

THE GLOBAL RESOURCE FOR ANTI-AGING

May 2012
Issue



Anti-Aging

MEDICAL NEWS



SPECIAL GUEST SPEAKER
SUZANNE SOMERS

2012 SPRING
OFFICIAL SHOW
HANDBOOK

20th

ANNUAL WORLD
CONGRESS ON
ANTI-AGING AND
REGENERATIVE
MEDICINE

I HAVE CANCER
CAN I RESTORE MY HORMONES?

**ADDRESSING
GUT HEALTH**
AS A VITAL COMPONENT
OF COMPREHENSIVE
ANTI-AGING PROGRAM

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20th

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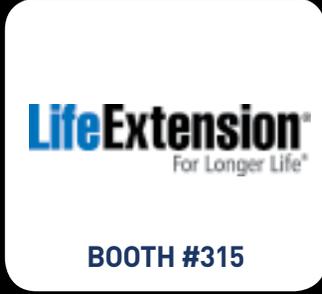
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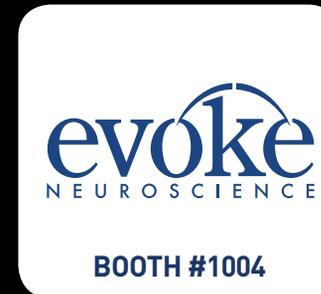
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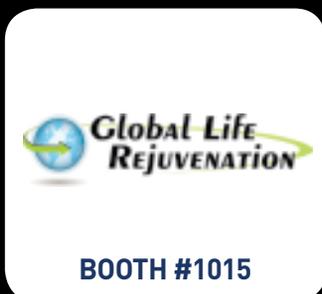
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Distinguished Colleagues:

The American Academy of Anti-Aging Medicine (A4M) welcomes you to Orlando for the Spring 2012 Session of the 20th Annual World Congress on Anti-Aging Medicine & Regenerative Biomedical Technologies.

As the American Academy of Anti-Aging Medicine (A4M) enters its second decade of educational service, we invite you to celebrate the pioneering achievements of the Anti-Aging scientific movement.

Historically, the physicians and scientists involved in anti-aging science have been the first to embrace innovative medical discoveries and technological advancements that have subsequently been mainstreamed. At the A4M World Congresses held continuously since 1993, over 100,000 physicians, health practitioners, and scientists have been among the first to learn of revolutionary life-enhancing, life-extending discoveries such as:

- The hormone melatonin as a cancer-fighting agent
- The importance of growth hormone in adulthood
- The identification of homocysteine as a marker for heart disease
- The role of Vitamin D beyond bone health
- The reversal of skin aging with non-/minimally-invasive techniques
- Advanced biomedical technologies including nanotechnology, DNA on a chip, and brain resuscitation
- Regenerative medical therapeutics such as stem cell therapies

To usher in our twentieth year and beyond, the A4M reaffirms its commitment to education. The A4M encourages physicians and health practitioners to pursue Certifications and Fellowships in the Anti-Aging Medical Specialty. The American Board of Anti-Aging & Regenerative Medicine and Fellowship certifications are recognized by consumers around the world who associate these credentials with quality anti-aging medical care. Visit www.a4m.com to learn more.

Tomorrow's medicine is here TODAY. We commend you for your foresight in attending in this world-renowned scientific Congress.

With your involvement, the anti-aging medical specialty continues to expand and become more widely accessible. We are confident that you will enjoy the spirit of educational exchange and dialogue at this conference and that you leave this event with an enhanced knowledge of the diverse array of interventions and therapeutics to promote the healthy, extended human lifespan.



Ronald Klatz

Ronald Klatz, M.D., D.O.
President, A4M



Robert Goldman

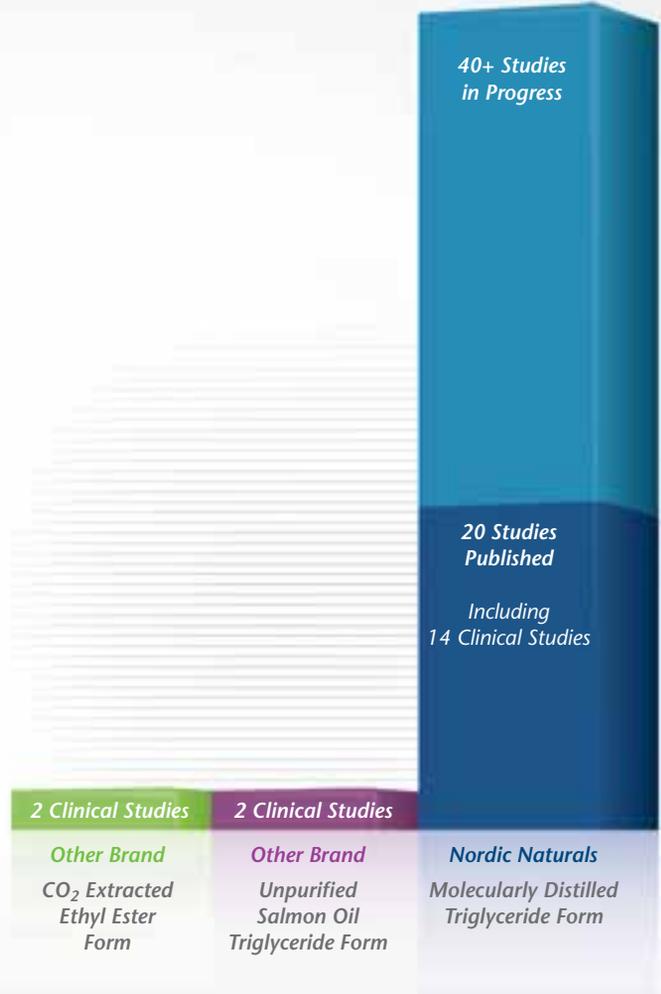
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FIRST LINE OF DEFENSE

EARN A CERTIFICATE IN PRIMARY CARE SKIN CANCER MEDICINE

The world's leading CME primary care skin cancer training program—available through the A4M—equips all kinds of physicians with the knowledge and skills to diagnose and treat skin cancer in their patients.

Do you know which country has the highest incidence of melanoma in the world? You might have guessed the United States, but the answer is Australia. On that sunny continent down under, leading research institutions have been pioneering new methods of diagnosing and treating skin cancer.

Those cutting-edge findings have been distilled into the world's leading CME primary care skin cancer training program, the Certificate in Primary Care Skin Cancer Medicine, created in collaboration with the University of Queensland, the country's largest medical school. More than 4,400 medical practitioners from all over the world have attended the overseas workshops—and the course is now available in the United States through the American Academy of Anti-Aging Medicine.

The program aims to optimize participants' knowledge in skin cancer medicine and empower them to meet existing and future marketplace challenges. From primary care physicians to nurse practitioners, participants will learn how to become the first line of defense against skin cancer for their patients.

The certificate course comprises both online and in-person training, led by expert specialists in dermatology, surgery, business and primary care workflow management, including Professor from University of Queensland, David Wilkinson, MD, PhD; Adam I. Riker, MD, FACS; Janice Johnston, MD; and Paul Elmslie, MBA. The course's interactive component makes it an especially unique offering. During the three-day workshop, delegates participate in presentations via wireless audience responders and work one on one with experts during hands-on procedural sessions.

MEET THE FACULTY



David
Wilkinson,
MD, PhD



Adam
I. Riker,
MD, FACS



Janice
Johnston,
MD



Paul
Elmslie,
MBA

After successfully completing the course, participants receive a Certificate in Primary Care Skin Cancer Medicine from the University of Queensland School of Medicine. The program can also earn prescribed credits by the American Academy of Family Physicians (see the A4M for details), as well as a credit toward a related unit in the Master of Medicine (Skin Cancer).

Here, we discuss the program with Dr. Johnston, owner/chief medical officer of Arrowhead Health Centers, a 150-employee, multi-specialty multi-location healthcare company that she founded in 1997. Johnston first took the class as a delegate a few years ago. After implementing its lessons into her own practice and reaping the benefits, she now teaches the course herself.

How do you describe the skin cancer course in your own words?

It's a really great introductory course to skin cancer training and treatment. Doctors can come away feeling confident in their skills in recognizing very common skin cancer lesions both with their naked eye and using dermoscopy, which is the handheld microscopy system that we teach as well.

What are some of the highlights?

The dermoscopy part of the course is quite exceptional. It's something that can help the doctor in diagnosing things you couldn't with your naked eye and instills a lot of confidence in knowing which lesion is something you should biopsy and which one is okay to leave alone. Most doctors have had little to no exposure to that, so it's an excellent tool to add to your practice.

school, but if you're not doing it on a regular basis, your skills can certainly not be up to where they need to be. Having someone who's skilled in that regard to help you with these skin procedures is just awesome, and you're well on your way by the time you leave the workshop.

What makes the program unique?

It's that combination approach where you're putting everything

Doctors can come away feeling confident in their skills in recognizing very common skin cancer lesions both with their naked eye and using dermoscopy.

The other thing is the hands-on experience that they get on the second day of the course, where they work with cutting the pork bellies. It's great to have a surgeon right there to kind of hold your hand and make sure that your skills are up to par so that, when you get back to the office, you feel confident to put what you learned into action.

Why is the interactive part of the workshop so important? We're all taught this kind of thing in medical

altogether in a short amount of time, and that you're getting the dermoscopy training and getting a hands-on experience with professionals who deal with this day in and day out. The other part that's really great is that there's a business application to it as well, so they review all the coding and documentation that you need to be doing so that, as soon as you go home, you can start it the next day.

Why was the course formatted to be partially online? What are the



benefits of that? The online part is reviewing hundreds of microscopic images of lesions. It helps with the doctor's pattern of recognition that they can review things over and over and really get confident about what they're looking at. There's only so many things that we can show during a weekend course, but at home, they can go over it until they do feel comfortable in their skills.

Who could benefit from this course work? This course is intended for more of a primary care audience, and that could be physicians or a mid-level provider: a nurse practitioner or a physician's assistant. A lot of gynecologists often function as primary care: They're seeing women coming in for their pap smears, and they'll see moles and things that are unusual. They need to be able know what they're looking at.

What is the ultimate goal of the skin cancer course? To bring awareness to skin cancer and how prevalent it is. One in five people will get skin cancer. Melanoma is a deadly disease. So if we can screen and educate our patients and make them aware that this is a problem that they need to be checked for, that's huge. A skin check is something that takes 10 to 15 minutes, but it could save your life.

How do you implement skin cancer training into your practice? I started with the course probably three years ago and, after the first course, started implementing its teachings immediately. We do biopsies and excisions and treatment. As the years have passed, my skills have gotten better and better, and the types of things that I'm handling now are much bigger in nature than I did at the beginning. So there's kind of something for everybody with this course, and you go with what your confidence is and what your knowledge is at the beginning, and

you start to feel more and more comfortable in your skills and what you can do.

Why do you think delegates have been so consistently pleased with the course, which has received a satisfaction rating of 91% or higher since its inception in 2006?

The hands-on surgical application is something they really enjoy because there's a lot of one-on-one supervision. And they really feel that they're getting something that's focused, and at the end of the weekend, they've gotten something out of it, and they feel well skilled to put it into practice the next day.

See your A4M Education Advisor for more details

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Doctors and scientists agree: An organic diet provides a dramatic and immediate protective effect against exposure to organophosphorus pesticides that are commonly used in agricultural production." (Source: Center for Disease Control)

The problem is that eating organic has been unaffordable for most.

Families want to follow the doctor's advice to adopt health lifestyle practices like eating organic fruits, vegetables, avoiding foods with pesticides, and using natural products to compliment a healthy-living diet; it's a good idea in theory but very hard and expensive to put into practice. That is, until now.

The Green PolkaDot Box™ (GPDB) is a new online buying membership that makes organic foods affordable to all families with FREE home delivery. GPDB offers a Costco-like solution for healthy, fresh and organic foods that has the potential to save healthy food consumers up to 60% off on their favorite organic foods and other healthy products.

"...it's something [doctors] have got to try and see for themselves before recommending it to their patients."

According to the Organic Trade Association, 87 percent of U.S. families — more than ever before — are starting to include some organic foods in their diets. But that has meant adding potentially thousands of dollars in organic food costs to their annual budget. No more. GPDB helps consumers slash their food budget for clean organic and natural foods.

Founder of the Green PolkaDot Box, Rod A. Smith, says the membership organization allows affordable access to "clean" non-GMO and healthy foods for everyone. "The time has finally arrived for consumers who eat healthy to break through the high price barrier that, until now, allowed only the wealthy to enjoy healthful organic and natural foods."



Smith believes that doctors are in the best position to inform their patients about this new shopping advantage. "We want health care practitioners to know they are acting in the best interest of their patients to recommend GPDB as the #1 source for clean organic foods at the lowest prices. But for doctors to truly appreciate the value we offer... it's something they've got to try and see, for themselves first, before they **can recommend it to their patients.**"

GPDB offers a growing number of over 1,500 products including products from Eden Organics, Annie's Home Grown, Bragg, Pangea Organics, Nutiva, Justin's, Happy Baby, Bob's Red Mill, Blue Diamond and hundreds of other top selling national brands; and a wide variety of fresh harvested organic produce.

Included in the list of GPDB fans are organic and non-GMO food organizations like *Natural News*, The Organic Consumers Association, *Health Living* magazine, Citizens for Health, and The Institute for Responsible Technology, and the American Academy of Anti-Aging Medicine (A4M). All are excited about what experts are calling one of the savviest solutions to come along in the health food industry.

The thousands of members of the Green PolkaDot Box say they enjoy saving money on healthy products, but they also appreciate the opportunity to shop according to their unique dietary lifestyle preferences: gluten-free, low glycemic (diabetes), low-sodium, dairy free, corn-free, soy-free and many others.

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20th Annual World Congress on Anti-Aging and Regenerative Medicine, Spring 2012 Session

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Aging and the Telomere Connection

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Abstract:

Telomeres are repetitive DNA sequences at the ends of linear chromosomes that serve as essential protective structures that maintain the integrity of chromosomal DNA. Each time a normal human cell divides some telomeric DNA sequences are lost. When telomeres are short, cells enter an irreversible growth arrest state called replicative senescence (or aging). There is mounting evidence that short telomeres correlate with age-associated diseases by limiting the ability of tissues to regenerate. This has led to the idea that telomere length could be a

good and highly reliable indicator (a biomarker) of biological (not necessarily chronological) aging. Telomere length measurements, especially measurements of the shortest telomeres, provide a molecular determinant about overall health. It has been shown that environmental stressors can lead to increases in oxidative damage and premature telomere shortening. Smoking, inflammatory disease, lack of modest levels of exercise, and excessive drinking can all contribute to increases in the rate of telomere shortening but in some instances these may be reversed by behavior modification. Just as cholesterol

and blood pressure measurements provide an indication of overall health, newly introduced highly quantitative telomere length assays also provide a window into one's overall health. While no one can predict how long any individual person will live, quantitative telomere tests are scientifically proven biological assays that correlate with the ability of human cells to proliferate and replenish tissues.

Background:

Aging is associated with the gradual decline in the performance of organ systems, resulting in the

loss of reserve capacity, leading to an increased chance of death ⁽¹⁾. In some organ systems, this loss of reserve capacity with increasing age can be attributed to the loss of functional cells ⁽²⁾. Chronic localized stress to specific cell types results in increased cell turnover, focal areas of replicative (aging) senescence ⁽³⁾ followed by predictable alterations in patterns of gene expression. This results in reduced tissue regeneration, culminating in many of the clinical pathologies that are largely associated with increased age.

Evidence that telomere shortening leads to replicative senescence:

Telomere length is a record of the history of and the potential for replication of human cells ⁽⁴⁾. Telomere dynamics in proliferating tissues might provide insight into not only the biology of aging but also the pathology of age-related diseases. A body of epidemiological and clinical data suggests that relatively short telomeres and accelerated telomere attrition are linked to factors that define aging and diseases of aging in humans ⁽⁵⁻¹²⁾.

There is also experimental support that most human proliferative tissues and organs including most somatic cells (even stem cells of renewal tissues) undergo progressive telomere shortening throughout life ⁽¹³⁾. While there have been many studies demonstrating correlations between telomere shortening and proliferative failure of human cells, the evidence that it is causal has now been directly demonstrated ⁽¹⁴⁾. Telomerase is a ribonucleoprotein enzyme that functions to lengthen telomere length during early fetal development and in some proliferative adult stem

cells. However, almost all adult tissues do not have detectable telomerase activity and telomere lengths decrease throughout life. Introduction of the telomerase catalytic protein component into normal human cells results in detection of telomerase activity ⁽¹⁴⁾. Normal human cells stably expressing transfected telomerase demonstrate telomere maintenance and extension of life span, providing direct evidence that telomere shortening controls cellular aging. The cells with introduced telomerase maintain a normal chromosome complement and continue to grow in a normal manner ⁽¹⁵⁾. These observations provide the first convincing evidence for the hypothesis that telomere length determines the proliferative capacity of human cells.

Evidence that telomere shortening is important in organ or tissue aging:

There is a relationship between replicative aging and skin pressure ulcers ⁽¹⁶⁾ that are believed to be caused initially by decreased circulation, leading to localized areas of necrosis. This is followed by attempts of the skin cells to regenerate. In the areas of pressure ulcers there is dramatic shortening of telomeres compared to adjacent normal areas. Other examples include patients with advanced and progressive human immunodeficiency virus infections who have specific T-cell deficiencies, patients with liver cirrhosis, patients with muscular dystrophy, and patients with bone-marrow exhaustion in myeloproliferative diseases. In spite of a diverse etiology, a common pathological mechanism is an increased turnover of stem-like cells leading to cellular senescence and then to a disease state. Thus,

in patients with progressive AIDs there is increased turnover of certain types of mature T-cells and at least initially the patient regenerates more T-cells. Proliferative failure due to telomere erosion may ultimately result in the patient having low T-cell counts, leading to opportunistic infections. In Duchenne muscular dystrophy, children are capable of walking for a few years but, because they have inherited a mutated dystrophin gene, their muscle fibers degenerate. To compensate, their muscle stem-like cells (satellite cells) regenerate new muscle fibers but these also degenerate and eventually the stem cells cannot keep dividing

...there is mounting evidence that in some aged-related disorders telomere decline in specific tissues and organs may contribute to aging and cancer vulnerability ⁽²²⁾.

and the muscle is replaced with fat (adipocytes). More recently alterations in key genes involved in maintaining telomeres have been demonstrated to be involved in human genetic diseases such as dyskeratosis congenita, sporadic bone marrow failure and idiopathic pulmonary fibrosis ⁽¹⁷⁻²¹⁾. In summary, there is mounting evidence that in some aged-related disorders telomere decline in specific tissues and organs may contribute to aging and cancer vulnerability ⁽²²⁾.

Gradual shortening of telomeres coincide with the long term aging process:

Under normal conditions most tissues can last a typical life span. However, with the improvement in sanitation, the development of antibiotics, vaccines, and modern pharmaceutical drugs, humans are living longer. A proliferative capacity for good maintenance and repair for 80-100 years would not have been selected for in evolutionary terms, when the average human lived at most 30-40 years. Today there is an increase in aged-related cellular decline in normal people who live to an exceptionally old age, while in the past problems from a limited cellular proliferative capacity was only observed in disease states⁽²³⁾. However in older individuals without diseases, there is an increased incidence of immunological deficiencies, chronic ulcers, wearing down of the vascular endothelium leading to arteriosclerosis, proliferative decline of retinal pigmented epithelial cells leading to age-related blindness and cancer. In order to track telomere lengths and potentially slow down or reverse the increased probability of telomere associated diseases, it is fair to ask if telomere tests would have utility as a clinical diagnostic assay.

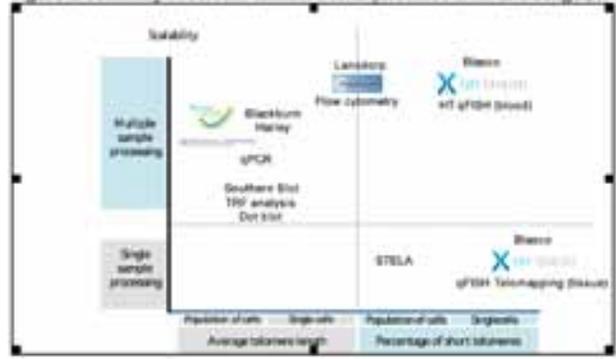
Are telomere tests ready for prime time?

We still know little about the dynamics of telomere length changes over multiple years in large human populations. It would be difficult for government funding agencies in today's financially challenged environment to support large scale longitudinal telomere research studies. Thus, the private sector has taken the lead and developed a series of telomere tests to determine if the multiple associations of

diseases with telomere length measurements hold up in placebo controlled studies. Since there are emerging classes of natural products (telomerase activators)⁽²⁴⁾ and genetic manipulations that may influence telomere biology and aging⁽²⁵⁻²⁶⁾, we need rigorous scientific telomere tests to prove the mechanism of actions. The ability of the private sector to start large scale longitudinal telomere measurements, including patients completing detailed questionnaires, will permit the scientific community

method provides information about the shortest telomeres in individual cells. However, as long as one can get sufficient DNA, data can be obtained. Another newly developed method is called STELA (single telomere length analysis). This is very low throughput but the advantage is that it can provide information on the shortest telomeres in a population of cells. It cannot easily be adapted to a commercial test at the present time due to turnaround is more than a week per assay, and this method does not provide information about the

Figure 1. Summary of current telomere tests (modified from Life Length, Inc.)



to assemble databases that will allow for determining statistically relevant sub-populations of patients that may benefit from telomere length modifications.

Telomere length measurement tests:

The basic laboratory research telomere test is known as TRF (terminal restriction fraction). This test is at present not suited for high throughput scale up, but provides a visual representation of the distribution of populations of cells using a Southern blot electrophoresis approach. Other laboratory assays being developed include a modification of the TRF assay using a dot blot approach which is similar but does not provide visualization of the range of telomere lengths (Figure 1). Neither the TRF nor dot blot

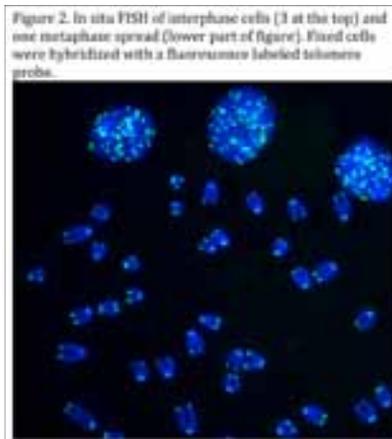
shortest telomeres in individual cells.

The qPCR method is not only used in some research laboratories but is also being used in some commercial situations (Telomehealth.com; Spectracell.com). The advantage of the qPCR method is that it is relatively fast and capable of high throughput. The disadvantage is that this test does not give information about individual cells so the results are generally averages of telomere lengths in populations of cells. The flow FISH method almost exclusively uses lymphocytes and is beginning to have some commercial development (Repeatdiagnostics.com; Figure 1). This method uses a FACS (fluorescence activated cell sorter) to analyze cells with different signals after hybridization with a fluorescence telomere probe. This

method provides the distribution of telomere lengths one cell at a time but only on the average of telomere lengths (not telomere lengths within individual cells). This method is CLIA certified for measuring telomeres as part of genetic counseling. The high throughput microscopic Q-FISH (Quantitative Fluorescence In Situ Hybridization) method is a highly reliable approach to quantitating telomere lengths and has the advantage over other methods of providing not only average telomere length per cell but also the number and distribution of the shortest telomeres in individual cells^(27, 28; Figure 2). This method permits visualization and quantitation in a microscope of individual cells so one can distinguish between a subset of cells containing very short telomeres and those that have very long telomere lengths (Figure 2). A commercial test (HT Q-FISH) has now been developed (Lifeflength.com). With the exception of HT Q-FISH, no other commercial laboratory method is available which can distinguish a single critically short telomere within one cell that may be triggering senescence. While initially a laboratory test with relatively low throughput, this approach has recently been commercialized into a high throughput method with an accuracy of 5% between tests (Lifeflength.com).

The last method is called Telomapping (Lifeflength.com) (US Patent N^o 8,084,203 B2). This is similar to Q-FISH but determines the telomere lengths on chromosomes from tissues (thus maintaining the topology of the samples). The advantage of this method is that archival formalin embedded paraffin sections can be used to determine if specific cells within a tissue have short telomeres. This is likely to

have important implications in the precancerous detection field. This method is slightly more time intensive and is not scaled up to high throughput analysis at the present time.



Bottom-line, telomere tests are ready for prime time and while there are several options, one needs to ask if the method delivers results that provide insights in critically short telomeres in individual cells which are universally regarded as the principal cause of replicative cell aging and age-related diseases (Dr. Carol Greider, Nobel Prize winner, 2009)⁽²⁹⁾.

Summary and future challenges:

Telomere biology is important in human aging and cancer. Cancer cells need a mechanism to maintain telomeres if they are going to divide indefinitely, and telomerase solves this problem. Thus, inhibition of telomerase may have utility in cancer therapeutics (30). Since almost all tissues show progressive shortening of telomeres with increased age, in some instances, organ failure may occur in chronic diseases of high cellular turnover. Therefore, telomere manipulations in cells of regenerative tissues may have utility in treating certain disorders in the aging population (24, 28). While

the aging process is complex and certainly cannot be explained solely on the basis of telomere biology, there is a growing consensus that in some situations telomere biology and telomere tests may have important utility similar to cholesterol assays or blood pressure monitoring measurements. This is still a young field and improvements will continue to occur. With longitudinal studies in individuals over many years trends will emerge that are likely to provide important insights into human disease. The challenge is to understand how telomere biology leads to increased aging vulnerability and to learn how to intervene in these processes.

Conflict of Interest:

Dr. Shay is a Scientific Consultant to Life Length, Inc. (www.lifeflength.com), Madrid Spain

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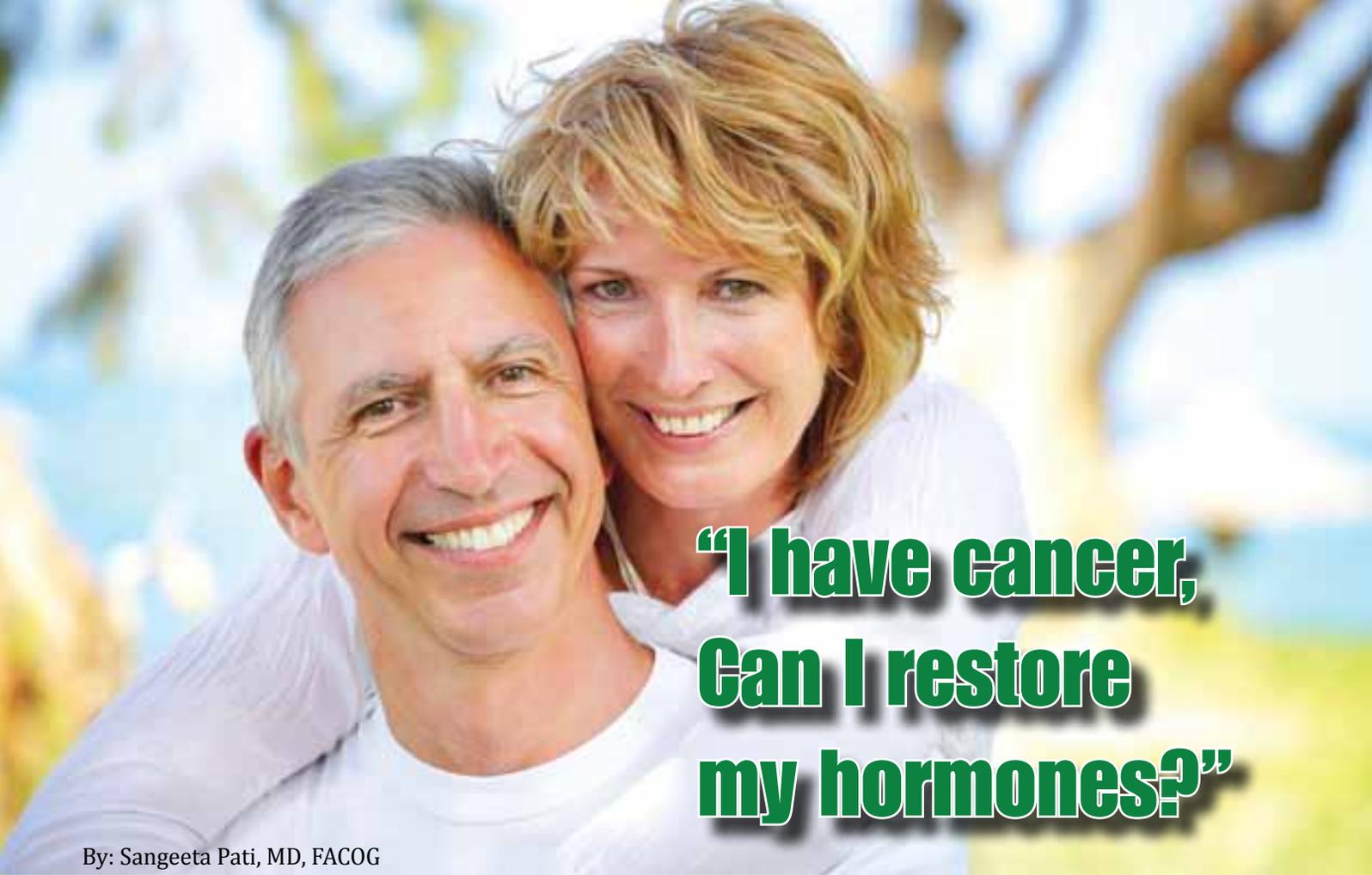


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“I have cancer, Can I restore my hormones?”

By: Sangeeta Pati, MD, FACOG

With increasing frequency I am faced with the patient who 1) has active cancer 2) is in remission, or 3) perceives a high risk for cancer due to family history and other risk factors. This patient is the one who is losing their marriage from a lack of a sex drive or the one who has severe osteoporosis and can not take standard therapies. This is the patient who goes from doctor to doctor on a quest for a better quality of life: better energy, better sleep, better sex drive and better mood. This is the one who believes that life does not end with cancer and is searching to get his/her life back. Everybody knows this patient.

When faced with this patient, the first question that we have to ask ourselves is what causes cancer ? Is it hormone restoration or hormone imbalance? Is it nutrient restoration or nutrient imbalance? Toxins?

Mental stress? Physical stress?
Family genetics?

A few facts to consider are:

- 1) Most women who get breast cancer are NOT on hormones, most men who get prostate cancer are NOT on testosterone. So, there are other more important contributors.
- 2) Genetics accounts for 15% of all breast cancers and less than 10% of all prostate cancers, the rest are accounted for imbalances in hormones and nutrients, the presence of toxins and stressors in the mind and body.
- 3) Cancer cells develop in the body on a regular basis. An optimally functioning immune system will detect these cells and destroy them.
- 4) With a compromised immune system, it takes 10-20 years for a cancer cell to grow to detection

limits. The good news is that we actually have an opportunity to detect imbalances that are creating immune deficiencies and correct them.

- 5) When we achieve better energy, mood and sleep we are correcting the immune system. For example, just by achieving 8 hours of solid sleep, we have enhanced the recuperation of the immune system.

In this article, I will share a comprehensive approach to this patient focused on the data supporting hormone restoration as a part of restoring both quality of life and an optimal immune system. I will also touch on the 5 components that must be addressed, since hormone restoration should never stand alone.



Introduction

The patient with cancer who wants hormones must understand the causes and corrections to address these imbalances by applying SaJune's 5-point model.

Hormone corrections:

Some hormone imbalances which contribute to cancer cell proliferation are :

Imbalance	Information and Data for the patient	Correction
Low Progesterone	<p>Low progesterone is associated with a 5.4 fold increase in breast cancer and a 10 fold increase in all cancers.¹</p> <p>Progesterone decreases the proliferation of cells in the breast and uterus.^{2,3}</p> <p>Progestin has an extra side chain which has been shown in several studies (including the WHI study) to increase the risk of cancers, strokes and heart attacks.^{4,5,6}</p> <p>Although the WHI study used Progestins, the largest study in the world (the French Cohort Study with over 80,000 women) used progesterone and found a slight reduction in breast cancer.⁹</p>	<p>Do NOT use progestins, such as medroxyprogesterone acetate.</p> <p>Measure and correct progesterone deficiencies with bio-identical progesterone.</p>
Low testosterone	<p>The higher the testosterone level, the lower the incidence of prostate cancer.^{7,8}</p> <p>Bioidentical testosterone reduces the proliferation of breast cells in vitro 5 fold.⁹</p>	<p>Correct testosterone levels to upper quartile of normal range with bio-identical testosterone.</p>
Low estriol	<p>Low estriol is associated with increased breast cancer.^{10,11,12,13}</p>	<p>Correct estriol in women with bio-identical estriol.</p>
High toxic estrone metabolites	<p>Estrone byproducts are carcinogenic.^{14,15}</p> <p>Premarin™ approximately 50% estrone.</p>	<p>Measure urine estrone byproducts to assure proper liver detoxification.</p>
Low melatonin	<p>Melatonin levels decline to less than 50% by the age of 40.</p> <p>Melatonin is a natural killer cell activator^{16,17,18,19,20,21}</p> <p>Melatonin has been used in the treatment of breast cancer.²¹</p>	<p>Encourage the use of melatonin in all patients whether sleep is an issue or not.</p>
High insulin	<p>Higher insulin levels are associated with increased cancer.^{22,23,24,25}</p> <p>Metformin lowers the incidence and recurrence of cancer.^{22,26}</p>	<p>Use a Metabolic Balance Program and Metformin as needed.</p>
T3	<p>Studies have shown a significant relationship of breast cancer with low T3, but not with T4 or TSH.²⁷</p> <p>T3 increases NK activity.²⁸</p> <p>T3 modulates our immune response by increasing Interleukin-2 receptors.²⁹</p>	<p>Thyroid should be corrected into the optimal range in every patient with T3 and T4.</p>

Basically, we don't get cancers when our hormones are raging and balanced; cancers grow when the hormones are low and imbalanced. Most people who get cancer are NOT on hormones.^{41,42}

Informed Consent for hormones

Every patient, male and female, is consented for hormone restoration. Full consent forms are posted at www.sajune.com.⁴⁰

The highlights of the hormone consent for women include:⁴⁰

1. In 2002, the WHI study reported data on over 16,000 women using oral Conjugated equine estrogen (Premarin) and a progestin (Medroxyprogesterone acetate). They reported 7 more heart attacks, 8 more breast cancers, 8 more strokes, 18 more clots and 6 fewer hip fractures per 10,000 women.
2. In 2010, the French Cohort study reported data on over 80,000 women using bioidentical progesterone and transdermal estradiol. They reported no increase in breast cancer. The addition of progestins increased the risk of breast cancer 69%.

The highlights of the hormone consent for men include:⁴⁰

1. Low testosterone levels are associated with increased heart attacks and prostate cancers. Side effects of excessively high testosterone levels include aggression, oily skin, hair loss, testicular shrinkage and infertility.
2. In a study of over 11,000 men, testosterone levels above 564 have been associated with a 41% lower incidence of cardiovascular mortality.

Nutrient corrections

Hormonal restoration should always be complimented with appropriate nutritional interventions. Also, nutrient deficiencies are known to cause immune compromise and increased cancer cell proliferation. Some facts to share with your patients include:

1. Methyl groups from cruciferous vegetables are needed to get rid of toxic estrogen metabolites.^{31,32,33}
2. Deficiencies of Vitamin D^{34,35},

iodine³⁶, zinc³⁷ and selenium²⁷ are associated with increased cancers.

3. Plant-based nutrition is associated with decreased cancer, while animal protein, such as casein from cow's dairy, is associated with a significant increase in cancer.³⁸

The nutritional program has three components:

1. The most important component is food, primarily based in Plant-based proteins and superfoods (such as aloe, maca, chlorella, coconut).

The intestines are where 75% of the immune system resides in the gut and mucosal associated lymphoid tissues. The symptoms of reflux, bloating, indigestion and constipation must be addressed as they indicate immune compromise.

2. Supplements should be minimal, but carefully chosen to avoid preservatives, fillers, magnesium stearate, radiation and heat-treated bottles. Raw materials should be spectrophotometrically tested for contaminants.
3. Measurement of nutrient status is a must. Using Spectracell Functional Intracellular Analysis™ for micronutrient levels, I have found a consistent pattern of significant deficiencies in patients with cancer. The most common ones include zinc, selenium, magnesium, Vitamin D, glutathione and anti-oxidant function. Obviously correcting these imbalances is part of a

comprehensive program in these patients.

Addressing toxins

Toxins block the normal function hormones and also the immune system. To address toxins you should:

1. Reduce exposure to electromagnetic fields (from cell phones and computers), skin and body products with known carcinogens, processed or GMO foods, household products, pesticides, phalates from plastics and the list goes on.
2. Enhance normal elimination systems by optimizing function of intestines, liver, gall bladder, lymphatic system, kidneys and skin. Aim for three bowel movements a day.

Some facts to share with your patients:

- The intestines are where 75% of the immune system resides in the gut and mucosal associated lymphoid tissues. The symptoms of reflux, bloating, indigestion and constipation must be addressed as they indicate immune compromise.
- The liver is the organ that processes hormones and removes toxins, dumping them into the intestines.
- Toxins, such as heavy metals, phalates and Bisphenol-A, block hormone function, compromise the immune system and can be measured in the urine.
- Anything you place on your skin or body is absorbed as efficiently as if you ingested it orally. So if you would not eat it, think twice about using it on your body.
- Increasing the pH of the tissues allows the body to naturally get rid of toxins. The most effective general principles

include increasing hydration, oxygenation and plant intake.

Mind balance

The patient's conscious and subconscious belief that they can correct their condition is probably the strongest component in recovery and achieving optimal health. Some thoughts to share with your patients :

- Deepak Chopra says that "Our cells are eavesdropping on our thoughts". The new field of neurobioimmunology reports that the minute you have a thought, you produce neuropeptides. All immune cells have receptor sites for neuropeptides, which means that we directly and physically influence our immune cells through our thoughts.
- Simplifying our lives through reduced commitments and reduced nervous system stressors (such as text alerts, TV, news) is one way to direct limited resources (nutrients and hormones) to optimizing the state of your health.
- Meditation, Tai-chi, Qi-Gong and simple breath awareness are effective methods to achieve this.

Body balance

When the body is pain-free and structurally sound, the blood, lymphatic fluids and energetic communications are optimal. A high muscle to fat ratio is associated with increased immunity.

Some facts to share with your patients:

- The recurrence rate for breast cancer is 50% lower with moderate intensity exercise four to five days a week.³⁹
- Exercise, especially a 20-minute

power walk, detoxifies the body by increasing blood flow in the liver, the kidneys and the lymphatic system.

- If you have pain, a neuron is firing. That neuron is using magnesium, B12 and all the other nutrients at a much higher rate than baseline. So, pain must be addressed.
- If you have a physical trauma including surgical scars, it can create a block in the flow of electrical impulses through the meridians.

Conclusion

Most people who get cancer or recurrent cancer are not on hormone restoration; they have hormone imbalances. They also have imbalances in nutrients, toxins, mind and body. Whether or not they ever balance their hormones, it is important to optimize as many of these areas as possible and desirable by the patient.

When the women with breast cancer or the man with prostate cancer walks back into your office saying "Doctor, I have good energy. I can sleep finally. My sex drive is back. I am happy." You can be sure that you have improved **both** their quality of life and their immune system by using a comprehensive approach to hormone restoration.

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Sangeeta Pati, MD, FACOG

Dr. Pati is a Georgetown University trained physician who has practiced traditional and holistic medicine extensively throughout the U.S. and internationally, for over 20 years. She is recognized by physicians all over the world as a foremost authority in the field of Bio-Identical Hormone Replacement Therapy.

Dr. Pati holds board certifications from the American Board of Obstetrics and Gynecology and the American Anti-Aging Board of Medicine.

She practices in Orlando, Florida.

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WEDNESDAY, May 16, 2012

▶ A4M BOARD CERTIFICATION WRITTEN EXAM

6:30pm-9:30pm **ABAARM/ABAAHP Written Exam**

THURSDAY, MAY 17, 2012

▶ A4M PRE-CONFERENCE WORKSHOPS

9:00am – 5:00pm **Putting it all Together: The Nuts and Bolts of Hormone Restoration in Men and Women**
Presented by: Sangeeta Pati, MD

9:00am – 5:00pm **Transforming Theoretical Into Practical: A Multidimensional, Integrative Approach to Treating Patients with Hormone Imbalance & HPA Axis Dysfunction**
Presented by: Lena Edwards, MD

8:00am – 5:00pm **Legal Medical Practice Seminar * (Non-CME)**

8:30am – 5:30pm **The Nuts and Bolts of Nutritional IV Therapy * (Non-CME)**
Presented by: Guy DaSilva, MD and Lark Swofford, RPh

▶ A4M BOARD CERTIFICATION ORAL EXAM

9:00am – 5:00pm **ABAARM Oral Exam**

▶ FAARM FELLOWSHIP – MODULES I, V, XVII

7:30am – 6:00pm **Module I: A Metabolic, Anti-Aging and Functional Approach to Endocrinology**

7:30am – 6:00pm **Module V: Clinical Intensives**

7:30am – 6:00pm **Module XVII - ACUP: Medical Acupuncture for the Integrative Physician/Practitioner**

▶ STEM CELL FELLOWSHIP – MODULE VI

7:30am – 6:00pm **Module VI: Stem Cells at the Frontiers of Disease and Aging**

▶ NETWORKING RECEPTION

6:00pm – 7:30pm **Networking Reception in the Exhibit Hall**

▶ SPONSORED WORKSHOP

7:30 pm - 9:00pm **Evening Workshops**

FRIDAY, MAY 18, 2012 • EXHIBIT HALL HOURS 11:00AM – 6:00PM

▶ FAARM FELLOWSHIP – MODULES I, V, XVII

7:30am – 6:00pm **Module I: A Metabolic, Anti-Aging and Functional Approach to Endocrinology**

7:30am – 6:00pm **Module V: Clinical Intensives**

7:00am – 7:00pm **Module XVII - ACUP: Medical Acupuncture for the Integrative Physician/Practitioner**

FRIDAY, MAY 18, 2012 • EXHIBIT HALL HOURS 11:00AM – 6:00PM CONTINUED

▶ STEM CELL FELLOWSHIP – MODULE VI

7:30am – 6:00pm **Module VI:** Stem Cells at the Frontiers of Disease and Aging

▶ CONFERENCE TRACKS

7:00am – 11:00am **General Session**

1:00pm – 4:00pm **Track 1:** Advanced Protocols In Anti-Aging Medicine

1:00pm – 4:00pm **Track 2:** Hormones and Nutritional Deficiencies

1:00pm – 4:00pm **Track 3:** A Metabolic Approach to Gut Health

1:00pm - 4:00pm **Track 4:** Aesthetic Medicine

▶ A4M BOARD CERTIFICATION ORAL EXAM

9:00am – 5:00pm **ABAARM Oral Exams**

▶ SPONSORED WORKSHOPS

6:30 pm-9:00 pm **Evening Workshops**

SATURDAY, MAY 19, 2012 • EXHIBIT HALL HOURS 10:00AM – 3:00PM

▶ FAARM FELLOWSHIP – MODULES I, V, XVII

7:00am – 5:30pm **Module I:** A Metabolic, Anti-Aging and Functional Approach to Endocrinology

7:00am – 5:30pm **Module V:** Clinical Intensives

7:00am – 7:00pm **Module XVII - ACUP:** Medical Acupuncture for the Integrative Physician/
Practitioner

▶ STEM CELL FELLOWSHIP – MODULE VI

7:00am – 5:30pm **Module VI:** Stem Cells at the Frontiers of Disease and Aging

▶ CONFERENCE TRACKS

7:00am – 11:00am **General Session**

1:00pm – 3:30pm **Track 1:** Advanced Protocols In Anti-Aging Medicine

1:00pm – 4:00pm **Track 2:** Hormones and Nutritional Deficiencies

1:00pm – 4:00pm **Track 3:** Advances in Anti-Aging Medicine

1:00pm - 4:00pm **Track 4:** Aesthetic Medicine

▶ A4M BOARD CERTIFICATION ORAL EXAM

9:00am – 5:00pm **ABAARM Oral Exam**

▶ EXHIBIT HALL

12:30pm **ENTER TO WIN – A Set of His and Hers Rolex Watches!**
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THURSDAY, MAY 17, 2012

- ▶ **NETWORKING RECEPTION:** We invite you to join us for cocktails and hors d'oeuvres in the Exhibit Hall
Room: Exhibit Hall
Time: 6:00pm – 7:30pm
- ▶ **CONNECTING PATIENTS WITH QUALIFIED HEALTHCARE PROFESSIONALS**
Presented by: ForeverHealth
Time: 7:30pm - 9:30pm
Special Guest Speaker: Suzanne Somers

FRIDAY, MAY 18, 2012

- ▶ **“I HAVE A CANCER, CAN I RESTORE MY HORMONES?”** Case Presentations on Restoring Hormones In the Woman With Breast Cancer and the Man With Prostate Cancer – Science And Application
Presented by: MD Prescriptives
Time: 6:30pm - 9:00pm
Speakers: Sangeeta Pati, MD, OBGYN with Special Guest Speaker: Dipnarine Maharaj, MD presents “Storing your Stem Cells for the Future”
- ▶ **THE LATEST IN THYROID THERAPIES –** Learn the Dangers of Ignoring TSH, the Benefits of TRH, Optimal T3/ reverse T3 ratio, the Connection Between Soy and Thyroid.
Presented by: Access Medical Laboratories
Time: 6:30pm - 9:00pm
Speaker: Edwin Lee, MD
- ▶ **CLINICAL TRIALS IN STEM CELL THERAPY: Paving the Way to a Healthier Future**
Presented by: The Ageless Regenerative Institute
Time: 6:30pm – 9:00pm
Speaker: Sharon McQuillan, MD
- ▶ **START YOUR OWN SUCCESSFUL CASH PRACTICE**
Presented by: Holtorf Medical Group
Time: 6:30pm - 9:00pm
Speaker: Kent Holtorf, MD
- ▶ **SEE ONE, DO ONE, TEACH ONE -** Incorporating a Metabolic Syndrome Protocol and HCG Weight Loss Program into your Practice.
Presented by: Homefirst
Time: 6:30pm - 9:00pm
Speaker: Mayer Eisenstein, MD, JD, MPH
- ▶ **LAB DOM (SUISSE) Latest Innovation in Cellular Therapy, Peptide Therapy & Precursor Stem Cells Technologies.**
Presented by: Lab Dom Suisse, Inc.
Time: 6:30pm - 9:00pm
Speaker: Dr. Anita Baxas, Dr. Edwin Vodak, Dr. John Wood, Dr. Mikhail Teppone and Michael Lodge - CEO - Lab Dom Suisse, Inc.
- ▶ **LOWER LEPTIN IS THE BIGGEST BREAKTHROUGH IN BODY FAT REVERSAL: Getting Thinner, Smarter, and Protect Yourself from Nuclear Crisis as you Get Older.**
Presented by: PATH Medical
Time: 6:30pm - 9:00pm
Speaker: Eric Braverman, MD and (Retired) General Bernard Loeffke, PA-C

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▶ WEIGHT LOSS CERTIFICATION

Courses: Module XIV: A-D

Module A: "Individualized Weight Management For The Patient" focuses on the complex causes of weight gain, individualized therapies and the roles of several factors on weight gain. Also covered are the science of food addiction and how to recognize genetic markers for effective recommendations.

Module B: The prevalence and risk factors of obesity are discussed in "Comprehensive Weight Loss for the Integrative Physician," as well as how the body processes nutrients. Several nutritional plans are reviewed, including testing for metabolic and digestive issues in applying weight loss therapies. The module also explores the influence of hormonal imbalances, eating disorders, inflammation, allergies and sleep disorders on weight changes.

Module C: "Weight Management" explores the body's immediate and cumulative responses to exercise intervention programs. The skeletal muscular changes that take place in obese and diabetic patients are presented, in addition to the maintenance of gastrointestinal health (via the 4R program, probiotics, etc.) and its role in energy and weight changes.

Module D: The goal of "Getting Healthy Together" is to teach clinicians how to consult with organizations on placing weight loss and health programs into groups of people, including houses of worship, schools, businesses, and communities.

Requirements: completion of FAARFM Modules XIV A-D; completion of USF Courses XIV 1-4; A4M Membership

DON'T MISS THE NEXT EVENT!

Module XIV (D) will be offered at the A4M event September 20-22, 2012 in Chicago, IL
Module XIV (A) will be offered at the A4M event November 1-3, 2012 in Atlanta, GA

► BRAIN FITNESS CERTIFICATION

Courses: Module XV: A-D

Module A: “The Basics of Brain Fitness and Memory Maintenance” explores factors that help or hinder cognitive functioning, specifically memory. Negative influences such as toxins, medications, stress and inflammation are outlined, while pro-cognitive factors like exercise, nutrients, good sleep hygiene and fatty acids are presented. Cognitive testing, relevant brain anatomy and risk factors for Alzheimer’s and dementia are also covered.

Module B: An in-depth look at the biology, anatomy and physiology of the brain is taken in “How the Brain Learns and Metabolism of the Brain,” particularly how it processes information. The stages and types of memory are reviewed, as well as factors that affect retention and learning, including gender differences. Other topics include Bloom’s Taxonomy, the brain and the arts, human attention and healthy brain aging.

Module C: “Memory Loss: A Practical Guide for Clinicians” instructs how to evaluate memory loss in patients and how to make a differential diagnosis, comprising such conditions as Alzheimer’s disease, vascular dementia and vascular cognitive impairment, frontotemporal dementia, normal pressure hydrocephalus and more. The behavioral and psychological symptoms of dementia are discussed, as well as medications for memory loss.

Module D: “Brain Fitness Therapies” outlines a road map for healthy brain aging, including implementing factors such as nutrients, sleep, diet, exercise and creative engagement to preserve cognitive function. The link between cardiovascular risk factors and cerebrovascular disease, and the role of excitotoxins in memory loss are also covered.

Requirements: completion of FAARFM Modules XV A-D; completion of USF Courses XV 1-4; A4M Membership

DON'T MISS THE NEXT EVENT!

Module XV (B) will be offered at the A4M event June 22-24, 2012 in Dallas, TX

Module XV (C) will be offered at the A4M event October 12-14, 2012 in Tampa, FL

Module XV (D) will be offered at the A4M event December 13-15, 2012 in Las Vegas, NV

► METABOLIC CARDIOVASCULAR HEALTH CERTIFICATION

Courses: XVI A-D

Module A: Vascular biology, vascular aging and vascular disease are the focus here. The module teaches how to apply nutrition, exercise and weight management programs in treating such conditions, as well as the clinical presentation and cardiovascular relationships among vascular biology, vascular aging and vascular disease. Laboratory testing and new, noninvasive diagnostic cardiovascular tests are also reviewed.

Module B: The pathophysiology of hypertension, dyslipidemia, cardiovascular disease and heavy metal toxicity in CVD are explored, as well as methods of patient care through weight management, nutrition and exercise. Also covered are the cardiovascular relationships and clinical presentation of such conditions, in addition to the selection and implementation of laboratory testing and noninvasive diagnostic tests.

Module C: Immunologic vascular disease is reviewed in depth, including pathophysiology, clinical presentation, prevention and treatment. Factors that impact cardiovascular disease, such as nutrigenomics, anxiety and hormonal balance, are explored, as well as methods of testing. The module also discusses nutritional and dietary therapies for prevention and treatment of cardiovascular disease.

Module D: The roles of various conditions in cardiovascular disease—such as dysglycemia, insulin resistance and diabetes mellitus—are presented. Methods for the prevention and treatment of cardiovascular disease, including stem cells and chelation therapy, are explored, in addition to occupational risk factors for heart disease, the effects of toxins in the heart and the role of solvents in the development of arrhythmias.

Requirements: completion of FAARFM Modules XVI A-D; completion of USF Courses XVI 1-4; A4M Membership

DON'T MISS THE NEXT EVENT!

Module XVI (C) will be offered at the A4M event October 12-14, 2012 in Tampa, FL

Module XVI (D) will be offered at the A4M event December 13-15, 2012 in Las Vegas, NV



► SPORTS MEDICINE AND NUTRITION CERTIFICATION

Courses: XIX A-D

Modules A-D: Helping athletes reach peak performance and success is the goal of these modules, which comprises the “science of eating,” diet programs, recipes and key nutrients. Factors that hinder such success are also reviewed, as well as the body’s physiological response to exercise, treatments for sports-related conditions, biometrics, eating disorders, the aging athlete and psychology.

Requirements: completion of FAARFM Modules XIX A-D; completion of USF Courses XIX 1-4; A4M Membership

DON'T MISS THE NEXT EVENT!

Module XIX (A) will be offered at the A4M event September 20-22, 2012 in Chicago, IL

► LIFESTYLE COACHING CERTIFICATION

Courses: Module XXIII: A-D

Module A: This module provides information on how to guide the patient to successfully changing lifestyle habits to create sustainable, long-term health. Several steps are discussed to bring the patient through this process and models for success are shown.

Module B: This module discusses the immune system and digestion, along with toxins, detoxification and diet. The key avenues for toxin entry are overviewed, along with digestion, absorption, deposition, and utilization of all nutrients. Diet prescription and food preparation are taught and techniques are reviewed for overcoming objections by the patient.

Module C: This module focuses on the concept of the co-active coaching model. It discusses fundamentals of the model, the basics of the co-active coaching relationship and the coaching power triangle. The program instructs on how to utilize co-active coaching contexts, principles, practices and other skills.

Module D: Several different topics of lifestyle coaching are explored, such as medication-induced nutritional depletions, foods as nutrients, treatment modalities, hormonal balance, energy levels and food additives. The module also discusses other factors that relate to health coaching—such as emotions, intuition, etc.—as well as skills to implement a comprehensive weight loss program for a patient.

Requirements: Completion of FAARFM Modules XXIII – A-D; completion of USF Lifestyle Health Coaching Courses 1-4; A4M Membership

DON'T MISS THE NEXT EVENT!

Module XXIII (A) will be offered at the A4M event June 22-24, 2012 in Dallas, TX

Module XXIII (B) will be offered at the A4M event October 12-14, 2012 in Tampa, FL

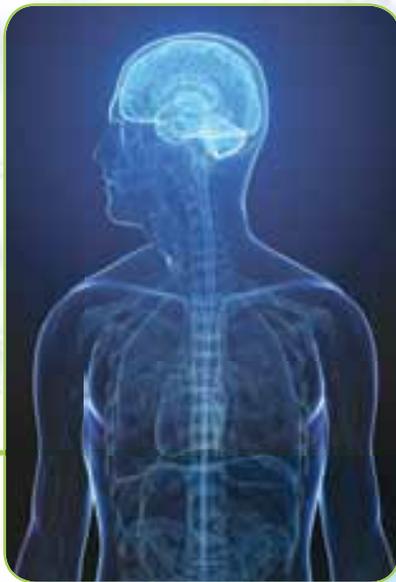
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ABAARM Written Examination

Wednesday, May 16, 2012 from 6:30 pm – 9:30 pm
The Marriott World Center Resort
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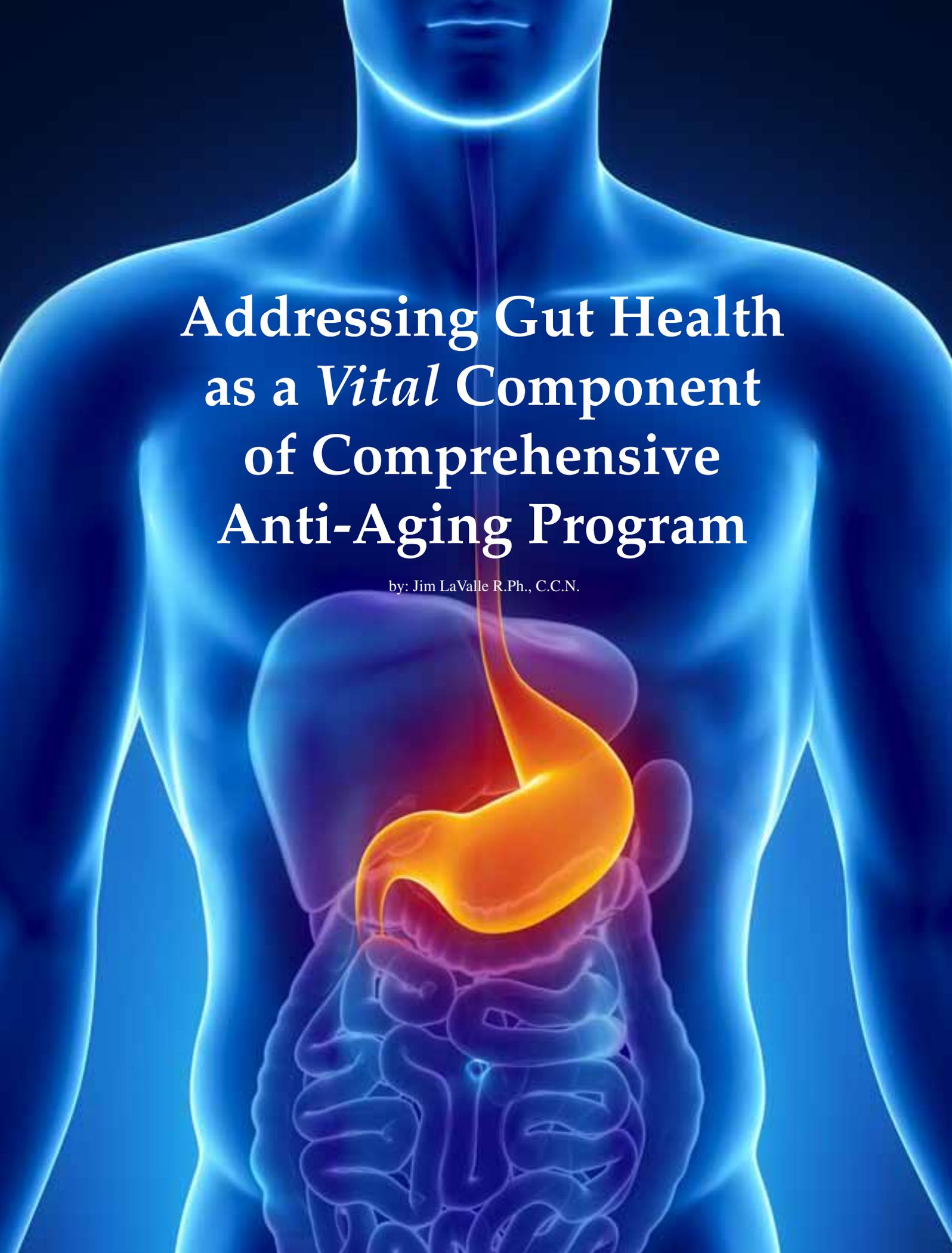
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Addressing Gut Health as a *Vital* Component of Comprehensive Anti-Aging Program

by: Jim LaValle R.Ph., C.C.N.

Many integrative medicine practitioners are now addressing their patients' health needs with a multi-systems "metabolic" approach, evaluating thyroid health, adrenal/stress issues, insulin and glucose regulation, toxicity, gut health, and regulation and balance of sex hormones. However, many times a program for gut health is not implemented unless gut-specific symptoms are glaringly present. As more information comes to light on the roles of a healthy intestinal barrier, it is becoming evident that addressing gut health is foundational to health and should be addressed for prevention of not just diarrheal episodes or irritable bowel but for everything from autoimmunity to heart disease.

The intestinal epithelium used to be strictly seen as important for the digestion and absorption of nutrients, but continued research is elucidating how important the "barrier" function is and furthermore that gut flora plays a central role in maintaining that barrier. In addition, we are learning how much the gut flora via this barrier impact and regulate the immune system. Some practitioners are now becoming aware of the role gut flora play in regulating and controlling the immune response, but most are not yet fully aware of just how much imbalances in gut flora can affect overall health and contribute to systemic inflammation via immune system dysregulation. This article will review the role gut microbiota play in immune regulation, and how dysregulation can lead not just to immune-related conditions but can even contribute to heart disease. It will also discuss how to re-regulate the intestinal milieu for improved management of

conditions or as part of a pro-active anti-aging program.

Background

To review, the human gastrointestinal tract contains an ecosystem containing hundreds of different microbial species, mostly bacterial and some fungal (yeast strains). Some of the primary commensal bacteria (probiotic) are the lactobacillus, bifidobacteria, bacteroides, eubacteria, streptococcus, and escheridia families. Commensal flora have long been known to be involved in host beneficial activities, such as helping to protect against pathogenic bacteria activity and production of certain vitamins. However, a much more complete picture is emerging as to how probiotics are involved in helping to maintain gastrointestinal linings as well as the role they play in systemic immune-regulating activity.

There are several mechanisms by which probiotic bacteria help maintain the mucosal lining, which consists of a single layer of epithelial cells (enterocytes) coated with mucous. In addition there are tight junction proteins between cells, which are critical to preventing the unintentional entry of intestinal contents into the bloodstream. First probiotics help produce short chain fatty acids, which have a variety of functions. As probiotics ferment fiber from the diet, short chain fatty acids are produced as a byproduct. These acids can damage the outer layers of pathogenic bacteria, helping to keep the unfriendly flora from proliferating and creating dysbiosis. In addition, the lower pH of the contents of the intestinal lumina also stimulates intestinal motility. And finally the SCFAs, like butyric acid, are a primary source of

fuel for enterocytes and also help to maintain viability¹.

In addition, probiotic signaling results the expression of several important proteins, such as those that increase mucin production by goblet cells. The mucin production is an important intestinal defense, helping to prevent adhesion of pathogenic bacteria and potential antigens in food or pollens, which enter into the intestine via swallowed nasal secretions. Other proteins that probiotics stimulate are B-defensin proteins, which are anti bacterial. Probiotics stimulate secretory IgA production and production of tight junction proteins. Secretory IgA binds not only to food and other antigens but to bacteria and viruses as well, and so is a primary substance in immune system activity, providing needed immune defense against bacteria and viruses while preventing unwanted over-activation of immune system in response to food or other antigens like pollen. If immune activation becomes excessive it can become a primary source of inflammatory cytokine production.

The multiple ways in which probiotics help hold pathogenic bacteria populations down while maintaining gut barrier function are also extremely important in preventive health. Recent research has found that endotoxins from these bacteria are a principle mediator of vascular inflammation that leads to atherosclerosis². While it's long been known that bacterial endotoxins can stimulate inflammation in vascular tissue, it has not been known that the gastrointestinal tract could be a primary source of these bacteria.

One endotoxin in particular, lipopolysaccharide (LPS), stimulates the production of toll-like receptor 4

(TLR-4) which goes on to stimulate a significant activation of immune cells and triggers inflammatory cytokine production³. There are TLR-4s located on cardiac cells. Heart failure is recognized as a multi-organ disease. Decreased blood perfusion leads to intestinal ischemia and altered permeability. When LPS are present it brings immune cell attack into cardiac tissue and actually starts to reduce contractility in the heart muscle; thus it's been identified that the gastrointestinal tract can play a role in heart failure. LPS can trigger cytokine release from muscle and other organs leading to damage⁴. Even low levels of LPS can reduce cardiac contractility and beta-adrenergic response, and induce heightened oxidative stress⁵. In a study published in March of this year the probiotic *Lactobacillus plantarum* altered genomic DNA expression and inhibited signal pathways of LPS related to TNF α , TLR4, TLR9⁶. LPA also has been shown to have anti-thyroid effects.

Gut barrier function then depends heavily upon well-balanced intestinal flora. So when probiotic populations are lacking there can be a tremendous breakdown in the integrity of the intestinal lining. With lowered production of SCFA's enterocytes lack the necessary fuel and can die off. If tight junction proteins are eroded, the gut becomes permeable to any molecule that can leak through. Reduced mucin can allow antigens to adhere to intestinal wall, bringing antigens into close proximity to immune cells in the submucosa resulting in heightened immune response. In addition, pathogenic bacteria populations will not be held in check, contributing to inflammation and disease via the effects of bacterial endotoxins.

Gut Microbiota Alteration

Gut flora can be altered by a variety of factors, including medications (antibiotics, oral contraceptives, proton pump inhibitors, corticosteroids, NSAIDs), sugar intake, bactericidal chemicals in water, pesticides residues in food, alcohol, stress, radiation, and intense exercise. Some factors alter flora by destroying both commensal and pathogenic bacteria, especially antibiotics. Other factors may cause imbalance by encouraging over-proliferation of a certain species, such as sugar intake resulting in promoting yeast overgrowth. *Candida* overgrowth has been associated with a variety of illnesses and disturbance. One of the underappreciated aspects of this is the production of mycotoxins and acetaldehyde. Acetaldehyde production is up regulated by fermentation processes, and is associated with liver damage along with alterations in dopamine and serotonin. Acetaldehyde causes dopamine to convert to salsolinol, and serotonin to beta-carboline both of which act as opioid agonists⁷. Acetaldehyde attaches to red blood cells limiting their penetration into capillaries, with a net result of compromised oxygen delivery⁸. Acetyl Coenzyme A production of energy is limited by acetaldehyde in a dose dependent fashion so cellular energy falters⁹.

A review of the history and progression of clinical assessment in chronic fatigue is a good example which can help elucidate the role of gut flora in immune aberrations and progression to a disease or syndrome. Initial medical investigations into possible etiology of chronic fatigue syndrome found an association between viral micro infections, like Epstein-Barre and cytomegalovirus, and chronic

fatigue. This made sense of the flu-like symptoms that are often seen in the condition. A next evolution was investigation into thyroid function, which made sense, since the thyroid hormone is responsible for the driving the cellular production of ATP, the body's energy molecule. Most people will recall that a metabolic systems approach to chronic fatigue had us focusing on the role of the thyroid in chronic fatigue.

A prime example is in the case of chronic fatigue syndrome (CFS), in which the symptoms are so extensive it can be easy to overlook a history of bowel symptoms, which studies have found as high as 92% of chronic fatigue patients report having had bowel symptoms at some time in their life¹⁰. Yet, dysbiosis seen in its new light, can be clearly linked to thyroid disturbances, lack of immune integrity, pain and mental symptoms.

In summary, assessing gut health and maximizing GI metabolism should be considered a primary purpose in supporting health and is an integral part of any anti-aging program.

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- 3 Yew, W. Endotoxin and the cardiovascular system. *J Am Coll Cardio* 2003;42:1633-1665.
- 4 Krack A, Sharma, R, Figulla HR, and anker SD. The importance of the gastrointestinal tract in the pathogenesis of heart failure. *Eur Heart Journal* 2005; 26:2368-2374.

For additional references please visit <http://www.a4m.com/conferences-exhibitors-a4m-medical-news.html>

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Once received, you're abstract and application will be reviewed by the A4M Program Committee and a decision will be communicated to you shortly thereafter. Please send all of the required materials to program@a4m.com. Please specify the name of the event you are applying for.

Thank you,

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DUBAI

Background

Dubai, one of 7 Emirates in the United Arab Emirates (UAE), is quickly becoming the primary destination for A4M overseas educational activities. Its favourable geographic position within the Arabian Peninsula and strong private healthcare system undoubtedly lends itself to a favourable setting for A4M's medical education events. Recent government initiatives have not only boosted Dubai's private healthcare sector but also propelled the city to become one of the top medical tourism destinations in the region. In addition to this, the UAE healthcare sector has proven robust against the global downward economic trend, and therefore remains a focus of investment by many private equity players. This is further enhanced by the region's ageing population and life-style related diseases such as diabetes which increase demand for innovative preventive, anti-aging protocols.

In 2011



In 2011, A4M launched its Aesthetic Anti-Aging Fellowship program in Dubai by affiliating itself to the region's top clinic for minimally invasive aesthetic procedures. Aesthetica clinic is now the overseas unique location for the program's clinical portion which teaches physicians the art of facial and body enhancement. The hands-on modules are supervised by Dr. Maria Angelo-Khattar who is a well renowned aesthetic expert in the region.

A4M also started to deploy a series of intensive seminars on the topic of BHRT which is becoming an increasingly hot topic since the licensing and opening of the first ever compounding pharmacy in Dubai last year.

Coming up!

The Aesthetic Fellowship "hands-on" modules IV and IV will be taught at Aesthetica Clinic on June 01-04 2012, and again in November.

A brand new Anti-Aging Medicine Symposium focusing on Bio-Identical Hormones and Nutritional Therapies will be presented by some of A4M's most eminent opinion leaders on November 10-11 at the Address Hotel. This will be complemented by Module V of the Anti-Aging Fellowship program (FAARM) and by written and oral Board examinations (ABAARM).



THAILAND

Background



Thailand is often considered as the medical hub of Asia with the majority of private and public practices now focusing on improving their standard of services as well as upgrading their facilities. The country offers a world-class medical infrastructure and the highest possible quality of care at a fraction of the cost of

similar procedures in developed countries. Thailand provides a range of healthcare services including sophisticated procedures like stem cell treatment and the demand for such treatment has increased not only due to the number of foreign patients but also because of the health-conscious consumers in Thailand.

A4M, in partnership with A4M Thailand, has been spreading information on cutting-edge preventive protocols at its congress which is held annually in Bangkok and has previously gathered up to 1,000 enthusiastic Thai and Asian physicians.

Coming up!

The next congress will be held on 07-09 September and will bring together US and local experts to present the latest research on anti-aging medicine to the local audience. Aesthetic medicine, being also a high growth medical area in Thailand, will be a featured topic presented by the exclusive event partner, ThaiCosderm. Due to popular demand, A4M now also hosts ABAARM written and oral exams in Bangkok.



AUSTRALIA

Background

The rising prevalence of lifestyle related chronic disease coupled with an ageing population and the related burden on the healthcare system is signalling a definite evolution of healthcare practice in Australia to a Preventative model. With one-third of Australian GPs and half of Pharmacists reporting practicing Integrative Medicine and Specialised compounding services in Australia having increased from 35 to over 200 in the past decade, it is no wonder that preventive, anti-aging medicine has flourished in this part of the world.



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The Role of **Mercury** in Cardiovascular Disease, Hypertension, and Stroke



By Mark C. Houston MD, MS, ABAARM, FACP, FAHA

Abstract and Summary

Mercury and cadmium have a high affinity for sulfhydryl (-SH) groups, inactivating numerous enzymatic reactions, amino acids, and sulfur-containing antioxidants (NAC, ALA, GSH), with subsequent decreased oxidant defense and increased oxidative stress. Both bind to metallothionein and substitute for zinc, copper, and other trace metals reducing the effectiveness of metalloenzymes. Mercury induces mitochondrial dysfunction with reduction in ATP, depletion of glutathione, and increased lipid peroxidation. An increased oxidative stress and reduced oxidative defense are common. Selenium and fish containing omega 3 fatty acids antagonize mercury toxicity. The overall vascular effects of mercury include increased oxidative stress and inflammation, reduced oxidative defense,

thrombosis, vascular smooth muscle dysfunction, endothelial dysfunction, dyslipidemia, immune and mitochondrial dysfunction. The clinical consequences of mercury toxicity include hypertension, CHD, MI, cardiac arrhythmias, reduced heart rate variability, increased carotid IMT and carotid artery obstruction, CVA, generalized atherosclerosis, and renal dysfunction, insufficiency and proteinuria. Pathological, biochemical, and functional medicine correlations are significant and logical. Mercury diminishes the protective effect of fish and omega-3 fatty acids. Mercury and cadmium inactivate COMT, which increases serum and urinary epinephrine, norepinephrine, and dopamine. This effect will increase blood pressure and may be a clinical clue to heavy metal toxicity. Cadmium concentrates

specifically in the kidney, inducing proteinuria, renal dysfunction and insufficiency. Cadmium is associated with hypertension, but less so with CHD, CVD and CVA.. Renal cadmium reduces CYP4A11 and PPARs, which may be related to hypertension, sodium retention, glucose intolerance, dyslipidemia, and zinc deficiency. Dietary calcium may mitigate some of the toxicity of cadmium. Mercury and cadmium heavy metal toxicity should be evaluated in any patient with hypertension, CHD CVD, CVA or other vascular disease. Specific testing for acute and chronic toxicity and total body burden using hair, toenail, urine and serum should be done.

INTRODUCTION

There is increasing concern regarding the overall health effects of chronic exposure to various

heavy metals in the environment. This is particularly true of mercury and less so with cadmium, lead, aluminum, iron and arsenic. The cardiovascular consequences of mercury and cadmium toxicity have not been carefully evaluated until recently. This paper will critically review the cardiovascular

exposure, fish and sea mammals are becoming an increasing environment source of potential mercury toxicity.^{1,2,4,5}

MERCURY BIOTRANSFORMATION AND BIOMETHYLATION

Mercury from various sources, including elemental mercury from earth sources or inhaled mercury vapor, methyl and ethyl mercury are converted by biomethylation to inorganic divalent mercury, the toxic form in human organs

per day. The typical amalgam is composed of 50% mercury, 25% silver and 25% tin, copper and nickel.^{4,6,7} Fish and sea mammals provide about 2 – 3 micrograms per day depending on the type and amount consumed.^{1,2,4,5} The long-lived large predatory fish such as swordfish, tilefish, shark and king mackerel contain about one microgram of methyl mercury per gram. Pike, whale, bass, tuna and trout are about 0.1 – 0.5 micrograms of mercury per gram. Nine vaccines that contain thimerosal (50 % mercury) as a preservative would give an estimated exposure of 62 micrograms of organic mercury.^{1,2,4,5} All other sources of mercury provide about 0.3 micrograms per day.^{1,2,4,5}

1. Elemental	Mercury Vapor (Hg ⁰) Stable Monoatomic Gas	Dental Amalga
2. Inorganic	Divalent Mercury (Hg ²⁺)	Toxic species in human tissue after conversion
3. Organic	Methyl Mercury (CH ₃ Hg ⁺) Ethyl Mercury (CH ₃ CH ₃ Hg ⁺)	Fish, sea mammals Thimersol vaccines

consequences of mercury and cadmium toxicity in humans as it relates to hypertension, generalized atherosclerosis, coronary heart disease (CHD), myocardial infarction (MI), cardiac arrhythmias, sudden death, cerebrovascular accidents (CVA), carotid artery disease, renal dysfunction and total mortality.

MERCURY

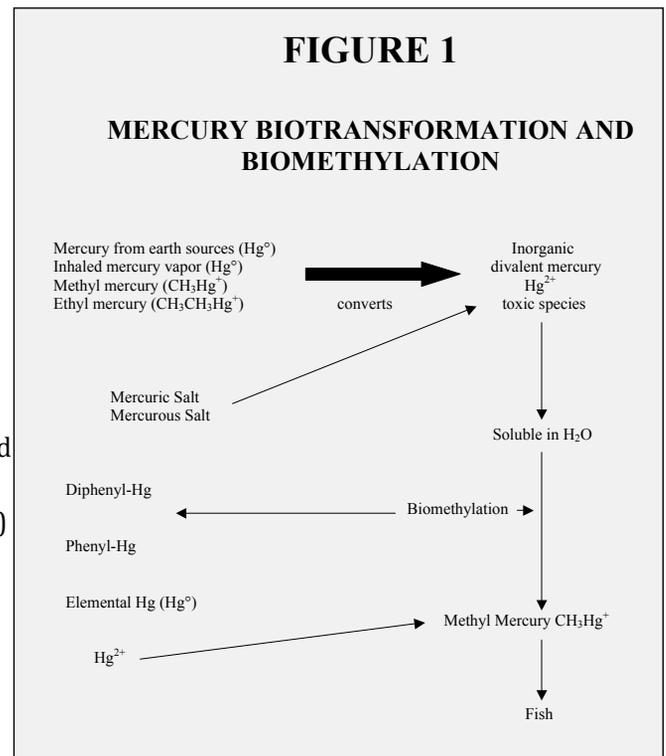
TYPES OF MERCURY

Mercury exists in three basic forms: elemental, inorganic and organic (Table 1).^{1,2,3,4,5} Dental amalgams are the most common source for elemental mercury vapor, which is a stable monoatomic gas. Inorganic mercury, which is a divalent compound, is the toxic species found in human tissue after conversion from the other forms. Organic mercury in the form of methyl and ethyl mercury is primarily from fish, sea mammals and thimersol vaccines. Although dental amalgams have historically been the major source of human

and tissues (Figure 1).⁴ Divalent mercury is soluble and stable in water, undergoes biomethylation to methyl mercury, which is found in high concentrations in certain fish and sea mammals. It is this source that is becoming the major source of human exposure to mercury. The Environmental Protection Agency has determined the safe daily intake of mercury to be less than 0.1 microgram/kg/day.⁴ However, 12 % of women have hair mercury above the level at which stopping consumption of highly contaminated fish would be advisable (1.0ug/g) ⁴. It is estimated that one dental amalgam filling releases about 3 – 17 micrograms of mercury vapor

IMPORTANT FACTS ABOUT MERCURY

Mercury is the most dangerous of all the heavy metals.⁸ It will modify the distribution and retention of other heavy metals.^{9,10,11} Mercury has no known physiological role in human metabolism, and the



human body has no mechanisms to excrete mercury actively.¹² Mercury, thus, accumulates during life so that the average 70 kg person has a total body burden of about 13 mg of mercury.⁸ Mercury has a high affinity for sulfhydryl groups (-SH), various enzymes and amino acids, N-acetyl cysteine (NAC), alpha lipoic acid (ALA) and glutathione (GSH), which provide about 10 – 50% of the plasma protein antioxidant capacity.^{8,12,13} Both NAC and ALA, as well as cysteine, are precursors for glutathione, which is the most potent intracellular anti-oxidant and protects against oxidative stress, inflammation and cardiovascular disease.^{3,4,5,8,9,12} This mercury-induced reduction in oxidant defense and increase in oxidative stress increase the risk for CVD and CVA. Selenium antagonizes some of the adverse effects of mercury by forming a seleno-mercury

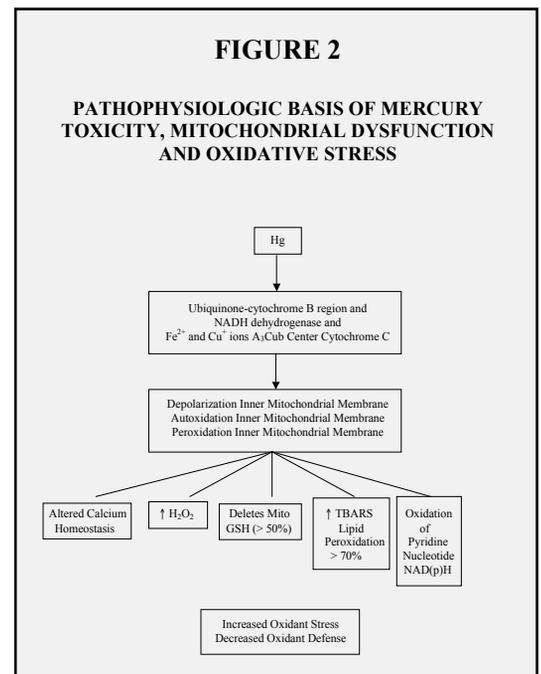
complex in tissue that is less toxic.^{9,14,15,16,17,18,19,20} Higher intake of selenium reduces mercury related CVD and CVA.

PHYSIOLOGICAL BASIS OF MERCURY TOXICITY

Mercury induces mitochondrial dysfunction and oxidative stress.^{21,22,23} The primary mitochondrial dysfunction occurs at the ubiquinone-cytochrome B region and with NADH dehydrogenase causing displacement of Fe⁺⁺ and Cu⁺ ions in the a3Cub center of cytochrome C (Figure 2). This results in depolarization and auto-oxidation of the inner mitochondrial membrane with lipid peroxidation and severe mitochondrial dysfunction. Physiologic consequences include increased hydrogen peroxide, depletion of mitochondrial glutathione by over 50%, increased lipid peroxidation markers such as TBARS by over 70%, oxidation of pyridine nucleotides such as NAD(p)H and altered calcium homeostasis.^{21,22,23} This severe mitochondrial dysfunction increases oxidant stress and reduces oxidant defenses, which has enormous health implications.

The primary three sources of mercury-induced lipid peroxidation include the Fenton reaction, affinity for sulfhydryl groups and selenium deficiency.⁸ Mercury serves as a direct catalyst in Fenton-type reactions and as an indirect catalyst via iron stimulation, which increases the production of radical oxygen species and superoxide anion.⁸ Mercury's high affinity for sulfhydryl groups (-SH), such as glutathione, NAC and ALA, which comprise much of the antioxidant capacity of plasma,

reduces both membrane and plasma antioxidant defense. Finally, insoluble complexes of mercury with selenium reduces selenium availability, which is a necessary co-factor for glutathione peroxidase (GPx) activity to break down hydrogen peroxides and various other toxic peroxidation products which further increases risk for CVD and CVA. Plasma and intracellular antioxidant capacity are both reduced.⁸



VASCULAR BIOLOGICAL EFFECTS OF MERCURY

Numerous toxic effects of mercury have been demonstrated in vitro, animal and human studies (Table 2). Mercury increases free radical production,^{3,24,25,26,27,28,29,30} inactivates antioxidant defenses,^{3,24,25} binds to thiol-containing molecules,^{3,24,25,26,31} binds to selenium forming seleno-mercury complexes reducing selenium availability for GPx activity,^{3,24,25,28,32} inactivates glutathione, catalase and superoxide dismutase,^{31,26,27,28} increases lipid peroxidation,^{29,33,34,35} increases oxidation of LDL (oxLDL) and increases plasma oxLDL complexes.⁸ Thrombosis is

VASCULAR BIOLOGIC EFFECTS OF MERCURY TABLE 2

1. Increased free radical production and increase in oxidative stress
2. Inactivation of antioxidant defenses
3. Mitochondrial dysfunction
4. Binds to thiol-containing molecules(sulfhydryl groups)
5. Binds to SE forming Se-Hg complex-mercury selenide which decreases Se available for cofactor with GPx
6. Inactivates glutathione, catalase, SOD
7. Increases lipid peroxidation in all organs
8. Increases oxLDL and oxLDL immune complexes
9. Increased platelet aggregation and thrombosis
10. Increased coagulation and thrombosis: increases Factor VIII, PF4, thrombin and reduces protein C
11. Inhibit endothelial cell formation and migration and decreases endothelial repair
12. Decreases nitric oxide bioavailability
13. Endothelial dysfunction
14. Increase apoptosis
15. Reduced monocyte function and phagocytosis
16. Immune function is impaired
17. Increased vascular inflammation with increase TNF – alpha and IL-6
18. Stimulation of vascular smooth muscle cells
19. Inactivation of paroxonase and other HDL proteins and enzymes
20. Translocation of membrane phosphatidyl serine
21. Activates phospholipase A2
22. Activates phospholipase D.

potentiated by increased platelet aggregation,³⁶ increases in Factor VIII, platelet factor-4²⁴ and thrombin with reductions in protein C.^{36,37} Endothelial cell formation and migration are reduced, which decreases vascular endothelial repair, decreases nitric oxide and causes endothelial dysfunction.³⁸ Apoptosis is increased,²⁶ monocyte function and phagocytosis are impaired,²⁶ immune function is reduced²⁶ and vascular inflammation is increased with elevations of TNF alpha and IL-6.²⁶ There is an increased production and release of superoxide anion from human neutrophils and monocytes,^{24,26} depolarization of the inner mitochondrial membrane with severe mitochondrial dysfunction^{21,22,23} and disruption of plasma membrane lipid integrity by translocation of phosphatidyl serine (PS).²⁶ Mercury stimulates proliferation of vascular smooth muscle cells³⁹ and inactivates paraoxonase, an extracellular antioxidative enzyme related to HDL, CHD and MI risk.^{40,41} The clinical consequences of these and other pathophysiological mechanisms explains the wide variety of cardiovascular diseases caused by mercury including CHD, MI, arrhythmias abnormal heart rate variability, generalized atherosclerosis, sudden death, CVA carotid artery stenosis, renal dysfunction and hypertension.^{4,5,6,7,8,9,13,15,19,28,42-74.} Mercury activates phospholipase A2 (PLA-2) and induces formation of arachidonic acid metabolites such as total prostaglandins, thromboxane B2 and 8 isoprostane in vascular endothelial cells and activates vascular endothelial cell phospholipase D.^{54,55,56,57,58.} Many of the cardiovascular consequences of mercury are

mitigated by concomitant intake of fish containing omega 3 fatty acids and by the intake of selenium^{8,48-53}. Even very low levels of chronic mercury exposure promote endothelial dysfunction as a result of increased inflammation, oxidative stress, reduced oxidative defense, reduction in nitric oxide (NO) bioavailability which increase the risk of CVD and CVA²⁷.

CLINICAL VASCULAR CONSEQUENCES OF MERCURY TOXICITY

The clinical consequences of mercury toxicity include hypertension,^{13,28,42,43,59,60,61,62,63,71,72,73,74} CHD,^{5,15,44,64,65} MI,^{19,42,44,64,65} reduction in heart rate variability (74), increase in carotid intimal medial thickness (IMT) (73) and carotid obstruction,¹³ CVA,^{42,72} generalized atherosclerosis,⁴ renal dysfunction and proteinuria⁴ and an overall increase in total and cardiovascular mortality.^{4,72} Gomez et al (72), followed 3,998 workers in mercury mines exposed to inorganic mercury from 1895 to 1994 and found a 2.78 x increase incidence of hypertension, 1.17 x increase risk of stroke and 1.51 x increase risk in total cardiovascular mortality, but no increase in CHD. In a study of Faroese whaling men, both toenail and hair mercury levels were significantly associated with increased carotid IMT and hypertension (73). Evidence from these and other epidemiologic and clinical studies suggest that people with high levels of urine, hair, blood and toenail mercury have an increased risk of cardiovascular diseases.^{5,8,9,13,53,60,63,64,71,72,73}

CORONARY HEART DISEASE AND MYOCARDIAL INFARCTION

In rabbits exposed to inhaled mercury vapor, the cardiovascular

and cardiac pathology includes bradycardia, thrombosis in small and medium caliber arteries, focal necrosis with thickening of the endocardium of the perivalvular regions, papillary muscles and valves, endothelial proliferation with inflammatory foci and focal edema, inflammation and fibrosis of the ascending aorta.⁴⁵

In a case control study in nine counties of 684 men with their first MI, there was a significant association of toenail mercury content, adipose tissue DHA and first MI.⁵ There was a 15% higher toenail mercury content as assessed by neutron activation analysis (NAA) in the men with their first MI compared to the control group (95% CI; 5 – 25%). The OR, risk adjusted, for MI was 2.16 in highest versus the lowest quintile (p = 0.006, 95% CI; 1.09 – 4.29). The adipose DHA was directly proportional to the mercury toenail content (p < 0.001) and the DHA content was inversely correlated to MI with an OR of 0.59 in the highest versus the lowest quintile (p = 0.02, 95% CI; 0.30 – 1.19). This important study concluded that there exists a positive, monotonic increase in the risk of MI with mercury toenail content above the 0.25 microgram/gram level, which was even steeper when adjusted for the DHA adipose tissue content. Mercury diminishes the cardiovascular protection of fish consumption. Another study substantiated these results in which the highest quartile of DHA with the lowest quartile of mercury was associated with a 67% reduction in CHD (p < 0.016).⁴⁶

However, in another large nested case control study of 33,733 male healthcare professionals

between the ages of 40 – 75 years (Health Professionals Follow-Up Study), no association between mercury toenail content assessed by NAA and CHD was found.⁹ However, if dentists were excluded, there was a nonsignificant correlation of toenail mercury and CHD. Also, subjects with the highest tertile of mercury and the lowest serum selenium level had a significant increase in CHD.

Other human studies have shown mixed results.^{6,8,42,43,47} Mercury miners had no relationship between CHD and serum mercury levels.²⁸ However, another study of European mercury miners showed a significant relationship of mercury exposure to total mortality (increase 8%), hypertension (increase 46%), CHD (increase 36%), renal disease (increase 55%) and CVA (increase 36%).⁴² A Finnish study found a significant relationship between hair mercury, 24-hour urine mercury and cardiovascular events.⁸ In patients with hair mercury in the highest tertile, over 2.0 micrograms/gram, and increased 24-hour urinary mercury, CHD and MI risk was increased two-fold ($p = 0.005$), cardiovascular death increased by 2.9 times ($p = 0.014$) and circulating oxLDL and immune complexes to oxLDL increased significantly ($p=0.01$). The Gothenburg Study showed no relationship between serum mercury content and the number of amalgam fillings and CHD or MI.⁶ The National Health and Nutrition Examination Survey from 1999-2002 found that levels of DHA and EPA as well as other nutrients in fish, even with elevated mercury levels, helped to offset the risk of CHD and MI⁽⁶⁶⁾. The fish intake resulted in lower levels of CRP and higher serum HDL cholesterol as

well⁽⁶⁶⁾. The risk of hypertension over ten years was highly correlated in a group of chemical factory workers exposed to mercury vapor⁽⁶⁷⁾. There is also a strong association of mercury toxicity with stroke and carotid atherosclerosis.

13,24,36,37,38,42,43,47,59,60,61,62,63,64 68,69,71

HYPERTENSION

The association of mercury toxicity and hypertension in humans is convincing,^{13, 42, 43, 47, 59, 60, 61, 62, 63, 64,}

⁷¹ Mercury miners were found to have significant increases in systolic blood pressure ($p < 0.01$) that correlated with lipid peroxidation and overall oxidative stress ($p < 0.01$).²⁸ European mercury miners had a 46% greater incidence of hypertension vs. aged-matched controls. Other studies have shown significant correlations with hair mercury content, hypertension and carotid IMT.¹³ In a study of 251 persons in the Brazilian Amazon, BP was significantly associated with total hair mercury levels. The OR for elevated SBP with total hair mercury over 10 mcg/g was 2.91 (1.26-7.28)⁽⁶⁰⁾. In 101 participants in the Wisconsin Sleep Cohort study, those in the upper quartile of blood mercury were 1.9 times more likely to be hypertensive ($p= 0.023$) and those in the upper quartile of hair mercury were 4 times more likely to be hypertensive ($p= 0.02$) but there was no change in brachial artery flow mediated vasodilation or the middle cerebral artery reactivity to CO₂⁽⁶¹⁾. In 732 Inuit adults, blood mercury level was correlated with SBP and pulse pressure ($p=0.0004$) and DBP ($p=0.069$)⁽⁶²⁾. In a comparative population study long term methyl mercury exposure, as measured by hair mercury levels, was associated with a risk of hypertension of 1.4 to 1.6 times in 833 patients⁽⁶³⁾. In a sample

of 1240 women age 16-49 who participated in the National Health and Nutrition Examination Survey 1999-2000, Vupputuri et al⁽⁶⁴⁾, found a significant increase in SBP with increasing levels of blood total mercury, but only among non-fish consumers. There was a 1.83 mm Hg increase in SBP for each 1.3ug/L increase in blood total mercury (95% CI: 0.36, 3.30; interaction $p=0.02$).

Pederson et al⁽⁷¹⁾ found an increase in pulse pressure using 24 hour ambulatory blood pressure monitoring (54mm Hg vs 50 mm Hg with $p<0.0001$) that was related to blood mercury levels ($\rho=.272$, $p < 0.01$) in a group of Greenlanders consuming more fish than a group of Danes. Mercury is also significantly associated with reduced heart rate variability (HRV) in addition to the increased pulse pressure and hypertension⁽⁷⁴⁾. A reduced HRV may predispose to ventricular fibrillation and sudden cardiac death, as well as being associated with angina, MI, CHD, CHF and all cause mortality.⁽⁷⁴⁾

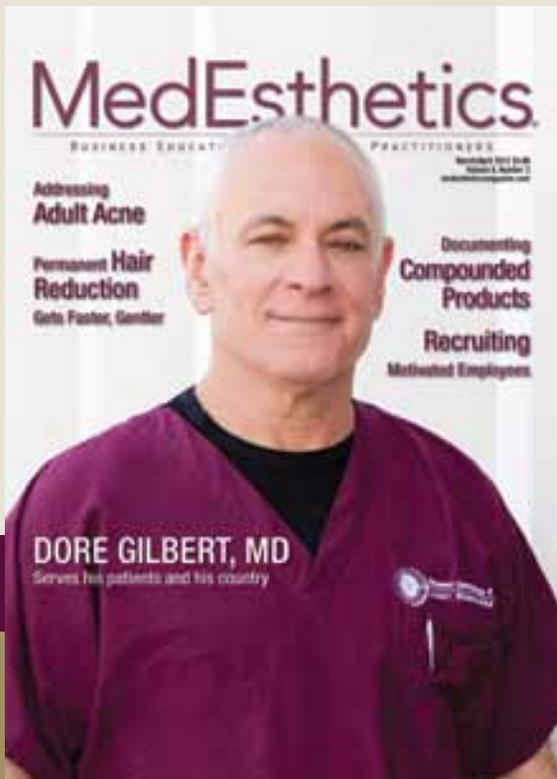
In acute and probably chronic mercury intoxication, mercury binds to the sulfhydryl group S-adenosyl methionine (SAM) and inactivates this enzyme, which is a necessary cofactor for catecholamine-*O*-methyl transferase (COMT), the enzyme needed to convert norepinephrine, epinephrine and dopamine by methoxylation.⁴³ This results in a clinical syndrome that resembles a pheochromocytoma crisis with malignant hypertension in acute mercury intoxication and significant increases in urinary catecholamines in chronic mercury toxicity. This can be a very helpful clinical clue to mercury-induced hypertension. It would be

important to measure baseline and provoked 24 hour urine mercury levels in patients with hypertension and increase history or evidence clinically of possible mercury exposure. Mercury also induces renal dysfunction and proteinuria, which contribute to sodium retention and hypertension.^{27,42,43,48} Studies have shown an increase in renal insufficiency in mercury miners by 55%.⁴² Mercury concentrates in the renal tubules and in the glomerulus and results in proteinuria, fibrosis, chronic renal dysfunction and renal insufficiency.^{27,47}

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FELLOWSHIP IN ANTI-AGING, REGENERATIVE & FUNCTIONAL MEDICINE

Director: Pamela Smith, MD, MPH

Dr. Smith is the director of the Center for Healthy Living and Longevity and the director of the Master's Program in Metabolic and Nutritional Medicine at the University of South Florida School of Medicine.



This modular training program includes hands-on clinical training and web broadcasts to discuss topics and experience with other trainees and experienced clinicians. Open to physicians, physician assistants, nurse clinicians and pharmacists, participants will leave the fellowship competent to practice anti-aging and regenerative medicine without supervision in his or her medical specialty.

How do you describe the content of FAARFM in your own words?

Basically, science has changed, and what we can now do with the help of science is to look at individualized, customized care, and we can now look at the cause of the problem instead of just looking at the symptoms.

What are some of the highlights?

There is the basic fellowship, there's an advanced fellowship and there are many electives along with certifications. To give you an example of our advanced metabolic fellowship in cardiovascular health, one of the head cardiologists in the country attended the first course and left saying, "90% of this information I've never heard before." So what we're doing is very cutting edge.

What do you hope to provide to participating doctors that they can't get elsewhere?

This is a very unique situation where, because we're the only master's program in the field, we have the professors that are the most published and many

times have actually discovered their area of expertise. For example, we have Russell L. Blaylock himself discussing excitotoxins; we have James Wilson himself discussing adrenal fatigue. We have the very best people not just in North America but in the world.

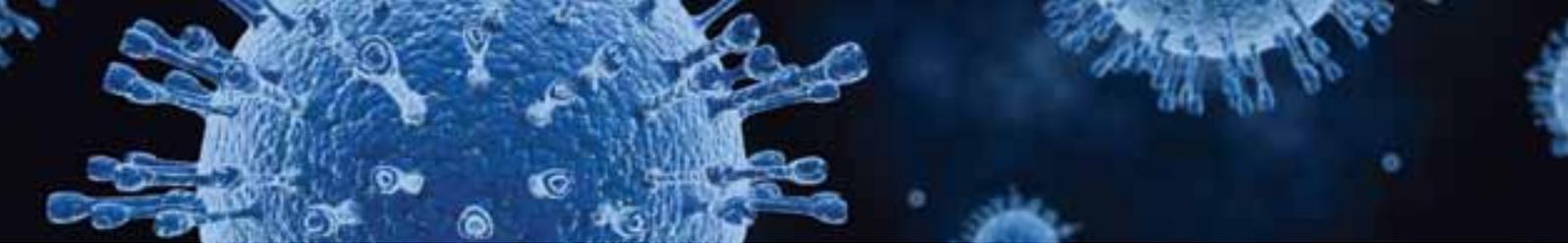
How vital is it for doctors to continue their education?

Medicine now is at a crossroads. There is a paradigm shift where everyone will soon be looking at an Anti-Aging, metabolic approach. It's a necessity for doctors to become involved in the training process because this is the medicine of the future, here today.

How could this type of training benefit the practice of the average physician?

We have in the fellowship program every single specialty represented—every single one of them. This kind of training—whether you're an ophthalmologist, a family practitioner or an ER doctor—it will benefit your patients. That's really the goal here: for patients to have the best care they can have.

Please contact your A4M Education Coordinator for more information on the Fellowship Programs
1-888-997-0112 | www.a4m.com



FELLOWSHIP IN INTEGRATIVE CANCER THERAPIES

Director: Mark Rosenberg, MD

Dr. Rosenberg is part of The Institute For Healthy Aging at the Sanctuary Medical Center.

Integrative Cancer Treatment is a unique therapy for treating individuals with cancer. The knowledge gained from attending this fellowship will allow practitioners to provide improved cancer treatments and utilize multiple modalities, including but not limited to, off-label pharmaceuticals, nutraceutical, vaccines and other types of immunotherapy, novel drugs/substances not yet approved in the U.S., dietary treatments, mind-body techniques, hyperthermia, homeopathy, in addition to traditional therapy.

How do you describe the content of the Fellowship in Integrative Cancer Therapies in your own words?

We get into the basics and biology of cancer, prevention, cutting-edge treatments from a natural as well as a conventional standpoint.

What do you hope to provide to participating doctors that they can't get elsewhere?

Conventional oncology training educates physicians regarding the three primary components of cancer treatment; surgery, radiation and chemotherapy. In this fellowship physicians will not only learn traditional diagnostic treatment modalities but also alternative, complimentary and state-of-the-art management techniques.

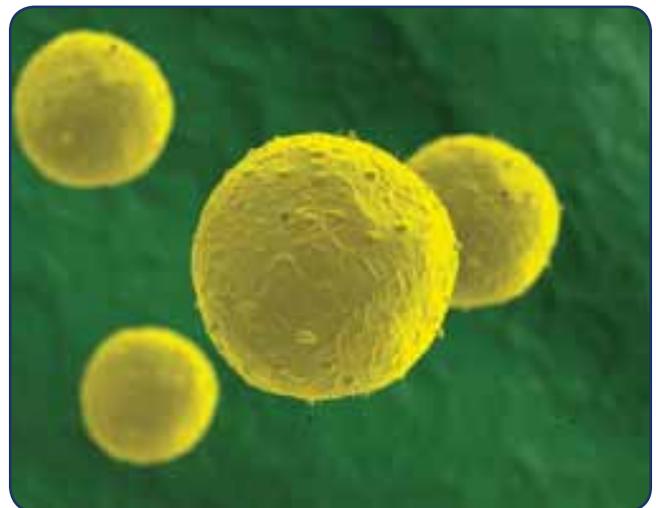
How vital is it for doctors to continue their education?

Medicine is in a great transition phase right now; so physicians must do their homework to keep pace with rapid change. Doctors who are treating cancer must continue to further their education until there is a cure.

How could this type of training benefit the practice of the average physician?

There is no better feeling than having a patient hug you and cry with you because her previously described terminal cancer is now stabilized or in remission.

The knowledge gained from attending this fellowship will allow practitioners to provide improved cancer treatments and utilize multiple modalities, including but not limited to, off-label pharmaceuticals, nutraceutical, vaccines and other types of immunotherapy, novel drugs/substances not yet approved in the U.S., dietary treatments, mind-body techniques, hyperthermia, homeopathy, in addition to traditional therapy.



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STEM CELL | FELLOWSHIP



STEM CELL THERAPIES FELLOWSHIP

Director: Dipnarine Maharaj, MD

Dr. Maharaj is the founder and medical director of the South Florida Bone Marrow/Stem Cell Transplant Institute.

In the Stem Cell Therapies Fellowship, you will learn how to treat the diseases associated with aging with stem cell therapies – the medicine of the future. A group of experienced clinicians presents a series of topics to cover the general principles and practice of stem cell biology and evidence-based treatments for physicians to optimize the health of their patients. After completion of this modular training program, physicians will be able to intelligently decide which stem cell protocols to recommend to their patients.

How do you describe the content of the Stem Cell Therapies Fellowship in your own words? It's a comprehensive education of the basics of stem cell science and how it applies to technology that we have for stem cell transplantation now and also the potential for the future.

What are some of the highlights? The focus of the fellowship has been to identify what are the cutting-edge areas of being able to translate from the lab into clinical practice. And the areas in which stem cells will be able to impact not only chronic diseases but all diseases and major disorders.

How vital is it for doctors to continue their education? It's critically important that physicians have a forum to be able to do that because the volume of information available from different resources is rising exponentially. They have to be able to attend our conference so it can be put into perspective for them.

How do you incorporate stem cell therapies into your patient care? I'm a bone marrow stem-cell transplant physician, since my early days in Scotland where I was a part of a pioneering program of stem cell transplantation. We have been able to utilize stem cells to repair the immune systems of patients with different types of blood disorders and cancers, including patients who

It's critically important that physicians have a forum to be able to do that because the volume of information available from different resources is rising exponentially.

have disorders of the immune system, and that is now a standard of care.

How could this type of training benefit the practice of the average physician? To really look at the diseases that they're treating and see how stem cell therapies and treatments and research that exists today could apply to the patients they are treating. Really it applies to all physicians because every single aspect of the body is affected by the regenerative potential of stem cells to heal the body.



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The Aesthetic

AntiAging Fellowship



AESTHETIC ANTI-AGING FELLOWSHIP

Sharon McQuillan, MD

Dr. McQuillan is the founder of The Ageless Aesthetic Institute.

The Aesthetic Anti-Aging Fellowship was created in recognition of the need to establish best practice standards in aesthetic medicine. The fellowship will enable medical professionals to learn the theory of aesthetic health and receive individualized, hands-on training in these procedures.

How do you describe the content of the Aesthetic Anti-Aging Fellowship in your own words?

We created the fellowship to create true understanding and expertise in doctors. That information comes from the three online modules. The remaining content comes from learning procedural expertise.

What are some of the highlights?

We're involved in research in the utilization of stem cells and platelet-rich plasma in aesthetic medicine, and we have developed proprietary techniques. So we have developed a stem cell isolation technique that allows our attendees to learn fat-transfer procedures as a much more successful procedure than fat transfer without stem cell augmentation.

How important is the interactive work?

Early in my training, a professor said to me, "You cannot learn how to ride a bike by watching someone ride a bike." And that is true.

What do you hope to provide to participating doctors that they can't get elsewhere?

The level of education that attendees receive is extensive, and we continually update the information. We make certain that our attendees are confident to offer their skill at the highest level. We're able to create pure expertise, and that is what patients want and deserve.

Also, we are aligned with the University of South Florida, and they provide our accreditation.

"These fellowships are such wonderful opportunities because the advances in functional, preventive and regenerative medicine and aesthetics have outpaced the ability for the traditional training systems to provide appropriate training. These education opportunities are extremely valuable for our attendees."—Dr. Sharon McQuillan

We are the only aesthetic training program that offers level-4 certification, which provides evidence that the attendee is confident to perform the procedure without supervision.

How vital is it for doctors to continue their education?

We're at an interesting time in medicine in that our knowledge is expanding at such a rapid rate that, unless you completed a residency yesterday, you are out of date unless you continually provide medical education for yourself. And, ultimately, our responsibility to patients is the best possible medical care.

How could this type of training benefit the practice of the average physician?

It's important to have a wider array of services because, as the population ages, successful practices will be the ones offering the full scope of Anti-Aging, functional medicine and aesthetics. Patients would prefer to go to one center that addresses all of those concerns.

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You're Invited

We invite you to join us for cocktails and hors d'oeuvres at

The
NETWORKING
Reception

Join Like-Minded,
Anti-Aging Professionals
for This Networking
Opportunity!

Thursday, May 17, 2012
6:00 pm – 7:30 pm
Exhibit Hall

CALL FOR MORE INFORMATION:

www.A4M.com
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