy do not really kill off all the spirochetes, just lower the number of spirochetes to a level where the immune system can keep them down; turning previously sick into healthy carriers".

As a consequence of the mass of knowledge I gathered from two continents and years of travel, I came to see the enemy as far smarter than the doctors chasing them. It is not a simple "bug" to be killed off by 2-4 weeks of Penicillin or doxycycline. It is the perfect parasite honed by billions of years of evolution in the ecosystem meant to survive at all cost and serving the highest purpose of James Lovelock Gaia scenario: Invading as many carbon-based warm-blooded organisms; Stay when organisms get old or weak due to lack of proper food in the biotope; see to their premature weakening and eventual death thereby promoting the younger, better fed and more apt.

A parasite not designed to succumb to a political correct medical minimalism or even by antibiotic maximalists. In patient after patient, I saw the spirochetes come back. And with awe and a curious admiration I saw how they evaded the immune system and how serum tests came out negative in more cases than our official health system likes to believe.

Burdened by the mass of knowledge loaded upon my shoulders after years of search I ended up with a threefold strategy which I tested out on volunteers: Immune therapy activating the natural inborn defense we all have. The second part is sound wave therapy I learned about in the U.S. which activates the nervous system and our cells. The third part is based on my pre-medical studies in university-physics and electronics: the use of tuned focused pulsating electromagnetic fields enhancing immunity, the lymphatic system and balancing the nervous system by tapping into cellular communication pathways.

Does it work? Well, it worked for me and a great number of volunteers. These days, I have had the honor to be supported in my work by the people from Ondamed in Germany by doing a pilot project to use their electromagnetic generators to help people for a better outcome when suffering from Lyme where antibiotics have failed. Based on years of experience and research I have a feeling that this may be the future.



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Bjørn Johan Øverbye  
University of Oslo: physics and math 1966-69  
University of Oslo: Medical science 1969-1976;  
Certified M.D. since 1977.

Dr. Bjørn Øverbye has been practicing holistic medicine at his private clinic in Arendal in Southern Norway since 1977. His patient population is drawn to his clinic from all over Scandinavia and represents those who have been discarded by the official health system as incurable. Since 2001 he has worked mainly with hypothyroidism and metabolic disease and since 2010 he has also been working with Lyme patients.

He is the author of 8 books on topics such as Acupuncture (3 books), Taoist Self-Healing (1 book), Electromagnetic Therapy (1 book), Metabolic Diseases (1 book), and Tick Borne Diseases (1 book). Dr. Øverbye has done extensive research on biophysics of electromagnetism on the human energy system (published in Canada) and completed a study on fibromyalgia under the tutelage of Dr. John C. Lowe in USA published on [www.thyroidscience.com](http://www.thyroidscience.com). The last three years he toured Scandinavia and other parts of Europe lecturing on nutrition, anti-aging, the circulation system and hypothyroidism.

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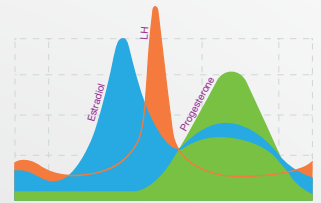
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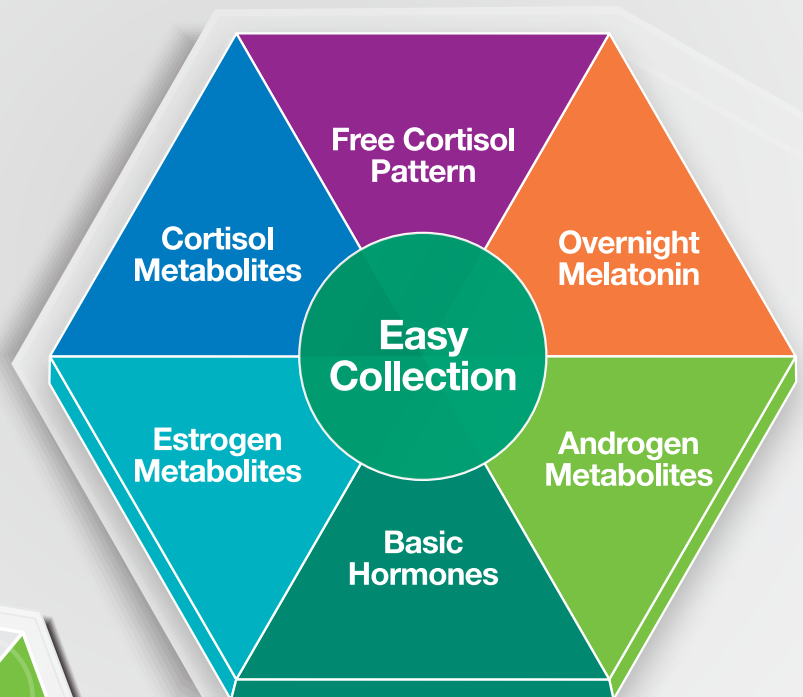


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### Thursday, December 10, 2015:

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| 7:30am – 6pm | Preconference Workshops |
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### Friday, December 11, 2015:

|                                 |  |
|---------------------------------|--|
| 7 am                            | General Session (Hall C)   |
| 10am                            | Exhibit Hall Opens   |
| 11:30am - 12 pm                 | <b>Physicians Lab - Exhibitor Hall - Booth 6034</b><br><b>Jennifer Landa, MD Book Signing - "The Sex Drive Solution for Women"</b>   |
| 1pm - 6pm<br>Afternoon Sessions | 1: The Beginning of Total Wellness<br>2: Toxic Soup<br>3: Buzzed on Inflammation<br>4: Healthy Affairs<br>5: Age Defying Treatments<br>6: Tools for Better Health  |
| 6:15pm - 7:05pm                 | <b>SPECIAL EVENING WORKSHOP!<br/>(Cocktails and Lite Fare)<br/>Sponsored By Physicians Lab</b><br><b>Q&amp;A Workshop with<br/>Dr. Jennifer Landa</b><br><b>"Treat Symptoms and Reduce<br/>Cancer Risk by Improving Estrogen<br/>Metabolism"</b> |

### Saturday, December 12, 2015:

|                                 |  |
|---------------------------------|--|
| 7am                             | General Session (Hall C)   |
| 10am                            | Exhibit Hall Opens   |
| 11am - 1pm                      | <b>Physicians Lab - Exhibitor Hall - Booth 6034</b><br><b>Enter Raffle to win FitBit - Winner Announced Sunday</b>   |
| 1pm - 6pm<br>Afternoon Sessions | 1: The Beginning of Total Wellness<br>2: The New Gut<br>3: Transitional Medicine<br>4: Healthy Affairs<br>5: Age Defying Treatments<br>6: Nutrition: Cause and Effects |

### Sunday, December 13, 2015:

|                                 |  |
|---------------------------------|--|
| 7am                             | General Session (Hall C)   |
| 10am                            | Exhibit Hall Opens   |
| 11:15am                         | <b>Physicians Lab - Exhibitor Hall - Booth 6034</b><br><b>FitBit Raffle Winner Announced</b>   |
| 1pm - 4pm<br>Afternoon Sessions | 1: Hormones and the Anti Aging Equation<br>2: Getting Personal<br>3: The Business of Medicine<br>4: Creative Wellness<br>5: Natural Beauty |



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# Demonstrated Improvement of Prematurely Aged Skin by Oral Intake of TA-65®

Fredric Stern MD, FACS,  
The Stern Center for Aesthetic Surgery, P.C.,  
Bellevue, WA 98004



Study sponsored by TA Sciences, Inc.

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## ABSTRACT:

Telomere attrition is the central hallmark of cellular senescence. TA-65® is a small molecule that has been shown to prevent telomere attrition. The main objective of this study was to evaluate cosmetic benefits of oral intake of TA-65® in subjects with prematurely aged skin (photo-aged skin). A randomized, double-blind, placebo-controlled study was carried out on thirty-five photo-aged subjects for sixteen weeks. Clinical assessments were performed at baseline and following four, eight and sixteen weeks of use. The VISIA® complexion analysis system (Canfield Scientific, Fairfield, NJ) showed that TA-65® may reduce pre-clinical damage, uneven pigmentation and wrinkles after eight weeks' daily capsule use. Histological analyses of biopsies revealed that the expression

of inflammatory cytokines declined, whereas elastin level increased after sixteen weeks' daily intake of TA-65® capsules, demonstrating the improvement of pre-maturely aged skin.

## INTRODUCTION:

Repeated exposure of ultra violet light results in photo-aging. In aging and photo-aging, human skin accumulates senescent keratinocytes and fibroblasts. Senescence not only limits the replicative potential of cells but also fuels inflammation associated with aging and photo-aging (Lasry and Ben-Neriah 2015). Thus anti-senescence compounds have tremendous potential as novel therapeutics. TA-65® is a small molecule telomerase activator derived from the Astragalus plant and has the ability to upregulate basal telomerase activity as well as reduce telomere attrition (Harley, Liu et al. 2011). This study was designed to test cosmetic skin improvement following the oral intake of TA-65®.





## OBJECTIVE:

The main objective of this study was to evaluate the cosmetic benefits of TA-65® capsules for skin care in subjects with prematurely aged skin.

## METHODOLOGY:

A randomized, double-blind, placebo-controlled study of thirty-five photo-aged subjects was carried out for sixteen weeks with clinical assessment performed at baseline and following four, eight and sixteen weeks of use.

The subjects were randomly assigned to either the placebo group or the treatment group. The placebo group took two placebo capsules per day for sixteen weeks. The treatment group took two TA-65® capsules (250 units each) per day for sixteen weeks. The placebo capsule did not contain the active ingredient, TA-65®, but is otherwise identical in formulation and packaging to the TA-65® capsule.



Figure 1: Visits at different time during study period

## VISIA® Complexion Analysis System:

The VISIA® complexion analysis system (Canfield Scientific, Fairfield, NJ) was used to objectively measure (Goldsberry, Hanke et al. 2014) wrinkles and brown spots at all indicated visits (Figure 1).

## PUNCH BIOPSY:

One subject from each group underwent a punch biopsy procedure, wherein a small skin tissue sample was taken at the beginning and end of the trial (16 weeks). The biopsy samples (3mm) were sent to the laboratories of Development Engineering Sciences, LLC (Flagstaff, AZ) which analyzed the molecular markers suggestive of the skin's health by nuclear counting, histology and Real Time – Polymerase Chain Reaction (RT-PCR). The following test methods were performed on the punch biopsy samples:

### Nuclear Counting:

The nuclear counting algorithm was used to count the number of cells present in the stratum basale and spinosum of the H&E stained sections. The algorithm was tuned per Aperio's algorithm user guide for nuclear quantification. Images acquired from the Hamatsu Nanozoomer digital slide scanner were analyzed using Aperio algorithms.

### Immunohistochemistry (IHC) Analysis:

Collagen 1, elastin and filaggrin presence in the dermis and epidermis, respectively, were quantified using a color deconvolution algorithm from digitally-scanned IHC slides. Collagen 1 was analyzed at a depth of 200µm below the stratum basale, into the dermis. Elastin was analyzed at a depth of 200µm above the hypodermis into the dermis. Filaggrin was measured in the stratum granulosum layer.

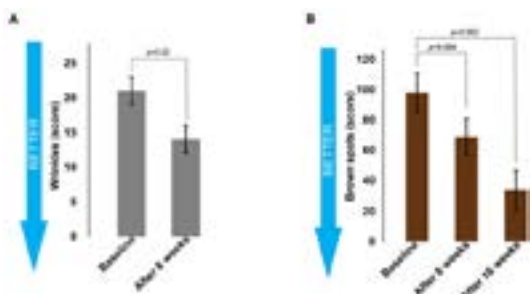
### Real-time PCR (RT-PCR):

IL-6, IL-8, TNF-α, MMP-1 and MMP-12 were measured by RT-PCR. Samples were analyzed in triplicate to calculate fold change (increase or decrease) in gene expression.

## RESULTS AND DISCUSSION:

The VISIA® complexion analysis system analyzed wrinkles and brown spots.

Compared to baseline, there was a significant reduction in the mean scores of wrinkles after taking TA-65® capsules for eight weeks (33% decline;  $p=0.02$ ). The mean scores (S.E) at baseline and after eight weeks were  $21 \pm 2$  and  $14 \pm 2$  respectively (Figure 2A). This result indicates that oral intake of TA-65® capsule can effectively reduce facial wrinkles.

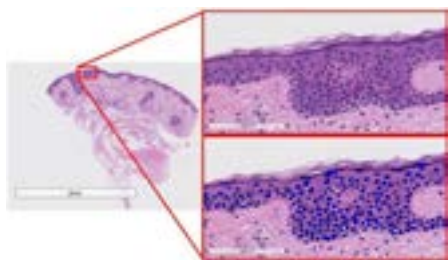


**Figure 2: Cosmetic benefits of TA-65® capsule.** Daily intake of TA-65® capsule reduced wrinkles by 33% after 8 weeks (A). Brown spots declined by 30% after 8 weeks and 65% after 16 weeks compared to baseline (B). p-values are estimated by the t-test.

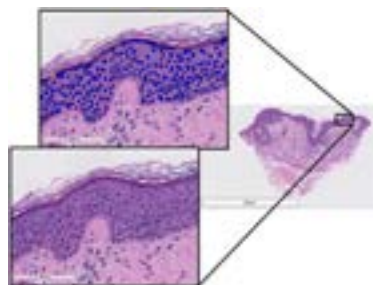
There was a significant reduction in the mean scores of brown spots after the oral intake of TA-65® capsules for eight weeks (30% decline;  $p=0.008$ ) and sixteen weeks (65% decline;  $p=0.002$ ). The mean scores (S.E) at baseline, eight weeks and sixteen weeks were  $98 \pm 13$ ,  $69 \pm 12$  and  $34 \pm 13$ , respectively (Figure 2B). These results indicate that oral intake of TA-65® capsules can diminish uneven pigmentation.

## Histopathology Findings:

Histopathology comparisons between active vs. placebo treatment sites at the sixteen week time point did not reveal any noticeable differences with respect to acanthosis, spongiosis, chronic inflammation, hyperkeratosis, epidermal mononuclear infiltration, or dermal edema. No trend change existed for prominence of any of these characteristics between active and placebo treated samples. Representative histopathology is shown in figure 3.



**Figure 3: H&E staining of punch biopsy at baseline in TA-65® group.** No acanthosis, spongiosis, chronic inflammation, hyperkeratosis, epidermal mononuclear infiltration or dermal edema were observed at baseline. Left panel is the low magnification (scale bar = 2 mm) and right panel is the high magnification (scale bars = 100µm)



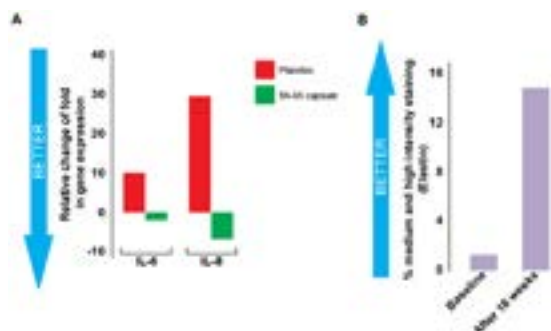
**Figure 4: H&E staining of punch biopsy after sixteen weeks'**

intake of TA-65® capsules. No acanthosis, spongiosis, chronic inflammation, hyperkeratosis, epidermal mononuclear infiltration or dermal edema were observed with 16 weeks' use of TA-65® capsules. Right panel is the low magnification (scale bar = 2mm) and left panel is the high magnification (scale bars = 100µm).

## Immunohistochemistry and RT-PCR:

Expression of pro-inflammatory molecules IL-6 and IL-8 increased in the skin of the subjects who took placebo capsules, whereas oral intake of TA-65® capsules reduced them within sixteen weeks (Figure 5A). A twofold reduction in the IL-6 and 6 fold reduction in the IL-8 were observed after sixteen weeks' intake of TA-65® capsules; in the placebo group, there is a tenfold increase in IL-6 and thirtyfold increase in IL-8. No improvement in the molecular markers TNF-α, MMP-1 and MMP-12 were observed following the oral intake of TA-65® capsules.

Elastin is a structural protein found in the dermis that provides flexibility to the skin and allows for it to elastically return to its native resting architecture. Figure 5B shows that the oral intake of TA-65® capsules increases the level of elastin by 93% after sixteen weeks. No improvement in the molecular markers collagen and filaggrin were observed following the oral intake of TA-65® capsules.



**Figure 5: Expression of inflammatory cytokines declines after 16 weeks' use of TA-65®.** IL-6 and IL-8 declines by twofold and sevenfold after sixteen weeks' oral intake of TA-65® capsule. (A). Elastin level increases by 93% after sixteen weeks' oral intake of TA-65® capsules (B).

## References:

- Goldsberry, A., C. W. Hanke and K. E. Hanke (2014). "VISIA system: a possible tool in the cosmetic practice." *J Drugs Dermatol* 13(11): 1312-1314.
- Harley, C. B., W. Liu, M. Blasco, E. Vera, W. H. Andrews, L. A. Briggs and J. M. Raffaele (2011). "A natural product telomerase activator as part of a health maintenance program." *Rejuvenation Res* 14(1): 45-56.
- Lasry, A. and Y. Ben-Neriah (2015). "Senescence-associated inflammatory responses: aging and cancer perspectives." *Trends Immunol.*

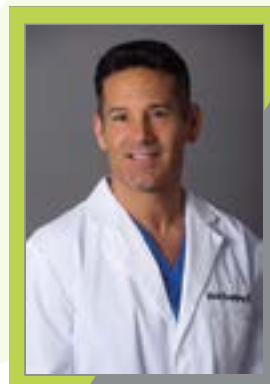
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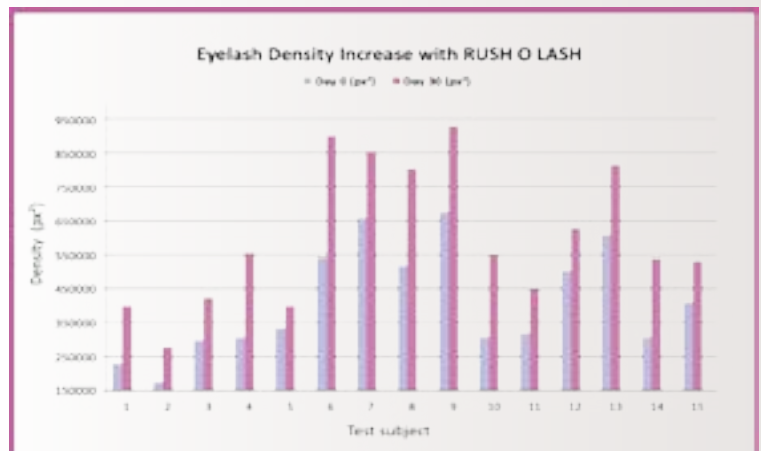
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|-------------------------------------|--------------------------|---------------------------|---------------|
| 1001                                | 225667                   | 397244                    | 76.03%        |
| 1002                                | 170845                   | 275846                    | 61.46%        |
| 1003                                | 296779                   | 422064                    | 42.21%        |
| 1004                                | 305472                   | 555983                    | 82.01%        |
| 1005                                | 332654                   | 398298                    | 19.73%        |
| 1006                                | 539452                   | 901300                    | 67.08%        |
| 1007                                | 660950                   | 854664                    | 29.31%        |
| 1008                                | 517962                   | 803979                    | 55.22%        |
| 1009                                | 675703                   | 928430                    | 37.40%        |
| 1010                                | 307875                   | 551420                    | 79.11%        |
| 1011                                | 318716                   | 449550                    | 41.05%        |
| 1012                                | 503245                   | 626310                    | 24.45%        |
| 1013                                | 608088                   | 816291                    | 34.24%        |
| 1014                                | 304936                   | 539486                    | 76.92%        |
| 1015                                | 409280                   | 531726                    | 29.92%        |
| Average Density (px <sup>2</sup> ): | 327700.4                 | 507304.8                  | <b>54.81%</b> |
| p                                   |                          | 0.000*                    |               |
| t                                   |                          | 9.193*                    |               |
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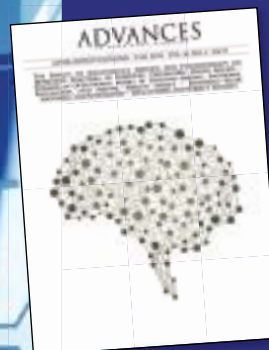
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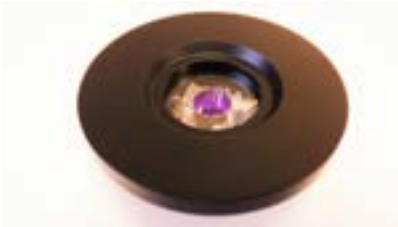
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**Presented by: Chris Meletis, ND**

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Dr. Meletis served as Dean of Naturopathic Medicine and Chief Medical Officer for 7 years, is a master formulator and was awarded the 2003 physician of the year by the American association of Naturopathic Physicians.

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ORIGINAL RESEARCH

# Novel Testing Enhances Irritable Bowel Syndrome Medical Management: *The IMMINENT Study*

Kelly Parsons, PhD; Julius Goepp, MD; Bryan Dechairo, PhD; Elizabeth Fowler, PhD; Nathan Markward, PhD; Patrick Hanaway, MD; Teresa McBride, ND; Darryl Landis, MD

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## Abstract

**PRIMARY STUDY OBJECTIVE:** To evaluate the economic utility of a fecal biomarker panel structured to suggest alternative, treatable diagnoses in patients with symptoms of irritable bowel syndrome (IBS) by quantifying, comparing, and contrasting health service costs between tested and non-tested patients.

**STUDY DESIGN:** Retrospective, matched cohort study comparing direct medical costs for IBS patients undergoing fecal biomarker testing with those of matched control subjects.

**METHODS:** We examined de-identified medical and pharmacy claims of a large American pharmacy benefit manager to identify plan members who underwent panel testing, were eligible for covered benefits for at least 180 days prior to the test date, and had data available for 30, 90, and 365 days after that date. We used propensity score matching to develop population-based control cohorts for each tested cohort, comprised of records

with IBS- related diagnoses but for which panel testing was not performed. Primary outcome measures were diagnostic and medical services costs as determined from claims data.

**RESULTS:** Two hundred nine records from tested subjects met inclusion criteria. The only significant baseline differences between groups were laboratory costs, which were significantly higher in each tested cohort. At each follow-up time point, total medical and gastrointestinal procedural costs were significantly higher in non-tested cohorts. Within tested cohorts, costs declined significantly from baseline, while costs rose significantly in non-tested control cohorts; these differences were also significant between groups at each time point.

**CONCLUSIONS:** Structured fecal biomarker panel testing was associated with significantly lower medical and gastrointestinal procedural costs in this study of patients with IBS symptoms.

Irritable bowel syndrome (IBS), a functional gastrointestinal (GI) disorder with unknown and probably multiple causes, is highly prevalent and costly. Ten to 20% of Americans suffer from IBS, with those in their prime years of productivity and employment being disproportionately affected.<sup>1-3</sup> The cumulative financial impact of IBS is greater than that of many other chronic illnesses, including asthma and migraine, and comparable to that of hypertension and congestive heart failure.<sup>4</sup>

The annual cost of IBS in the United States is estimated to be more than US \$20 billion.<sup>5</sup> In 2005, a study of one Fortune 100 company revealed that IBS direct costs to the employer were 1.5 times higher in affected employees (\$6364) than those accrued by a matched sample of controls (\$4245).<sup>4,6</sup> This resulted in an estimated \$1.9 million in costs borne by that employer alone. Furthermore, 43% more claims per beneficiary are filed with health payers on behalf of IBS patients, a positive difference that climbs to 180% for prescription claims.<sup>4,7</sup>

The bulk of the direct cost burden of IBS is related to excessive prescription of diagnostic procedures that (1) are administered in an unstructured, serial fashion over the course of many months or years and (2) arise from the concerns of clinicians and patients who wish to rule out every credible competing diagnosis.<sup>8,9</sup> IBS patients undergo significantly more diagnostic testing than matched controls, with odds ratios for common and expensive studies such as endoscopy and radiological imaging tests ranging from 2.5 to 5.7.<sup>7</sup> As many as 50% of patients being evaluated for IBS will undergo colonoscopy<sup>10</sup>; 25% of all colonoscopies performed in the United States are for evaluation of IBS symptoms.<sup>10,11</sup>

Despite such aggressive testing, the overwhelming majority of these procedures show normal findings in patients being assessed for IBS. Among the group of diagnoses that are typically being considered during a clinical evaluation, only maldigestion of lactose occurs at a frequency greater than 5%. Additionally, organic pathologic conditions, such as colorectal cancer and inflammatory bowel disease (IBD), occur at levels of less than 1%, and at equal or lower frequencies than they do in the general population.<sup>12,13</sup> Even in patients with "alarm features," for whom more invasive testing is currently recommended, organic disease was identified in only 3% of patients with suspected IBS in a study of 575 subjects; 1% had gastrointestinal cancer, 1.2% had IBD, and 0.7% had malabsorption.<sup>14</sup>

By contrast, a growing body of evidence suggests that, rather than being a single diagnostic entity, IBS instead represents an "umbrella" diagnosis comprised of different, often treatable conditions.<sup>15</sup> Habba et al demonstrated that 98% of patients had a final diagnosis that differed from IBS, and 68% of studied patients had treatable bile acid abnormalities or related conditions.<sup>15</sup> Furthermore, 98% of the latter group showed a favorable response to therapy, a figure vastly higher than that generally accepted for symptomatic response in IBS in general.<sup>15</sup>

Others have shown a meaningful prevalence of exocrine pancreatic insufficiency (6.1%) in subjects who fulfilled concurrent Rome criteria for IBS, using fecal pancreatic elastase levels as a diagnostic tool.<sup>16-19</sup> Similarly, fecal calprotectin levels have been demonstrated to effectively differentiate IBS from IBD,<sup>20-23</sup> and, when used as an alternative, noninvasive diagnostic testing may reduce the demand for colonoscopies and associated costs by as much as 50%, with the attendant realization of substantial cost savings.<sup>24</sup>

A computer-simulated economic analysis undertaken by the National Health Service in the United Kingdom showed that the use of fecal calprotectin was less costly and more diagnostically discriminative than routine blood tests — erythrocyte sedimentation rate (ESR), C reactive protein (CRP), serological markers, other neutrophil product markers, labeled white cell tests, and M2-pyruvate kinase—that are currently employed to categorize the inflammatory profiles of IBD and IBS.<sup>25</sup> Use of calprotectin testing resulted in fewer unnecessary endoscopies and an increase in the number of patients who were correctly diagnosed.

Many other underlying and readily treatable causes of IBS symptoms exist. These include celiac disease/gluten sensitivity, intestinal parasites and protozoans, and intestinal dysbiosis.<sup>5,17-19,22,23,26-28</sup> Emerging evidence suggests that there may exist a colonic microbiome pattern unique to IBS patients<sup>29-31</sup>; the advent of 16S ribosomal DNA polymerase chain reaction amplification may allow rapid detection of such patterns within the gut microbiome.<sup>32-38</sup>

We recently completed a retrospective review of 2256 records from patients who underwent simultaneous, parallel testing for a group of fecal biomarkers relevant to disorders that may produce IBS symptomatology, with treatable diagnoses suggested in 82.8% of cases.<sup>39</sup>

The combination of awareness of the multifaceted nature of IBS and availability of low-cost fecal biomarker testing means that clinicians now have the ability to rapidly screen for, and in many cases identify specific, treatable diagnoses that produce the symptom constellation of IBS, while excluding dangerous conditions (such as IBD) with acceptable diagnostic accuracy.

The accurate evaluation of a broad array of GI functional biomarkers might also provide much-needed comfort to patients and clinicians alike and support implementation of symptom-based, psychosocially sensitive interventions with greater confidence. With concrete, objective laboratory information in hand that excludes significant inflammatory pathophysiology and guides a targeted treatment regimen leading to quicker improvement in patient symptoms, clinicians might be expected to order fewer expensive, invasive tests in attempts to rule out potentially significant alternative disease states. As a result, payers might in turn realize substantial cost savings.

We hypothesized that a structured, parallel, fecal biomarker panel would reduce total and GI-related diagnostic testing costs compared to the routine approach to diagnosis and managing IBS.

To test this hypothesis, we designed a retrospective cohort study to compare healthcare utilization and costs in patients whose clinicians made use of one such fecal biomarker panel (Genova Diagnostics, Asheville, North Carolina, <http://www.gdx.net>; detailed in Table 1), and matched controls, who received standard evaluation for IBS. The study, part of a series of investigations into the use of fecal biomarker testing in IBS, was named IMMINENT (Improved Medical Management of IBS Needs Enhancement by Novel Testing) in recognition of the needs of clinicians to find better ways to understand the biology of their patients who present with symptoms consistent with IBS.

Performance characteristics of these biomarkers for diagnoses that may present as IBS have been published elsewhere for pancreatic elastase,<sup>54-57</sup> calprotectin,<sup>58-60</sup> eosinophil protein X,<sup>61</sup>

Clostridium difficile,<sup>62,63</sup> parasitology exam,<sup>64</sup> 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.

## Methods

### OBJECTIVES:

The objective of the project was to evaluate the utility of the fecal biomarker panel in a clinical setting by quantifying, comparing, and contrasting health service and pharmacy costs incurred by panel-tested and –non-tested IBS patients.

### DESIGN

We chose a retrospective, matched cohort design to compare the direct medical costs incurred by IBS patients tested with the fecal biomarker panel with those of matched control subjects.

### SETTING

We examined the medical and pharmacy claims of a large American managed pharmacy benefit manager patient database (Medco Health Solutions, now part of Express Scripts, St Louis, Missouri).

### ETHICS CONSIDERATIONS

Because this study used only de-identified records of claims data, no protected health information could be linked to individual patients. Consent for use of medical and pharmacy claims data for research purposes was obtained by participating insurance carriers. For these reasons, institutional review board approval was not deemed necessary.

### PATIENT POPULATION

#### Case Cohorts

Medical and pharmacy claims of plan members were searched to identify a cohort of patients who had been tested with the fecal biomarker panel by Genova Diagnostics, and who had one or more IBS-related diagnoses (Table 2). Because of major administrative changes at the participating institutions, actual percentage breakdowns for each ICD-9 code are not available. In a related study of a similar population,<sup>39</sup> ICD-9 codes 789 (abdominal pain), 564.1 (IBS), and 797.1 (diarrhea) accounted for more than three-quarters of all records.

Records were eligible for inclusion in the study (1) if the patient had been continuously eligible to receive benefits for at least 180 days preceding and 30, 90, or 365 days following the fecal biomarker panel test date and (2) if each member's sponsoring client had approved the use of medical and pharmacy claims for research purposes. For this study, all data were de-identified prior to analysis, and no protected health information was recorded.

This selection process resulted in identification of three longitudinally nested cohorts (Table 3). The M30 cohort (209 patients) consisted of patients with records available at 30 days after the fecal biomarker panel test date; the M90 (203 patients) consisted of members of the M30 cohort for whom data were available at 90 days after the test date; and the M365 (132 patients) consisted of M90 patients for whom data were available at 365 days after the test date.

#### Control Cohorts

A population-based control cohort of patients with IBS-related diagnoses (Table 2) was created for each tested cohort. Each control cohort was created from a randomly selected pre-match pool of non-tested members who submitted a claim for one of the IBS-related diagnoses during the 30 days before or after each tested subject's test date. Similar inclusion criteria were then applied. To read the rest of this article please visit <http://ow.ly/ScrGN>

Table 1 Selected Components of the Fecal Biomarker Panel

| Selected Biomarkers                                   | Description   |
|---|---|
| Pancreatic elastase                                   | Pancreatic elastase-1 (PE1) is a proteolytic enzyme secreted by the exocrine cells of the pancreas. Fecal PE1 testing provides a convenient, noninvasive, and reliable method of evaluating exocrine pancreatic function, well before steatorrhea occurs. <sup>18,19,40,41</sup>  |
| Calprotectin  | Calprotectin is a 36 kDa protein highly expressed in neutrophils, where it comprises up to 60% of the cytosol content. As a surrogate marker for intestinal neutrophil activity, fecal calprotectin levels >50 microg/g are considered a reliable indicator of neutrophil-mediated inflammation in the intestinal mucosa. <sup>42,43</sup>                      |
| Eosinophil protein X (EPX)                            | EPX is a cationic protein found in eosinophils. Upon degranulation, these proteins are released, mediating the eosinophilic immune response. <sup>44-46</sup>   |
| Clostridium difficile                                 | Once thought to be associated nearly exclusively with exposure to antibiotics, bowel infection with Clostridium difficile (C diff) is now recognized as being increasingly common in those without known antibiotic exposure (as many as 45.7% of people with culture-proven C diff infection had no antibiotic exposure in the past 90 days). <sup>47,48</sup> |
| Parasitology exam (microscopy and enzyme immunoassay) | A variety of protozoan parasitic infestations can produce symptoms of chronic diarrhea, bloating, and abdominal pain that can overlap with those of IBS; all of these organisms are also capable of causing post-infectious IBS. <sup>49,50</sup>   |
| Gut microbiota  | Beneficial flora controls potentially pathogenic organisms, influences nutrient production, removes toxins from the gut and stimulates the intestinal immune system (GALT). <sup>28,51-53</sup>   |

Table 2 Diagnostic Codes for IBS-related Diagnoses

| ICD-9 Code | Diagnosis   |
|------------|---|
| 564.0      | Constipation, unspecified                                       |
| 564.01     | Slow-transit constipation                                       |
| 564.1      | Irritable Bowel Syndrome  |
| 564.9      | Functional intestinal disorder, unspecified                     |
| 579.9      | Unspecified intestinal malabsorption                            |
| 787.91     | Diarrhea  |
| 789        | Abdominal pain  |
| 789.06     | Abdominal pain, epigastric                                      |
| 789.07     | Abdominal pain, generalized                                     |
| 536.8      | Dyspepsia and other specified disorders of function of stomach  |
| 536.9      | Unspecified functional disorder of stomach                      |
| 558.9      | Other and unspecified noninfectious gastroenteritis and colitis |
| 787.3      | Flatulence, eructation, and gas pain                            |



**Table 3** Summary of Selection Process

|  |        |
|--|--------|
| Records with fecal biomarker panel                     | 37 945 |
| Records matched to Express Scripts database            | 6892   |
| Records eligible for studya                            | 1656   |
| Records including pharmacy data                        | 1112   |
| Records with data for 30 days after index date (M30)   | 209    |
| Records with data for 90 days after index date (M90)   | 203    |
| Records with data for 365 days after index date (M365) | 132    |

**Author Affiliations** Express Scripts, St Louis, Missouri (Drs Parsons, Dechairo, and Markward); Lupine Creative Consulting (Dr Goepp); Genova Diagnostics, Asheville, North Carolina (Drs Fowler, McBride, and Landis); Institute for Functional Medicine, Gig Harbor, Washington (Dr Hanaway).

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# MitoQ – Research Review of 5 New Studies

MitoQ is a novel mitochondria-targeted antioxidant. It is well established to support mitochondrial function, reduce cellular oxidative stress and is proving to have significant potential as an anti-aging compound. Over the past decade MitoQ has been extensively researched, with over 200 papers published on its effect. Here we look at some of the more interesting papers from the last year and comment on the results.

## The mitochondrial-targeted antioxidant, MitoQ, increases liver mitochondrial cardiolipin content in obesogenic diet-fed rats.

**Authors:** Fouret et al.

**Abstract:** Cardiolipin (CL), a unique mitochondrial phospholipid, plays a key role in several processes of mitochondrial bioenergetics as well as in mitochondrial membrane stability and dynamics. The present study was designed to determine the effect of MitoQ, a mitochondrial-targeted antioxidant, on the content of liver mitochondrial membrane phospholipids, in particular CL, and its fatty acid composition in obesogenic diet-fed rats. To do this, twenty-four 6week old male Sprague Dawley rats were randomized into three groups of 8 animals and fed for 8weeks with either a control diet, a high fat diet (HF), or a HF diet with MitoQ (HF+MitoQ). Phospholipid classes and fatty acid composition were assayed by chromatographic methods in liver and liver mitochondria. Mitochondrial bioenergetic function was also evaluated. While MitoQ had no or slight effects on total liver fatty acid composition and phospholipid classes and their fatty acid composition, it had major effects on liver mitochondrial phospholipids and mitochondrial function. Indeed, MitoQ both increased CL synthase gene expression and CL content of liver mitochondria and increased 18:2n-6 (linoleic acid) content of mitochondrial phospholipids by comparison to the HF diet. Moreover, mitochondrial CL content was positively correlated to mitochondrial membrane fluidity, membrane potential and respiration, as well as to ATP synthase activity, while it was negatively correlated to mitochondrial ROS production. These findings suggest that MitoQ may decrease pathogenic alterations to CL content and profiles, thereby preserving mitochondrial function and attenuating the development of some of the features of metabolic syndrome in obesogenic diet-fed rats.

*Ref: Biochim Biophys Acta. 2015 Oct;1847(10):1025-35.*

**Comment:** CL is an important component of the inner mitochondrial membrane, constituting 20% of total membrane composition. It is essential for the optimal function of numerous enzymes that are involved in mitochondrial energy metabolism. A decline in CL composition has been associated with a number of conditions including diabetes, AD, PD, NASH, CVD and CFS. Aside from the interesting results of this research showing MitoQ provides protection to the liver from a HF diet; this research shows MitoQ is CL protective, supporting mitochondrial membrane integrity and function.

## Mitochondrial anti-oxidant protects IEX-1 deficient mice from organ damage during endotoxemia.

**Authors:** Ramsey H Et al.

**Abstract:** Sepsis, a leading cause of mortality in intensive care units worldwide, is often a result of overactive and systemic inflammation following serious infections. We found that mice lacking immediate early responsive gene X-1 (IEX-1) were prone to lipopolysaccharide (LPS) -induced endotoxemia. A nonlethal dose of LPS provoked numerous aberrations in IEX-1 knockout (KO) mice including pancytopenia, increased serum aspartate aminotransferase (AST), and lung neutrophilia, concurrent with liver and kidney damage, followed by death. Given these results, in conjunction with a proven role for IEX-1 in the regulation of reactive oxygen species (ROS) homeostasis during stress, we pre-treated IEX-1 KO mice with Mitoquinone (MitoQ), a mitochondrion-based antioxidant prior to LPS injection. The treatment significantly reduced ROS formation in circulatory cells and protected against pancytopenia and multiple organ failure, drastically increasing the survival rate of IEX-1 KO mice challenged by this low dose of LPS. This study confirms significant contribution of mitochondrial ROS to the etiology of sepsis.

*Ref: J Int Immunopharmacol. 2014 Dec;23(2):658-63.*

**Comment:** Sepsis is a whole body inflammatory response to an infection. Severe sepsis and septic shock have mortality rates as high as 80%. This paper, along with others, confirms the significant contribution of mitochondrial ROS (mROS) to the etiology of sepsis and suggests, if we are able to lower mROS, that we may be able to improve survivability of this condition.

## A mitochondrial-targeted Coenzyme Q analog prevents weight gain and ameliorates hepatic dysfunction in high-fat-fed mice.

**Authors:** Fink Et al.

**Abstract:** We hypothesized that the mitochondrial-targeted antioxidant, mitoquinone (MitoQ), known to have mitochondrial uncoupling properties, might prevent the development of obesity and mitigate liver dysfunction by increasing energy expenditure, as opposed to reducing energy intake. We administered MitoQ or vehicle (ethanol) to obesity-prone C57BL/6 mice fed high-fat (HF) or normal-fat (NF) diets. MitoQ (500 µM) or vehicle (ethanol) was added to the drinking water for 28 weeks. MitoQ significantly reduced total body mass and fat mass in the HF-fed mice but had no effect on these parameters in NF mice. Food intake was reduced by MitoQ in the HF-fed but not in the NF-fed mice. Average daily water intake was reduced by MitoQ in both the NF- and HF-fed mice. Hypothalamic expression of neuropeptide Y, agouti-related peptide, and the long form of the leptin receptor were reduced in the HF but not in the NF mice. Hepatic total fat and triglyceride content did not differ between the MitoQ-treated and control HF-fed mice. However, MitoQ markedly reduced hepatic lipid hydroperoxides and reduced circulating alanine aminotransferase, a marker of liver function. MitoQ did not alter whole-body oxygen consumption or liver mitochondrial oxygen utilization, membrane potential, ATP production, or production of reactive oxygen species. In summary, MitoQ added to drinking water mitigated the development of obesity. Contrary to our hypothesis, the mechanism involved decreased energy intake likely mediated at the hypothalamic level. MitoQ also ameliorated HF-induced liver dysfunction by virtue of its antioxidant properties without altering liver fat or mitochondrial bioenergetics.

*Ref: Exp Ther. 2014 Dec;351(3):699-708.*

**Comment:** This interesting research suggests MitoQ can mitigate weight gain in a HF diet. Whilst the results achieved were expected, the mechanism was not and proposes that a centrally mediated effect is involved. MitoQ again showed liver protective properties in the presence of a HF diet and significantly reduced membrane lipid peroxidation supporting the earlier study showing cardiolipin protection.



# The mitochondrial-targeted antioxidant MitoQ ameliorates metabolic syndrome features in obesogenic diet-fed rats better than Apocynin or Allopurinol.

**Authors:** Feillet-Coudray Et al.

**Abstract:** The prevalence of metabolic syndrome (MetS) components including obesity, dyslipidemia, insulin resistance (IR), and hepatic steatosis is rapidly increasing in wealthy societies. It is accepted that inflammation/oxidative stress are involved in the initiation/evolution of the MetS features. The present work was designed to evaluate the effects of three major cellular ROS production systems on obesity, glucose tolerance, and hepatic steatosis development and on oxidative stress onset. To do so, 40 young male Sprague-Dawley rats were divided into 5 groups: 1-control group, 2-high fat (HF) group (60% energy from fat), 3-HF+ MitoQ (mitochondrial ROS scavenger), 4-HF+ Apocynin (NADPH oxidase inhibitor), 5-HF+ Allopurinol (xanthine oxidase inhibitor). After 8 weeks of these treatments, surrogate MetS, mitochondrial function, and oxidative stress markers were measured in blood and liver. As expected, rats that were fed the HF diet exhibited increased body weight, glucose intolerance, overt hepatic steatosis, and increased hepatic oxidative stress. The impacts of the studied ROS inhibitors on these aspects of the MetS were markedly different. MitoQ showed the most clinically relevant effects, attenuating body weight gain and glucose intolerance provoked by the HF diet. Both Apocynin and Allopurinol showed limited effects suggesting secondary roles of xanthine oxidase (XO) or NADPH oxidase-dependent ROS production in the onset of oxidative stress-dependent obesity, glucose intolerance, and hepatic steatosis process. Thus, MitoQ revealed the central role of mitochondrial oxidative stress in the development of MetS and suggested that mitochondria-targeted antioxidants may be worth considering as potentially helpful therapies for MetS features.

*Ref: Free Radic Res. 2014 Oct;48(10):1232-46.*

**Comment:** In this paper we see further evidence that mitochondrial ROS are central to the development of metabolic syndrome secondary to a HF diet. Interestingly we again see weight gain attenuating effects from MitoQ in the presence of a HF diet. An improvement in glucose tolerance suggests targeting mROS as a sensible strategy for those with pre-diabetes.

## FFA-ROS-P53-mediated mitochondrial apoptosis contributes to reduction of osteoblastogenesis and bone mass in type 2 diabetes mellitus.

**Authors:** Li Et Al

**Abstract:** This study evaluated the association between free fatty acid (FFA), ROS generation, mitochondrial dysfunction and bone mineral density (BMD) in type 2 diabetic patients and investigated the molecular mechanism. db/db and high fat (HF)-fed mice were treated by Etomoxir, an inhibitor of CPT1, MitoQ, and PFT- $\alpha$ , an inhibitor of P53. Bone metabolic factors were assessed and BMSCs were isolated and induced to osteogenic differentiation. FFA, lipid peroxidation and mtDNA copy number were correlated with BMD in T2DM patients. Etomoxir, MitoQ and PFT- $\alpha$  significantly inhibited the decrease of BMD and bone breaking strength in db/db and HF-fed mice and suppressed the reduction of BMSCs-differentiated osteoblasts. Etomoxir and MitoQ, but not PFT- $\alpha$ , inhibited the increase of mitochondrial ROS generation in db/db and HF-fed mice and osteoblasts. In addition, Etomoxir, MitoQ and PFT- $\alpha$  significantly inhibited mitochondrial dysfunction in osteoblasts. Moreover, mitochondrial apoptosis was activated in osteoblasts derived from db/db and HF-fed mice, which was inhibited by Etomoxir, MitoQ and PFT- $\alpha$ . Furthermore, mitochondrial accumulation of P53 recruited Bax and initiated molecular events of apoptotic events. These results demonstrated that fatty acid oxidation resulted in ROS generation, activating P53/Bax-mediated mitochondrial apoptosis, leading to reduction of osteogenic differentiation and bone loss in T2DM.

*Ref: Sci Rep. 2015 Jul 31;5:12724*

**Comment:** Mitochondrial dysfunction and increased ROS are closely tied to metabolic syndrome and diabetes. Here we see a link between mitochondrial dysfunction and bone loss in T2DM. An inhibitor of CPT1, an inhibitor of mitochondria ROS production and an inhibitor of P53 were evaluated to understand the process driving bone loss. MitoQ, by inhibiting mitochondrial ROS, was able to reduce bone loss in this model of T2DM.

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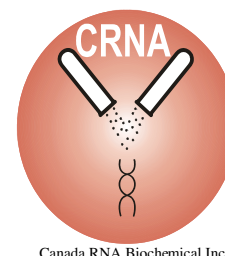
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Since 2008, our company has been dedicated to regenerative cellular therapy, our range of therapies include: extracting stem cells from fat, bone marrow and umbilical cord for tissue regeneration. We also offer ozone therapy and growth factor platelets application for articular regeneration and tissues.



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### **CherylLeeMD Sensitive Skin Care/AVAMR • Booth #3074**

144 S. Main St., Ste. 300, Alpine, UT 84004 USA

Ph: 877-622-4626 • Fax: 800-208-7255

Email: BryceStevens@CherylLeeMD.com • Website: www.CherylLeeMD.com

CherylLeeMD Sensitive Skin Care offers an intelligent skin care approach with its TrueLipids products and provides global skin barrier optimization through the effective use of patent-pending technology. With an overall equilibrium approach to the skin barrier, TrueLipids products help normal skin maintain a youthful glow AND benefit countless skin problems. As the most useful and multitasking line of skin care products on the market, TrueLipids contains anti-aging formulations and answers for redness, dry skin, enlarged pores, excess oil, irritation and allergy. TrueLipids brings a whole new meaning to hypoallergenic, non-toxic skin care.



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### **Chi's Enterprise, Inc. • Booth #6039**

1435 N. Brasher St., Anaheim, CA 92807 USA

Ph: 714-777-1542 • Fax: 714-777-7186

Email: postmaster@chi-health.com • Website: www.chi-health.com

Since 1986, Chi's Enterprise has marketed science-based standardized herbal supplements, including MYOMIN, an aromatase reducer. Since 2010, in BHRT conference, Dr. Jonathan Wright always recommends Myomin for reducing estradiol and increasing testosterone in BHRT users. Other supplements include OXYPOWER for reducing C-Reactive Protein/inflammation faster and improving the heart, lungs and energy; VEIN LITE for reducing homocysteine and amyloid peptide for the heart and brain; and ANGIOSTOP for inhibiting angiogenesis and receptor tyrosine kinases, VEGFR, EGFR, PDGFR and FGFR.



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### **Chronic Illness Recovery • Booth #3047**

P.O. Box 10756, Fort Worth, TX 76114 USA

Ph: 888-846-2474 • Fax: 480-772-4701

Email: info@chronicillnessrecovery.org • Website: www.chronicillnessrecovery.org

Chronic Illness Recovery provides information about resolving symptoms of autoimmune and chronic inflammatory diseases with inflammation therapy. This novel treatment is based on the theory that non-resolving inflammation is caused by the immune system's futile attempts to eradicate an intracellular infection. Elevated 1,25(OH)2D (calcitriol) and low 25(OH)D (which is erroneously diagnosed as vitamin D deficiency) are diagnostic markers of this inflammatory process. CIR also provides a counseling service for patients who are on inflammation therapy.



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### **Clear Mind Center • Booth #5039**

21870 Hancock Ln., Nathrop, CO 81236 USA

Ph: 714-609-7140 • Fax: 949-726-0700

Email: admin@clearmindcenter.com • Website: www.clearmindcenter.com

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### **Cleveland HeartLab • Booth #8035**

4738 Gateway Cir., Ste. A1110, Dayton, OH 45440 USA

Ph: 888-236-8019 • Fax: 937-434-2241

Email: info@criticalcareassessment.com • Website: www.criticalcareassessment.com

Cleveland HeartLab (CHL) offers innovative, scientifically proven, and medically relevant biomarkers that are predictive of cardiovascular risk. CHL's novel biomarker technologies are offered through our CAP-accredited and CLIA-certified next-generation clinical reference laboratory. CHL's mission is to aid clinicians in identifying risk and reducing cardiovascular events.





### Clinical Resolution Laboratory, Inc. • Booth #4070

1400 W. Lambert Rd., Ste. C, Brea, CA 92821 USA

Ph: 213-384-0500 • Fax: 213-365-0053

Email: [info@clinicalresolution.com](mailto:info@clinicalresolution.com) • Website: [www.clinicalresolution.com](http://www.clinicalresolution.com)

Clinical Resolution Laboratory is committed to anti-aging health and technology by developing instruments and techniques that produce exceptional results in the cosmetic and medical industries. Our product line includes a wide range of leading edge applications in the form of crèmes, serums, masks, and washes mainly for anti-aging and hair loss. We are committed to providing unsurpassed research and development for custom-formulated cosmeceutical, nutraceuticals and personal care products.

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### College Pharmacy • Booth #5038

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Ph: 719-262-0022 x178 • Fax: 719-262-0035

Email: [info@collegepharmacy.com](mailto:info@collegepharmacy.com) • Website: [www.collegepharmacy.com](http://www.collegepharmacy.com)

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### Cornerstone Advisors • Booth #6071

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Email: [info@buildbeyond.com](mailto:info@buildbeyond.com) • Website: [www.buildbeyond.com](http://www.buildbeyond.com)

Cornerstone Advisors is a boutique wealth management firm located in Bellevue, Washington. As wealth and life builders, we provide investment management, family office services, and targeted financial counsel to wealthy individuals, families and organizations. Our services are driven by a team of advisors who understand the complexities associated with wealth and the importance of building integrated solutions that deliver results.

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205 Webster St., Bethlehem, PA 18015 USA

Ph: 484-241-0588

Email: [brandon@cryoconcepts.com](mailto:brandon@cryoconcepts.com) • Website: [www.cryoclearrx.com](http://www.cryoclearrx.com)

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### Crystal Clear Digital Marketing • Booth #5046

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Ph: 888-611-8279 • Fax: 305-230-2350

Email: [sales@crystalcleardm.com](mailto:sales@crystalcleardm.com) • Website: [www.crystalcleardm.com](http://www.crystalcleardm.com)

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Email: service@nutrilab.com • Website: www.customvitepro.com

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### Derme & co • Booth #6021

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Ph: 800-847-8302 • Fax: 206-333-0608

Email: [orders@designsforhealth.com](mailto:orders@designsforhealth.com) • Website: [www.designsforhealth.com](http://www.designsforhealth.com)

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### Diagnos-Techs Inc. • Booth #5073

19110 66th Ave. S., Bldg G, Kent, WA 98032 USA

Ph: 800-878-3787 • Fax: 425-656-2871

Email: [diagnos@diagnostechs.com](mailto:diagnos@diagnostechs.com) • Website: [www.diagnostechs.com](http://www.diagnostechs.com)

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Douglas Laboratories is a globally recognized leader in the development and manufacturing of innovative, science-driven nutritional supplements. We offer personalized one-on-one consultation from credentialed experts to help you guide your clients to the right nutritional supplements for healthy aging and active lifestyles.<sup>†</sup> Our 60-year heritage of product efficacy, patient education and customer service makes us the right partner to get results for your clients and maximize revenue for your practice. <sup>††</sup>These statements have not been evaluated by the Food and Drug Administration. This product is not intended to diagnose, treat, cure, or prevent any disease.



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### Dr. Emma's Healthy Solutions, LLC • Booth #6045

340 Rte. 34, Ste. 202, Colts Neck, NJ 07722 USA

Ph: 732-475-7155

Email: [de@dremma.com](mailto:de@dremma.com) • Website: [www.DrEmma.com](http://www.DrEmma.com)

We are a physician-owned company that provides educational seminars and training materials for providers to help with the implementation of diet, health, and wellness counseling in office. We also supply patient materials such as brochures and diet guides, and nutritional supplements such as protein shakes and bars. Dr. Emma is well known for her research in HCG as a hormone adjunct, rather than the diet shot. She has also been on the media circuit for the past several years for her research expertise.



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### Drug Crafters • Booth #4085

5680 Frisco Square Blvd., #1100, Frisco, TX 75034 USA

Ph: 214.618.3511 • Fax: 214.618.3539

Email: [pharmacist@drugcrafters.com](mailto:pharmacist@drugcrafters.com) • Website: [www.drugcrafters.com](http://www.drugcrafters.com)

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### EndyMed Medical Inc • Booth #6046

790 Madison Ave., Ste. 402, New York, NY 10065 USA

Ph: 212-772-0077

Email: [info@endymedusa.com](mailto:info@endymedusa.com) • Website: [www.EndyMed.com](http://www.EndyMed.com)

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Email: docnetwork@foreverhealth.com • Website: www.ForeverHealth.com

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1723 N. Loop 1604 East, Ste. 202, San Antonio, TX 78232 USA

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Email: customerservice@genomind.com • Website: www.genomind.com

Genomind is a personalized medicine company bringing innovation to mental healthcare through genetic testing. Genomind is comprised of pioneering researchers and thought leaders in psychiatry and neurology and specializes in pharmacogenetic laboratory testing for psychiatry. Genomind is committed to partnering with clinicians to improve their patients' lives. Learn more at [www.genomind.com](http://www.genomind.com).

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The logo for Genomind, featuring the word "GENOMIND" in a bold, sans-serif font. The letter "O" is stylized with a green and blue circular graphic element.

### **Genova Diagnostics, Inc. • Booth #6013**

63 Zillioa St., Asheville, NC 28801 USA

Ph: 800-522-4762

Email: info@gdx.net • Website: www.gdx.net

Genova Diagnostics is an advanced clinical laboratory that applies a systems-based approach to the diagnosis, treatment and prevention of complex chronic disease. We utilize standard clinical laboratory biomarkers in a panelized manner designed to identify the root cause of disease in specific biological systems. We synthesize the results into easy-to-read and actionable results so you can execute a treatment protocol for your patients.

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The logo for Genova Diagnostics, featuring the word "GENOVA" in a large, serif font, with "DIAGNOSTICS" in a smaller, sans-serif font below it. To the right of the text are four colored dots: orange, red, green, and blue.

### **GenoVive • Booth #4028**

250 Plaque St., Harahan, LA 70123 USA

Ph: 504-734-3030 • Fax: 504-734-6354

Email: info@genovive.com • Website: www.genoviveusa.com

GenoVive uses each individuals' unique genetic profile to determine the appropriate balance of macronutrients, which work in harmony with their unique metabolism, to help them lose weight most effectively. GenoVive is the first comprehensive program that offers an all-natural meal and exercise program for effective and sustained weight loss. To learn more about the latest scientific developments in the field of Nutrigenomics, please visit us at booth #4028.

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The logo for GenoVive, featuring a stylized green leaf-like graphic to the left of the word "genoVive" in a lowercase, sans-serif font. Below the word is the tagline "weight loss designed by science" in a smaller font.

### **German - French Society for Thymus Therapy • Booth #6062**

48, Place du 14 Juin Grimaud, France

Ph: 33-4-86801388

Email: info@thymus-therapie.org • Website: www.thymus-therapie.org

Thymus Therapy, as part of Xenogene Cell Therapy, is a powerful therapy which has been used for many decades in beauty and medical treatments. Do you have more questions? Can Thymus Therapy help you? Please ask us!

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The logo for the German - French Society for Thymus Therapy, featuring a stylized graphic of a flame or leaf in red and blue to the left of the text "GERMAN - FRENCH SOCIETY FOR THYMUS THERAPY" in a sans-serif font.

### **Global Beauty Private Label Skincare • Booth #1024**

10015 Muirlands Blvd., Ste. C, Irvine, CA 92618 USA

Ph: 949-770-4290 • Fax: 949-951-1926

Email: alicia@globalbeauty.net • Website: www.globalbeauty.net

Global Beauty Private Label Skincare manufactures high performance, clinical strength skincare. Retinols, growth factors, acne gels and chemical peel products for all skin care needs. Effective products, low minimums, turnkey labeling, and quick turnaround times make us the #1 choice for private label skincare.

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The logo for Global Beauty Private Label Skincare, featuring the words "GLOBAL BEAUTY" in a large, serif font, with "private label skin care" in a smaller, lowercase, sans-serif font below it.

### **Global Life Rejuvenation • Booth #6038**

161 E. Main St., Denville, NJ 07834 USA

Ph: 973-627-7888 • Fax: 973-627-4158

Email: dglaab@globalliferejuvenation.com • Website: www.globalliferejuvenation.com

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The logo for Global Life Rejuvenation, featuring a stylized globe graphic to the left of the words "Global Life" in a serif font, with "REJUVENATION" in a bold, sans-serif font below it.





### Go Tattless Franchising LLC • Booth #4043

3370 N. Haden Rd., Ste. 123-250, Scottsdale, AZ 85251 USA

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Ph: 480-585-1744

Email: greif@hairsciencellc.com • Website: www.formula82m.com

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### Hallandale Pharmacy • Booth #4055

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Email: chanau@hanauhealthgroup.com • Website: www.hanauhealthgroup.com

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### Hansderma • Booth #5071

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### Harvest Tech • Booth #5065

10811 W. Collins Ave, Lakewood, CO, 80123 USA

Ph: 303-231-4691

Email: biologics@terumobct.com • Website: www.harvesttech.com

Harvest Technologies is the leading cellular therapy global manufacturer that develops point-of-care products to process and concentrate multiple biologics. These include high-density platelet rich plasma (APC+®), marrow aspirate concentrate (BMAC®) and adipose tissue, all concentrated using the SmartPrep3 Multicellular Processing System.

### **Hawaiian Moon • Booth #4060, 7071**

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### **Healeon Medical, Inc. • Booth #2061**

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Ph: 407-331-6292

Email: info@hnbc.us • Website: www.HnBconnection.com

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26 S. Baltic Pl., Meridian, ID 83642 USA

Ph: 208-995-2822 • Fax: 208-884-2067

Email: cassie.r@healthyhabitsmc.com • Website: www.healthyhabitsmc.com

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Email: info@healthycell.com • Website: www.healthycell.com

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Ph: 855-387-6466

Email: order@hevertusa.com • Website: www.hevertusa.com

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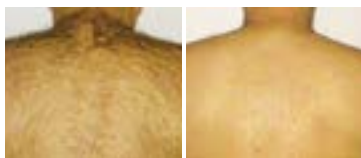
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The company specializes in design, development, manufacturing and distribution of non-invasive new technology-based medical devices. ICON Technology Development owns several pending patents and has several awards. Since 2014, we are developing and manufacturing our own new line of products under the brand name "Multiscan". The new products are based on Bio impedance spectroscopy, Galvanic Skin Response, Heart Rate Variability and Digital Pulse Wave Analysis.



### Ideal Protein of America • Booth #9015

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### **Immunosciences Lab, Inc • Booth #1037**

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### **InMode • Booth #4078**

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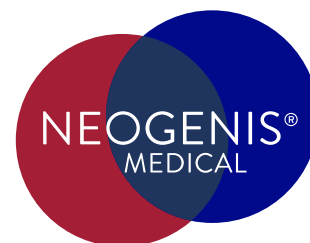
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Eric Braverman, MD author bestselling books Younger You, Younger (Thinner) You, The EDGE Effect and more. As President of the PATH Foundation he has co-authored numerous studies with commissioners of health, generals, the director of NIDA, etc. He is the director of PATH Medical and THN. Vitamins, information, and more available at booth.

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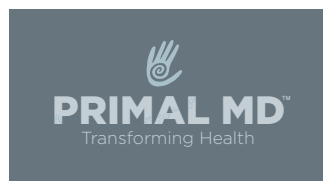
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Email: [front.desk@quicksilverscientific.com](mailto:front.desk@quicksilverscientific.com) • Website: [www.quicksilverscientific.com](http://www.quicksilverscientific.com)

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Email: [mgaza@quincybioscience.com](mailto:mgaza@quincybioscience.com) • Website: [www.prevagenpro.com](http://www.prevagenpro.com)

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Email: [kburton@sequoiadiagnostics.com](mailto:kburton@sequoiadiagnostics.com) • Website: [www.sequoiadiagnostics.com](http://www.sequoiadiagnostics.com)

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### **SottoPelle • Booth #6049**

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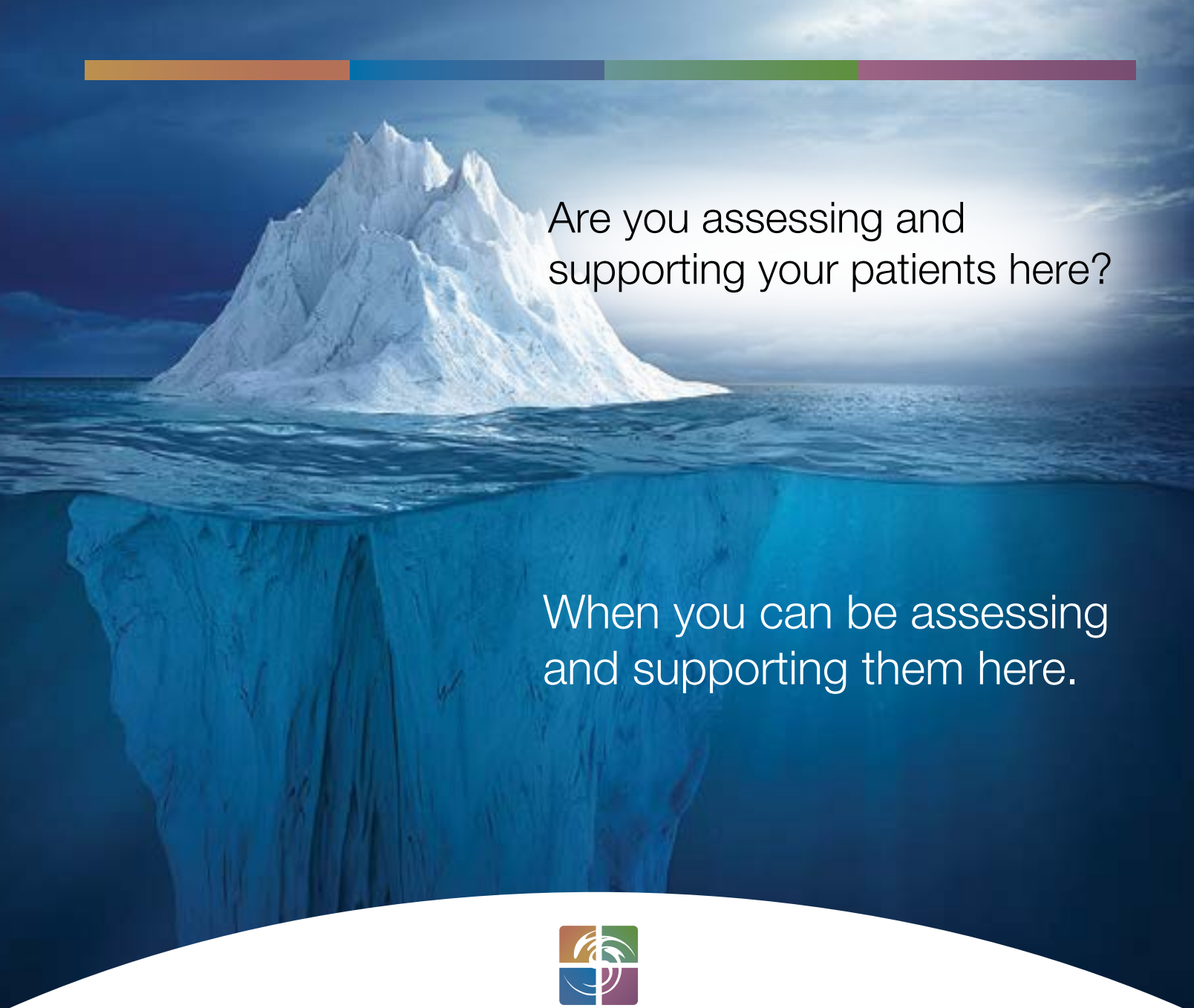
### **South Beach Symposium**

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Ph: 904-309-6262

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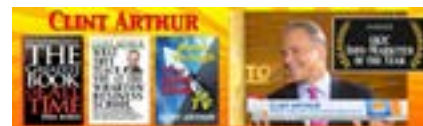
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### **Tango Advanced Nutrition, Inc. • Booth #7047**

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Ph: 866-778-2646 • Fax: 866-778-2646

Email: info@puretango.com • Website: www.puretango.com

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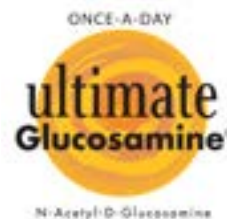
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