

WELCOME TO Las Vegas



THE FUTURE OF MEDICINE TODAY

Distinguished Colleagues,

The American Academy of Anti-Aging Medicine (A4M) welcomes you to Las Vegas for the Winter 2013 session of the 21st Annual World Congress on Anti-Aging, Regenerative & Aesthetic Medicine, certification programs, workshops, and A4M Fellowships.

The A4M is proud that our 26,000 physicians, practitioners, and scientific members from 120 nations worldwide have made a lasting and palpable commitment that expands the availability of advanced biotechnologies and leading-edge preventive healthcare throughout the world. Now in its third decade of educational service, the A4M's scientific educational programs have trained over 100,000 medical professionals worldwide.

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

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

With warm regards,



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



Robert Goldman, M.D., Ph.D., D.O., FAASP
Chairman, A4M

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THE FUTURE OF MEDICINE TODAY

21st Annual World Congress on Anti-Aging, Regenerative & Aesthetic Medicine

Table of Contents

- General Information 4
- Conference Bonuses 5
- Special Guests 8
- Schedule at-a-Glance 10
- Sponsored Workshops 18
- Product Theater Schedule 20
- Mitochondria Resuscitation & Genetic Expression: The Key To Healing Every Disease?
Chris D. Meletis, N.D. 25
- Some Effects of Food Intolerance on Athletic Performance & Weight
Roger Deutsch 37
- Propitious Modulation of Fibroblast Quiescence Mediated by Cytokine Expression
Mediated from Embryonic Stem Cells
*Ron Shane, Ph.D., OMD; Andrew Crain; Andrei Golovko; Benjamin Morpurgo;
Alexa Dillberger; Taylore Bonn; Amber Buz-Zard; Evan Snyder* 46
- A New Approach to Telomere Measurement
Dr. Marcelo Viegas, PhD 55
- The Next Generation of Omega-3 Anti-Aging Supplements
Sharon McQuillan, MD 66
- Product Announcements 75
- Exhibit Floorplan 94
- Exhibitor Index 95
- Exhibitor Listings 99
- Advertising Index 163

TABLE of contents



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THE FUTURE OF MEDICINE TODAY

21st Annual World Congress on Anti-Aging, Regenerative & Aesthetic Medicine
December 12 -15, 2013 • Las Vegas, NV

General Information

GENERAL Information

BADGES:

The official registration badge should be prominently displayed at all times for admission into the meeting rooms and Exhibit Hall, during show hours and official events.

SHOW REGULATIONS:

1. An official picture ID is required to pick up name badge.
2. Name badge must be worn at all times during show hours and official events.
3. No one under the age of 18 will be admitted into the Exhibit Hall.
4. This event is open to healthcare professionals and their invited guests only.
5. Photography is permitted in the Exhibit Hall only by press, with the prior permission of show management, and prior permission from the firm(s) whose booth(s) are being photographed. Videography in the Exhibit Hall (motion video, film, or digital) is permitted only at discretion and with advance permission of show management and the prior permission of individual(s) and/or firm being photographed.
6. Audio-recording (tape or digital) of this event is expressly prohibited.
7. Unauthorized solicitation is prohibited. Solicitation of business on the event premises by anyone other than official exhibitors is strictly forbidden. Solicitation of business in, and in the immediate proximity of the event is extremely prohibited. In addition, non-A4M and/or commercial literature of any kind may not be distributed near or at the event registration area and all event-related general public areas.
8. No medical procedures are permitted to take place in Exhibit Hall booths without prior permission of show management.
9. Smoking is prohibited on the exhibit floor and in all meeting rooms.
10. Show management reserves the right to expel any individual and/or company without recourse or refund at its sole discretion.
11. **PHOTO RELEASE:** Anyone in attendance at any A4M event may be subject to any photography or videography conducted during the event which may be used in any form for future media purposes. Please contact A4M with any concerns.



THE FUTURE OF MEDICINE TODAY

21st Annual World Congress on Anti-Aging, Regenerative & Aesthetic Medicine
December 12 -15, 2013 • Las Vegas, NV

Conference Bonuses

- **The A4M Conference App** (for smart-phones)

Keep the event in the palm of your hand! Scan a QR code or download the app directly from a link and utilize A4M's integrative conference app to help boost your experience and navigate your conference time wisely. Dedicated web page coming soon at www.A4M.com.

- **Evening Workshops**

Thursday, December 12th • Friday, December 13th

Attend free evening workshops featuring highlighted speakers discussing the latest advancements in regenerative medicine (non CME)

- **Eye Opener Giveaways**

The Early Bird Gets the Worm! The first to arrive at select morning sessions will receive special prizes

- **Exhibit Hall**

Friday, December 13th 11:00am - 6:00pm • Saturday, December 14th 11:00am - 7:00pm • Sunday, December 15th 11:00am - 2:00pm

Visit our premier exhibit hall showcasing over 250 booths

- **New Product Theater**

Friday, December 13th 11:00am - 1:00pm; 4:00pm - 6:00pm • Saturday, December 14th 11:00am - 1:00pm.

Visit the New Product Theater where companies will promote their products and services via a main-stage demonstration or presentation in the exhibit hall

- **Networking Reception**

Saturday, December 14th 5:00pm-7:00pm.

Mix and mingle with other distinguished medical professionals and exhibitors

- **Fellowship Graduation**

Saturday, December 14th

Congratulations to the Fellowship class of 2013!

- **Camaro Giveaway**

Play the Exhibit Hall game and enter for your chance to win a FREE car! **Drawing will take place Sunday, December 15th at noon.**

- **A4M Bookstore**

Come browse our extensive selection of educational literature featuring current research and technological advances; visit with an A4M Educational Coordinator to discuss the literature as well as educational opportunities available

Quality in compounding.

PCCA carries the torch.

We know that quality compounding starts with quality chemicals. That's why we conduct 14 tests and analyses on every ingredient and reject almost 200 chemical lots each year. But it doesn't stop there. It also means providing the industry's best education and training programs (over 115 in 2013) combined with more consulting pharmacists on-hand than anyone else and access to more than 8,000 formulas.

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21st Annual World Congress on Anti-Aging, Regenerative & Aesthetic Medicine

Special Guests



Arnold Schwarzenegger

Governor Arnold Schwarzenegger will receive the **Infinity Award**, the highest honor bestowed by the A4M. A4M presents this award to Gov. Schwarzenegger to recognize his early leadership role in securing landmark funding in the area of stem cell research, piloting an initiative to direct \$3 billion in government funds to the field. In addition, the **Infinity Award** recognizes Gov. Schwarzenegger's lifetime accomplishments as a champion bodybuilder, Hollywood action hero, successful businessman, environmentalist, philanthropist, best-selling author, and California's 38th Governor.



Judith Reichman, MD

As an outspoken expert on women's healthcare issues, Dr. Reichman balances the demands of a thriving Los Angeles practice in gynecology, infertility, and menopause. She is the author of several books and is best known for her two-part series on PBS, "Straight Talk on Menopause with Dr. Judith Reichman".



Suzanne Somers

Suzanne Somers is a dedicated and an informed health advocate and has become the face and voice of alternative and integrative medicine. With her New York Times bestselling books, Suzanne singlehandedly has achieved what the movement has wanted to accomplish for many years. She has increased awareness regarding the benefits of BHRT, Anti-Aging Medicine, stem-cell therapies, chemo-free cancer cures, and the often-overlooked causes of obesity and how to restore our bodies.



Travis Stork, MD

ER physician and host of the Emmy Award-Winning talk show, "The Doctors," Dr. Stork is a fervent believer in helping patients feel empowered when it comes to their health. He is passionate about enlightening individuals on simple ways to attain and maintain good health based on their own life circumstances. He takes pride in teaching people how to prevent illness before it happens.



THE FUTURE OF MEDICINE TODAY

21st Annual World Congress on Anti-Aging, Regenerative & Aesthetic Medicine Maximize your Experience with a Specialty Workshop

Thursday, December 12, 2013



Testosterone Deficiency and Therapy in Men Presented by Abraham Morgentaler, MD, FACS

This workshop will provide an in-depth educational opportunity regarding the exciting and rapidly evolving field of testosterone deficiency (hypogonadism) in men. Presented by a pioneer in the field, Dr. Morgentaler is one of the first, in the US, to provide testosterone therapy to otherwise healthy men who developed age-related declines in testosterone, and performed the research that altered the conventional wisdom that testosterone was risky for prostate cancer.



Personalized Lifestyle Medicine Presented by Pamela W. Smith, MD, MPH, MS; Mark Houston, MD; Mark Rosenberg, MD; Tana Amen, RN

Personalized medicine stands poised to transform healthcare over the next several decades and also offers the possibility improved health outcomes and has the potential to make healthcare more cost-effective. This workshop will include patient case studies, information on hormones, cardio health and more. The workshop will demonstrate practical applications and how physicians can implement into their own practice.



Pellet Therapy Presented by James Mahoney, DO

Bio-Identical Hormone Therapy has become a central pillar of Anti-Aging and Age Management Medicine. Subcutaneous pellets are in great demand due to their obvious impact on negative symptoms associated with aging as well as dramatic enhancement in sexual, cognitive, and athletic function. Simple, affordable treatment can be accomplished in less than 10 minutes, 3 – 4 times per year with guaranteed compliance to create benefits unavailable with any other form of therapy. Expert training in all aspects of pellet therapy is critical to success for patients and health professionals.



Practice Management Certification: Advancing the Business of MEDICINE Presented by Jay Shorr BA, MBM-C and Expert Faculty

This is a 2-part certification program, with part 1 offered in Last Vegas; Part 2 in Orlando 2014.

Healthcare is changing – quicker than we think! Too many practices are still on the sidelines, trying to understand new regulations, compliance, technology – and so much more. The broad responsibilities and detailed requirements of managing a revenue cycle, compliance regulations, human resources, health information and general business processes make a Practice Management Certification a MUST. Discover the keys that change your practice.



Advances in Body Contouring Presented by Sharon McQuillan, MD and Expert Faculty

The market for body shaping procedures has experienced tremendous growth as patients' desire youthful body contours to match their youthful facial enhancements, as well as their youthful energy levels as result of Bio-Identical Hormone Replacement. It is predicted that body contour procedure volume will increase 12.8% annually to more than 7.4 million procedures by 2015.

Body contouring treatment modalities include mesotherapy, non-invasive laser and light devices for skin tightening and cellulite reduction, laser lipolysis, and surgical methods and devices for fat and cellulite. This workshop will provide a substantial update regarding the latest advances available to contour the body, reduce inches, and reduce cellulite for experienced as well as novice aesthetic practitioners.

Sunday, December 15, 2013



Menopause/Andropause: Improving the Health and Happiness of your Patients with Bio-Identical Hormones Presented by: Jonathan V. Wright, MD and Daved Rosensweet, MD

Come learn from these pioneer holistic physicians and enhance the lives of your patients in this rewarding world. This 1-day intensive workshop is designed to establish or augment your "Monday Morning" effectiveness in understanding, implementing and modulating bio-identical hormone treatment programs in medical practice. The presenters lay a foundation of basic information, as well as delve into advanced clinical complexities and subtleties. This course will assist the beginner as well as enrich the seasoned practitioner. Doctors Wright and Rosensweet also devote time to address your specific submitted questions, cases and challenges.



THE FUTURE OF MEDICINE TODAY

Schedule-At-A-Glance

21st Annual World Congress on Anti-Aging, Regenerative & Aesthetic Medicine

Thursday, December 12, 2013 Specialty Workshops

		Presented By:
9:00am-5:00pm	Testosterone Deficiency & Therapy in Men	Abraham Morgentaler, MD, FACS
7:30am-6:00pm	Personalized Lifestyle Medicine	Pamela W. Smith, MD, MPH, MS; Mark Houston, MD Mark Rosenberg, MD & Tana Amen, BSN, RN
9:00am-5:00pm	Pellet Therapy	James Mahoney, DO
8:00am-5:00pm	Practice Management Certification	Jay Shorr, BA, CMBM,CAC, Course Director Including Expert Faculty
8:00am-6:00pm	Advances in Body Contouring	Sharon McQuillan, MD

Sunday, December 15, 2013

		Presented By:
8:00am-6:00pm	Menopause/Andropause: Improving the Health & Happiness of your Patients with Bio-Identical Hormones	Jonathan V. Wright, MD & Daved Rosensweet, MD

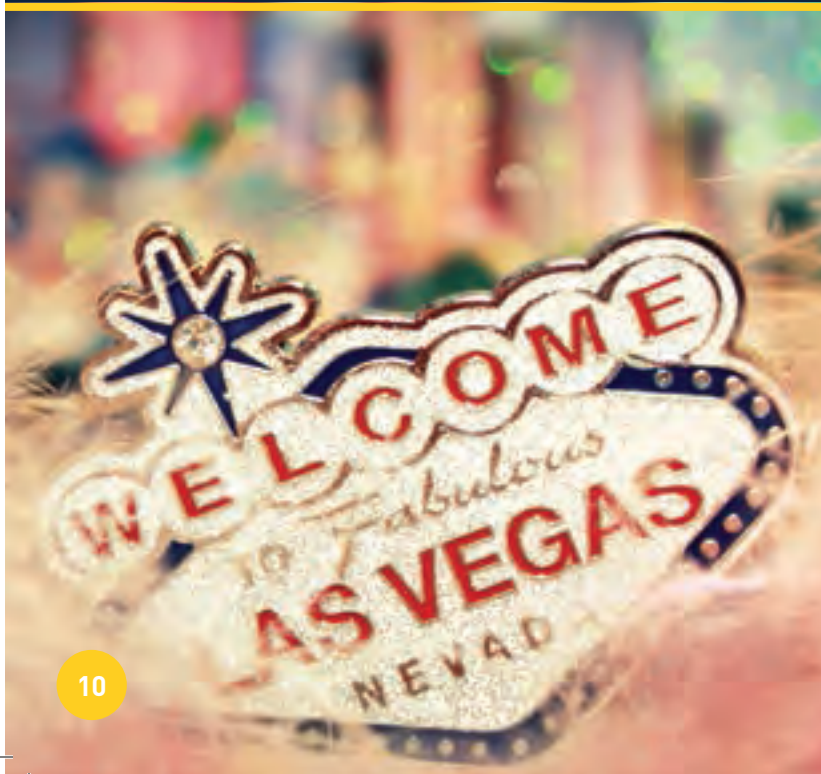
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ESTABLISHED 1992, A4M REPRESENTS 26,000 PHYSICIANS & SCIENTISTS FROM 120 COUNTRIES WORLDWIDE

22nd Annual World Congress on Anti-Aging, Regenerative & Aesthetic Medicine



SAVE THE DATE

December

10-13, 2014

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Schedule-At-A-Glance

21st Annual World Congress on Anti-Aging, Regenerative & Aesthetic Medicine

Friday, December 13, 2013 Conference

• Morning General Session •		Presented By:
7:00am-7:40am	The Use of T3, Botanicals & Nutrition to Optimize Thyroid Function, Peripheral T4 to T3 Conversion & Well-Being	E. Denis Wilson, MD
7:40am-8:20am	New & Revolutionary Concepts in the Diagnosis & Treatment of Hypertension	Mark Houston, MD, MS, ABAARM, FACP, FAHA, FASH
8:20am-8:40am	Opening Remarks (Non-CME)	Ronald Klatz, MD, DO & Robert Goldman, MD, PhD, DO
8:40am-9:20am	Testosterone Therapy in Men: Panacea, Scourge, or the Next Big Thing In Medicine?	Abraham Morgentaler, MD, FACS
9:20am-10:10am	Slow Your Clock Down: On-Label, Off-Label, Grey-Label	Judith Reichman, MD
10:10am-11:00am	Our Time has Come (Non-CME)	Suzanne Somers
LUNCH (On your own) • 11:00am-1:00pm		
Exhibit Hall Hours • 11:00am-6:00pm		
• Session 1-The Nuts & Bolts of Hormones •		Presented By:
1:00pm-2:00pm	Introduction to Bio-Identical Hormone Deficiencies	Thierry Hertoghe, MD
2:00pm-2:45pm	Testosterone-Low Testosterone or Andropause in Men	Ronald Rothenberg, MD
2:45pm-3:00pm	Hypothyroidism	Ronald Rothenberg, MD
3:00pm-4:00pm	Estrogens, Progesterone, Testosterone for Women	Jennifer Landa, MD, OB/GYN
• Session 2-Health of the Peri/Menopausal Woman •		Presented By:
1:00pm-4:00pm	A Comprehensive Approach to Common Clinical Conditions in the Peri/Menopausal Woman	Filomena Trindade, MD
• Session 3-New Hot Health Topics •		Presented By:
1:00pm-1:45pm	A Clinical Application to Hormone Testing & Nutrition	Jim LaValle, RPh, MS, CCN
1:45pm-2:30pm	Hormone Replacement Therapy and the Cancer Profile	EK Schandl, MD, PhD
2:30pm-3:15pm	Pain Management with Transdermal Technology	Gus Bassani, PharmD
3:15pm-4:00pm	Immunotherapy: A Physician's Scientific Guide to Cow Colostrum & Its Uses	Andrew Keech, PhD, DSc
• Session 4-Intergrative Age Management Health Topics •		Presented By:
1:00pm-1:45pm	The Development of Mitochondria-Targeted Therapies for Age Associated Diseases	Michael Murphy, PhD
1:45pm-2:30pm	Aging and the Short Telomere Connection	William Andrews, PhD
2:30pm-3:15pm	Rehabilitation of Age-Related Pulmonary Function Decline	James Lineback, MD
3:15pm-4:00pm	More than just Rickets: The Newly Rediscovered Anti-Aging Properties of the Amazing Sunshine Vitamin D	Mayer Eisenstein, MD, JD, MPH
• Session 5-Advances in Aesthetic Medicine •		Presented By:
1:00pm-1:40pm	The Unifying Theory on Beauty	Steven Dayan, MD, FACS
1:40pm-2:20pm	Groundbreaking New Information on Delaying Skin Aging	Patrick Bitter, MD
2:20pm-3:00pm	Cosmetic Applications of Platelet Rich Plasma	Robert Bowen, MD
3:00pm-3:30pm	Clinical & Aesthetic Innovations: Low Level Laser Therapy	Mark Nestor, MD, PhD
3:30pm-4:00pm	Managing & Marketing your Cosmetic Practice in Today's Economy	Jay A. Shorr, BA, MBM-C

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Schedule-At-A-Glance

21st Annual World Congress on Anti-Aging, Regenerative & Aesthetic Medicine

Saturday, December 14, 2013 Conference

• Morning General Session •		Presented By:
7:00am-7:30am	Detoxify for Life	John Cline, MD
7:30am-8:00am	Pellet Therapy: Symptoms & Survival	James Mahoney, MD
8:00am-8:30am	Healing the Mind With Food	Tana Amen, BSN, RN
8:30am-9:00am	Anti-Aging Benefits of Novel Marine Oil (Omega 3) From New Zealand Green-Lipped Mussel	Sharon McQuillan, MD
9:00am-9:35am	Should you Allow your Child to Play Football? Update on Concussion Management, Prevention and Treatment	Joseph Maroon, MD
9:35am-10:15am	Telomeres, Aging and Disease	Mark Rosenberg, MD
10:15am-11:00am	Your Best Life	Travis Stork, MD
LUNCH (On your own) • 11:00 am – 1:00 pm		
Exhibit Hall Hours • 11:00 am – 7:00 pm		
• Session 1 – The Nuts & Bolts of Hormones •		Presented By:
1:00pm-2:00pm	The Importance of BHRT on the Cardiovascular System	Thierry Hertoghe, MD
2:00pm-2:40pm	Neurotransmitters & BHRT Effects on the Brain & How they are Used in Treating Psychological Disorders	Eric Braverman, MD
2:40pm-3:00pm	Update on State's and Congress's new laws regarding compounded prescription drugs	John Graseola, RPh
3:30pm-4:00pm	How to prescribe Anti-Aging medicine. The nuts and bolts for optimal hormone replacement therapy. Labs, algorithms, follow up testing.	Ronald Rothenberg, MD
• Session 2-Advances in Stem Cell Therapies •		Presented By:
1:00pm-1:45pm	Cutting Edge Concepts for the Regenerative Medicine Physician in the Use of Stem Cell & PRP Injections	Joseph Purita, MD
1:45pm-2:30pm	The Role of Nitric Oxide in Cellular Aging: Telomeres, Mitochondria & Stem Cells	Nathan Bryan, PhD
2:30pm-3:15pm	Could Metabolic Syndrome, Lipodystrophy, & Aging be Mesenchymal Stem Cell Exhaustion Syndromes?	Eduardo Mansilla, MD
3:15pm-4:00pm	Current Review of the Role of Stem Cell Banking in Preventive Medicine	Terry Grossman, MD
• Session 3-Clinical Case Studies–The Male/Female Patient •		Presented By:
1:00pm-1:45pm	Resolving Hashimoto's Thyroiditis	Alan Christianson, MD
1:45pm-2:30pm	Physiologic Dosing & Laboratory Findings for Women on BHT	Paul Savage, MD
2:30pm-3:15pm	Man Boob Nation: An Integrative/Solutions Based Strategy to Low Testosterone	Nathan Goodyear, MD
3:15pm-4:00pm	Interactive Patient Case Reports (Non-CME)	David Zava, PhD
• Session 4-Advances in Aesthetic Medicine •		Presented By:
1:00pm-1:40pm	Toxin Complications	Edward Zimmerman, MD
1:40pm-2:20pm	Clinical Signs of Aging Skin Facial Wrinkles: Aesthetic Role of Equol Isomers for Aging Skin	Edwin Lephart, PhD
2:20pm-3:00pm	Three Dimensional RF Technology Utilization-A New Paradigm for Face & Neck Rejuvenation	Daniel Man, MD
3:00pm-3:30pm	Incorporation of Men into Your Aesthetics Practice	Keith Jeffords, MD, DDS
3:30pm-4:00pm	Business Start-Up for Spa & Medical-Spa	Manon Pilon
• Afternoon General Session •		Presented By:
4:00pm-5:00pm	Infinity Award Recipient (Non-CME)	Governor Arnold Schwarzenegger

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Schedule-At-A-Glance

21st Annual World Congress on Anti-Aging, Regenerative & Aesthetic Medicine

Sunday, December 15, 2013 Conference

• Morning General Session •

Presented By:

7:00am-7:30am	Genetic Susceptibility & Targeted Interventions for Genetic Integrity, Obesity, Diabetes, Lipid Dysregulation & Inflammatory Pathways	Chris Meletis, ND
7:30am-8:00am	Regenerative Techniques for Non-Surgical Joint & Spine Repair	Peter Fields, MD, DC
8:00am-8:30am	Learning How to Talk to Patients	Erika Schwartz, MD
8:30am-9:10am	Leveraging Lifestyle Medicine	Thomas Williams, PhD
9:10am-9:40am	Stem Cells: Understanding Aging as a Platform to Enhance Tissue Regeneration	Massimo Dominici, MD
9:40am-10:15am	How to Maintain Memory at Any Age	Pamela Smith, MD, MPH, MS
10:15am-11:00am	Pharmaceutical Approaches to Treating Aging: The Sirtris Story	Christoph Westphal, MD, PhD

LUNCH (On your own) • 11:00 am – 1:00 pm

Exhibit Hall Hours • 11:00 am – 2:00 pm

• Session 1 – The Health of the Aging Brain •

Presented By:

1:00pm-1:45pm	Brain Fitness: Every Little Aging Part of the Brain & Repairing It	Eric Braverman, MD
1:45pm-2:30pm	Cognitive Function in Middle Age	Thomas F. Collura, PhD, MSMHC, BCN, NCC & Christen Stahl, MA, NCC
2:30pm-3:15pm	Prevention & Treatment for the Aging Brain	Andrew Campbell, MD
3:15pm-4:00pm	Integrating Neuroscience Methods into Peak Performance & A Functional Medicine Practice	David Hagedorn, PhD & Nicole Hagedorn, DO

• Session 2-Clinical Case Studies •

Presented By:

1:00pm-1:40pm	Adrenal Slumber: Measuring & Managing the Consequences of Adrenal Insufficiencies on Sleep Pathology	Kelly Olson, PhD
1:40pm-2:20pm	Simplifying The Complex Patient	Deborah Matthew, MD
2:20pm-3:00pm	Weight Loss Utilizing Food Sensitivity Testing, Elimination & Treatment of the Gut: A Case Study	Shelena C. Lalji, MD, FACOG
3:00pm-3:30pm	Avoiding Pitfalls & Picking the Right Lab Test	Mark Newman, MS
3:30pm-4:00pm	The Vitamin Conundrum	Theodore Piliszek, MD

• Session 3-Advances in Aesthetic Medicine •

Presented By:

1:00pm-1:45pm	The Revolutionary Microcannula Injection Technique	Anil Rajani, MD
1:45pm-2:30pm	Orthostatic Liposuction: A Must for Abdominal Contouring	John Hamel, MD
2:30pm-3:15pm	Successful Treatment of Acne Vulgaris with Probiotics	Sharon McQuillan, MD

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- **Step One**
You will receive an email with your CME login and password for the CME evaluation site, approximately 5 days after the conclusion of the event.
- **Step Two**
Visit www.A4MCME.com
- **Step Three**
Login with your assigned CME number and password from the email. (Refer to step 1)
- **Step Four**
Fill in your Personal Details. Continue.
- **Step Five**
Select the Conference to be evaluated and fill in all of the appropriate evaluations for each session you attended only.
- **Step Six**
You will receive notification of acknowledgement immediately on the screen after completion of the evaluation. Please log out and log back in, go to the conference's evaluation screen to print or save your certificate. You will see a yellow banner at the top of the screen that says Print Now.

Save CME number and password for future CME evaluations.

Online modules, FAARM Module V and Module XXIII are NOT awarded CME credit.

**If you need any assistance please contact A4M at
888-997-0112 ext. 7575 or CME@A4M.com**



THE FUTURE OF MEDICINE TODAY

Mix & Mingle at the Networking Reception

Date: Saturday, December 14th
Time: 5:00 pm – 7:00 pm

Location: Exhibit Hall

Network with other distinguished Medical Professionals and learn about the latest products and services from our exhibitors

CAMARO GIVEAWAY



CAMARO®



CELEBRATING 21 YEARS
OF INNOVATION IN ANTI-AGING, REGENERATIVE & AESTHETIC MEDICINE

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Camaro

2nd PRIZE
Clearlight Infrared Sauna
Premier IS-2 or (Royal IS-2)*
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3rd PRIZE
Set of A Fashion Hayvin
Jewelry Bracelets
\$1500 Value

How to Enter:

1. Visit The Booths Of Each Camaro Sponsor

The sponsors and their booth numbers are shown on the entry form. Just ask each sponsor to individually stamp your passport in their logo square, complete your contact details and deposit your entry card at the Camaro Booth # 5063.

2. You Must Be A Registered Conference Delegate

to the 21st Annual World Congress on Anti-Aging, Regenerative & Aesthetic Medicine. Exhibitors, sponsors and their staff are not eligible to win. YOU MUST BE PRESENT TO WIN.

3. The Camaro Giveaway Will Be Held On

Sunday, December 15th at 12:00pm in the Exhibit Hall.

4. A W-9 Tax Form Must Be Filled Out

prior to prize collection.

DRAWING
EXHIBIT HALL
DECEMBER 15th 12:00PM



Booth #7040



Booth #4047



Booth #7031



Booth #8012



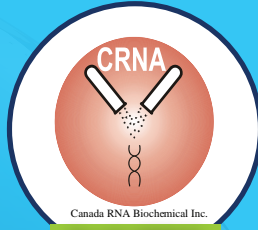
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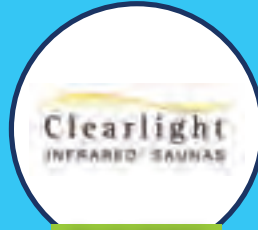
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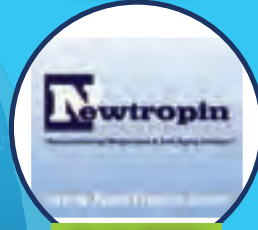
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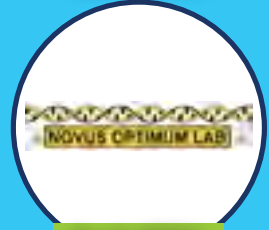
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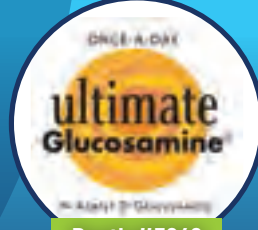
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Thursday, December 12th

6:15PM – 8:30PM **Topic:** Advanced Biomarker Testing: Case Based Application
Presented by: Dr. Tara Dell
Location: Marco Polo
Sponsored by: Health Diagnostic Laboratory, Inc.



6:15PM – 8:30PM **Topic:** The Most Potent Natural Solution for Pain & Inflammation
Presented by: Sharon McQuillian, MD
Location: Casanova
Sponsored by: OMAPREM



6:15PM – 8:30PM **Topic:** Reverse Skin Aging with Cosmeceuticals and Hormones
Presented by: Pam Smith, MD; Rick Rhodes, Pharm.D
Location: Marco Polo
Sponsored by: University Compounding Pharmacy



Friday, December 13th

6:30PM – 8:30PM **Topic:** Regenerate Your Practice: Validated In-Office Stem Cell Therapy
Presented by: Sharon McQuillian, MD
Location: Bassano
Sponsored by: Ageless Regenerative Institute



6:30PM – 8:30PM **Topic:** Exclusive Physicians Workshop
Presented by: BodyLogicMD, Inc.
Location: Veronese
Sponsored by: BodyLogicMD



6:30PM – 8:30PM **Topic:** Cellular Detoxification
Presented by: Silvia Binder, ND, PhD; John Cline, MD; Mark Armstrong, OMD, AD, AC
Location: Bassano
Sponsored by: Ondamed



6:30PM – 8:30PM **Topic:** Breaking the Aging Code of the Brain and Body
Presented by: Eric Braverman, MD
Location: Titan
Sponsored by: PATH Medical



6:30PM – 8:30PM **Topic:** Solving Adrenal Fatigue
Presented by: Sangeeta Pati, MD
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Friday, December 13th


11:00 am **Topic:** A Neuroendocrine Model for Healthy Aging: Analytical Precision Directing Targeted Therapeutic Options
Presented by: Sharon Norling, MD, MBA, Board-certified OB/GYN, Integrative/Holistic Medicine (ABIHM)
Sponsored by: Sanesco Health 

11:30 am **Topic:** Learn how to reduce monthly payments by as much as \$8,500.00 a month while increasing profits & collections with Universal Healthcare Lending's Comprehensive Financing Solutions.
Presented by: Steve Miller
Sponsored by: Universal Healthcare Lending 


12:00 pm **Topic:** Advanced Facial & Body Fractionated Co2 Laser Therapy
Presented by: Gregory W. Chernoff, M.D. F.R.C.S
Sponsored by: Cutting-Edge Aesthetics 

12:30 pm **Topic:** Experiences with InBody
Presented by: Thomas W. Storer, PhD
Sponsored by: BioSpace 

4:00 pm **Topic:** Implement Medical Weight Loss into Your Practice to Keep Your Patients Healthy & Your Business Growing
Presented by: Dr. Kimball Lundahl, DC, ND President/CEO
Sponsored by: Healthy Habits Management Co. 

4:30 pm **Topic:** The Latest in Website Design & Marketing Technology
Presented by: Patricia Storey, RPh, FACA
Sponsored by: Storey Marketing 

5:00 pm **Topic:** ONCOblot test, the first universal test that detects cancer early and discriminates among different tissues of origin
Presented by: Dr. Claudia Hanau, N.D.
Sponsored by: Mornuco 

5:30 pm **Topic:** Natural anti-inflammatory compounds comprising omega-3 fatty acids, tocopherols & tocotrienols, coenzyme Q10, vitamin D3 & carotenoids in the prevention & management of hyperlipidemia, cardiovascular disease & hypertension
Presented by: Dr. Andreas Papas
Sponsored by: A.C. Grace 

Saturday, December 14th

11:00 am **Topic:** Antibiotics are Killing Us – Center for Disease Control
Presented by: Doug Wyatt, Founder of the Center for Nutritional Research
Sponsored by: Sovereign Laboratories 

11:30 am **Topic:** Discovery of negatively charged water droplets of water
Presented by: Jim Kaszyk, Bachelor of Chemistry, Developer of The Ageless Secret
Sponsored by: The Ageless Secret 

12:00 pm **Topic:** The Discipline of Mixed Martial Arts
-1:00 pm **Presented by:** Master Alan Goldberg; Silvio "Mighty Dragon" Simac; Michael Jai White; Cynthia Rothrock 

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• Advanced Metabolic Endocrinology Certification •

This program will discuss various areas of hormonal imbalances in men and women including advanced topics in adrenal health, breast health, estrogen metabolism, late life hypogonadism and prostate cancer.

Courses: Fellowship Module I, A – D

Module A	Functional overview of hormonal changes that manifest in men and women with aging
Module B	Advanced Hormonal Prescribing and Herbal Therapies for Women
Module C	Advanced Insulin Therapies and Hormones/Botanicals and Pregnancy
Module D	Men's Health and Advanced Compounding for Hormonal Treatments

• Weight Management Certification - Only available online •

In this program you will learn the unique relationship between adrenal dysfunction stress with hormonal imbalance and weight gain plus weight loss plateaus. Participants will recognize and understand the prevalence of adult obesity and the risk factors involved and understand the bio-chemistry of how the body breaks down proteins, fats and carbohydrates.

Courses: Fellowship Module XIV, A – D

Module A	The complex causes of weight gain
Module B	Risk factors of obesity and digestive issues in applying weight loss therapies
Module C	This module explores the body's immediate and cumulative responses to exercise intervention programs
Module D	The goal of "Getting Healthy Together"

• Brain Fitness Certification - Only available online •

One of the major medical issues that will affect all of our patients is how to maintain memory throughout their life. The Brain Fitness Modules (Module XV: A-D) are a group of four modules that will give you new skills to help patients prevent memory loss along with treat patients who already have cognitive decline.

Courses: Fellowship Module XV, A – D

Module A	The Basics of Brain Fitness and Memory Maintenance
Module B	How the Brain Learns and Metabolism of the Brain
Module C	Memory Loss: A Practical Guide for Clinicians
Module D	Brain Fitness Therapies



THE FUTURE OF MEDICINE TODAY



Certification Programs



• Metabolic Cardiovascular Certification •

The modules will start with basic teaching of vascular biology concepts and how this relates to vascular disease. Vascular aging pathophysiology, diagnosis, prevention and treatment will be reviewed as well. Dyslipidemia will be defined based on new basic science and clinical research. Inflammation, oxidative stress, the role for expanded lipid profiles using LDL and HDL particle size and number are reviewed in the context of the pathophysiology of vascular damage. Proper analysis of CV risk factors, mediators and CV risk scoring will be taught using the COSEHC risk analysis methods. Review of CVD labs and noninvasive CV tests will be reviewed. The role of heavy metals in CVD will be extensively reviewed based on a functional medicine model.

Courses: Fellowship Module XVI, A – D

Module A	How to apply nutrition, exercise and weight management programs related to vascular biology
Module B	Components of CardioVascular Disease
Module C	Nutritional and dietary therapies for prevention and treatment of cardiovascular disease
Module D	Various conditions in cardiovascular disease

• Sports Medicine Certification •

This certification program focuses on the “science of eating,” including diet programs, recipes and nutrients that help athletes reach peak performance and success. Factors that hinder such success are also reviewed. The program discusses the body’s physiological response to exercise, treatments for sports-related conditions, biometrics, eating disorders, the aging athlete and psychology.

Courses: Fellowship Module XIX, A – D

Module A	The Body’s Physiological Response to Exercise
Module B	Treatments for Sports-Related Conditions
Module C	Biometrics and Eating Disorders
Module D	The Aging Athlete and Psychology

• Lifestyle Coaching Certification •

The Certification in Lifestyle Coaching teaches the healthcare practitioner how to properly administer information that will positively impact clinical outcomes and improve the overall standard of care. The client needs to live a healthy lifestyle and this course teaches you how to teach your client the proper steps. It’s about setting up your clients to succeed.

Courses: Fellowship Module XXIII, A – D

Module A	Wellness Revolution and how you can become a part of the solution
Module B	Basic Nutrition
Module C	Fundamentals of a co-active coaching model
Module D	Counseling the Patient and Improving Energy

• Certification Requirements •

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Completion of corresponding USF Courses
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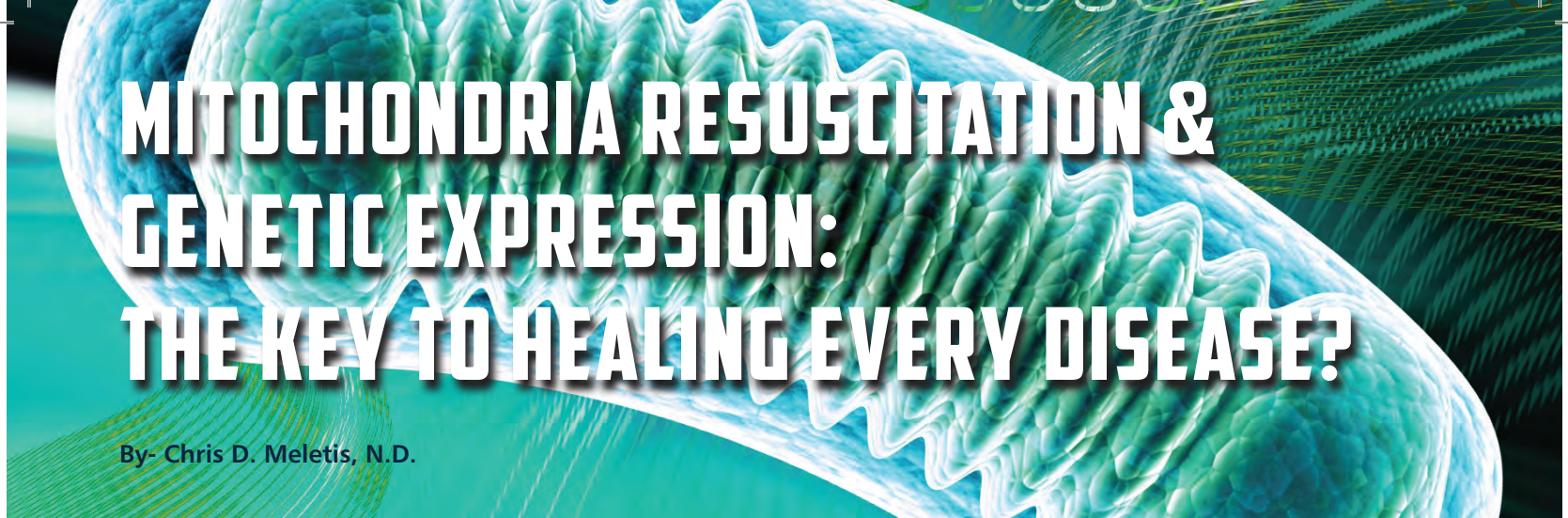
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MITOCHONDRIA RESUSCITATION & GENETIC EXPRESSION: THE KEY TO HEALING EVERY DISEASE?

By- Chris D. Meletis, N.D.

Takeaway Points

- Cellular function is dependent upon sufficient mitochondrial performance.
- In mitochondrial DNA (mtDNA), oxidative damage occurs at a rate five to 10 times higher than in nuclear DNA.
- No matter what disease you're treating in a given patient, the chances are good that mitochondrial dysfunction has played a critical role.
- Genetics, sleep apnea, aging, environmental toxins and certain drugs can all harm mitochondrial function.
- Lifestyle modifications as well as certain supplements such as L-carnitine, CoQ10 and D-ribose can support mitochondria health.

The human body is comprised of approximately 50 to 75 trillion cells, and each requires sufficient fuel at the mitochondrial level to manifest their genetically programmed functions. These functions depending on the cell location may manifest as proper thyroid, adrenal, pituitary, hypothalamic function or something as relatively mundane as wound healing, hair growth, or ocular health. Mitochondria are intracellular organelles present in all mammalian cells, except red blood cells. Each cell is stuffed with several hundred to more than a thousand mitochondria.

Simply put, mitochondria are the batteries of the cells because they synthesize the universal energy molecule ATP. They accomplish this through electron transport and oxidative phosphorylation (OXPHOS), in conjunction with the oxidation of metabolites by the Krebs's cycle and the breakdown of fatty acids by oxidation.

Because of their ability to generate ATP, for a long time mitochondria were known exclusively for their ability to act as the main fuel for the basic energy demands of the cell.

The Multi-Talented Mitochondria

Researchers are discovering that mitochondria play an even greater role than previously thought. For example, mitochondria not only fuel the cells, they also serve as the executioner for unhealthy cells.¹ Without mitochondria, the initiation and execution of apoptosis—the process by which the body weeds out unhealthy cells—wouldn't occur.

The mitochondria also help control calcium homeostasis. In addition, it's in the mitochondria of the cells where reactive oxygen species (ROS) are produced. Ironically, mitochondria also are an immediate target of attack from those same reactive oxygen species and are therefore especially vulnerable to oxidative stress.²⁻³


In mitochondrial DNA (mtDNA), oxidative damage occurs at a rate five to 10 times higher than in nuclear DNA.⁴ Mitochondria lack the protective histones—highly alkaline proteins that act like a spool around which DNA winds—present in other DNA.⁴ This can have grave consequences in the body because damaged mitochondria burn fat and other energy substrates less efficiently.⁴ This not only creates less energy, but also sends the mitochondria into a vicious cycle whereby ROS damage the mitochondria, in turn causing the mitochondria to produce even more damaging ROS.

Even though mitochondrial DNA is not part of the genetic code in the nucleus of the cells because it's an extranuclear genome, mitochondrial DNA mutations can induce epigenetic changes in the nucleus of cells. This can create genomic instability in the nuclear genome, leading to disease.⁵

In fact, no matter what disease you're treating in a given patient, the chances are good that mitochondrial dysfunction has played a critical role.

Mitochondrial Saboteurs

Our mitochondria are continually confronted with factors that can jeopardize how well they function.



Mitochondrial health is driven to a certain extent by genetics. We've all inherited our mitochondrial DNA from our mothers. Almost no mitochondria are present in sperm.⁴ So if the maternal mitochondrial DNA isn't healthy, the child who inherits the mtDNA will have to overcome this challenge.

But genetics are only part of the story. Lifestyle factors are important, too. For example, chronic stress. When rats were subjected to the stress of overcrowding in cages, they suffered from inflammation. Additionally, mitochondrial activity was disturbed throughout the intestine, triggering intestinal stress-related disorders, such as irritable bowel syndrome.⁶⁻⁷

Sleep apnea is another cause of mitochondrial dysfunction. The intermittent hypoxia that occurs during sleep apnea generates a large amount of ROS in the mitochondria. The ROS are produced at a greater rate than they are eliminated, creating oxidative stress that damages the mitochondria in the brain.⁸

Age also plays an important role. Aging mitochondria become progressively inefficient. When mitochondria are working efficiently, a process called mitophagy causes the turnover of cellular constituents. This eliminates dysfunctional or damaged mitochondria, counteracting degeneration, reducing inflammation and preventing unwarranted cell loss. The combination of mitochondrial dysfunction and decreased expression of mitophagy-regulating genes that can occur with aging may be to blame for age-related diseases.⁹

We're also exposed to a whole host of substances that can damage mitochondria. Certain drugs, for example, have this effect. Statins are some of the worst offenders. Researchers have shown that simvastatin causes mitochondrial respiratory dysfunction associated with increased generation of superoxide at complexes-I and II of the mitochondrial respiratory chain.¹⁰ Two of the most common side effects of statins are myopathy and rhabdomyolysis. Studies suggest that these side effects may be linked to impairment of mitochondrial functions caused by depletion of coenzyme Q10 (CoQ10).¹⁰

Antibiotics have similar effects on the mitochondria. Antibiotics cause mitochondrial dysfunction and ROS overproduction in cells, producing oxidative damage to DNA, proteins and membrane lipids. Mice treated with antibiotics exhibited elevated oxidative stress markers in the blood, oxidative tissue damage and up-regulated expression of key genes involved in antioxidant defense mechanisms.¹¹

Environmental toxins also impair mitochondria. In one animal study published in January 2013, exposure to BPA—a toxin commonly found in some plastics, food storage containers, liners of metal cans and

cash register receipts—caused mitochondrial defects in beta cells. These defects included depletion of ATP, loss of mitochondrial mass and membrane potential and alterations in expression of genes involved in mitochondrial function and metabolism.¹² Other toxins such as persistent organic pollutants (POPs) also are known to cause insulin resistance instigated by the mitochondrial dysfunction that occurs after exposure to POPs.¹³

BPA's effects on the mitochondria aren't limited to beta cells. In another animal study published in June 2012, researchers found that even low levels of BPA—below the no observed adverse effect level—can cause liver damage and mitochondrial dysfunction by increasing oxidative stress and inflammation. The study authors administered two doses of BPA, 0.05 and 1.2 mg/kg body weight per day, to mice, for five days. Both treatments damaged the structure of the liver mitochondria, although oxygen consumption rate and expression of the respiratory complex decreased only at the higher dose.

In the BPA-treated mice, the expression of the mitochondrial respiratory complex III and V was also reduced compared to the control mice. During BPA treatment, antioxidant enzyme levels in the liver plummeted, so that liver cells had no protection against the oxidative stress caused by BPA, resulting in mitochondrial dysfunction.¹⁴

When these same researchers conducted an in vitro study using a lower and higher dose of BPA, both doses caused a decline in the oxygen consumption rate, ATP production and the mitochondrial membrane potential.¹⁴


Airborne pollutants also can create mitochondrial dysfunction. Being exposed to airborne pollutants causes an increase in mitochondrial DNA copy number, a marker of mtDNA damage, as well as alterations in mitochondrial gene expression.¹⁵ Heavy metals such as mercury can have similar effects on mitochondria in the brain.¹⁶

When it comes right down to it, no matter which of these saboteurs is causing the mitochondrial dysfunction, the result is the same—the body develops one or more diseases.

Diseases of the Brain

The brain is more vulnerable to oxidative stress compared to other areas in the body because it has high oxygen demands and contains a high degree of polyunsaturated fats, which are sensitive to ROS attacks. Additionally, the brain can only partly defend itself against ROS build-up, and it has a higher percentage mitochondria due to its high energy requirements.¹⁶⁻¹⁷

The damage that occurs in brain mitochondria under



conditions of oxidative stress plays an integral role in several diseases of the brain. Researchers have observed mitochondrial dysfunction in virtually every neurodegenerative disease.¹⁸

Mitochondria are involved in the biochemical pathway by which beta-amyloid protein—a key factor in the development of Alzheimer’s disease (AD)—can lead to neuronal dysfunction.¹⁹ Additionally, researchers have identified a number of mitochondrial abnormalities in the hippocampal neurons of Alzheimer’s patients compared to age-matched controls.²⁰⁻²²

Biopsies from Alzheimer’s brains showed a significant reduction of mitochondria, except at the site where mitochondria are degraded.²⁰⁻²¹ These mitochondrial abnormalities were accompanied by oxidative damage, indicating that the mitochondria were damaged during the progression of Alzheimer’s.²⁰ Researchers also reported a significant decrease of mitochondrial complex IV activity in the cortical regions of Alzheimer’s brains.²² Other evidence suggests that beta-amyloid may directly disrupt mitochondrial function and contribute to the deficiency of energy metabolism and neuronal death seen in Alzheimer’s. Studies have found that beta-amyloid was localized to mitochondria in brains of Alzheimer’s patients.²³ When beta-amyloid was present in the mitochondria, there was impaired mitochondrial metabolism and increased mitochondrial ROS production.²³

Beta-amyloid treatment of mitochondria also is thought to cause oxidative injury to mitochondrial membranes, disrupt lipid polarity and protein mobility and inhibit key enzymes of the mitochondria respiratory chain, leading to increased mitochondrial membrane permeability.²⁴⁻²⁵

Other evidence suggests that the defects in mitochondria that occur during Alzheimer’s disease start occurring before the formation of beta-amyloid plaques, indicating that the mitochondria dysfunction occurs early in the development of Alzheimer’s.²⁶

“Therefore,” one group of researchers wrote, “early mitochondrially targeted therapeutic interventions may be effective in delaying the onset and progression of AD.”²⁶

Furthermore, mutations causing genetic forms of Parkinson’s disease or hereditary neuropathies impact key molecular players involved in the recycling of defective mitochondria.¹⁸

In May 2013, researchers demonstrated for the first time that dysfunctional mitochondria in brain cells also can lead to learning disabilities due to the fact dysfunctional mitochondria aren’t efficiently cleared from the cell. This interferes with the operation of other healthy mitochondria and leads to insufficient energy production in the cell.²⁷

Mitochondrial Function and Autism

Knowing the role that mitochondria play in brain health, it is perhaps not surprising that a number of studies have found a connection between mitochondrial dysfunction and autism. Evidence is beginning to indicate that, in the brains of people with autistic spectrum disorder (ASD), mitochondrial function and intracellular redox status are compromised in the pyramidal neurons and that this mitochondrial dysfunction occurs during early childhood when ASD symptoms first appear.²⁸

Mitochondrial damage provides a plausible explanation as to why mercury exposure may be implicated in the development of autism, as mercury causes mitochondrial malfunction.¹⁶

There is some indication that patients who have autistic spectrum disorder and mitochondrial disease often show signs and symptoms uncommon to idiopathic ASD such as cardiac, pancreatic or liver dysfunction, growth retardation and becoming easily fatigued.²⁹

Mitochondrial Function and Heart Health

Like the brain, the heart contains a larger number of mitochondria due to its high metabolic activity. In the heart, approximately 35 percent of the cardiomyocyte volume is comprised of mitochondria.³⁰

Heart function is directly dependent upon the health of the mitochondria. Following a heart attack or conditions that damage heart muscle, the mitochondria become dysfunctional. They then produce an enzyme known as CaM kinase, which promotes leakiness of mitochondria and increases heart muscle damage by allowing too much calcium to enter the mitochondria. This is responsible for the death of heart cells. If too many cells die, it leads to heart failure.³⁰


Mitochondrial Function and Cancer

Mitochondrial dysfunction occurs in many types of human cancer cells. Cancer cells undergo increased oxidative stress partly due to mitochondrial malfunction. The increased ROS in cancer cells may in turn further lead to stimulation of cellular proliferation and cell migration and invasion, contributing to carcinogenesis.³¹

Mitochondrial Function and Metabolic Syndrome

Mitochondrial dysfunction is the common denominator for most of the risk factors of metabolic syndrome, including central obesity, high cholesterol and triglycerides, hypertension, insulin resistance and type 2 diabetes.³²

Multiple studies suggest that human insulin resistance is accompanied by impaired mitochondrial OXPHOS. Researchers think this is because of the accumulation of triglycerides or toxic lipids (such as ceramide or diacylglycerol), paired with reduced mitochondrial



density. Furthermore, elderly people often have decreased insulin sensitivity due to age, which is associated with a defect in mitochondrial function in muscle.⁹

Other Mitochondrial-Related Conditions

Mitochondrial dysfunction is involved in other diseases as well. Recently, researchers discovered that mitochondrial dysfunction is involved in airway epithelial injury and asthma development.³²

In chronic fatigue syndrome (CFS) and fibromyalgia, it is thought that reactive oxygen species reduce mitochondrial function, which may result in muscle pain and fatigue. Mitochondrial malfunction occurs in leukocytes of CFS patients and in muscle cells of fibromyalgia patients, providing a logical explanation for the muscle pain. Additionally, researchers think that mitochondrial dysfunction might also occur in central neural cells, resulting in lowered ATP levels, leading to generalized hypersensitivity and chronic widespread pain.³³⁻³⁴

Irritable bowel syndrome is another mitochondrial-related condition. IBS is worsened or caused by chronic stress, which damages the mitochondria in the intestine.⁶⁻⁷

Migraines and depression also are linked to mitochondrial malfunction.³⁵ Some drugs used to treat depression positively affect mitochondrial health and reduce inflammation, indicating that these two mechanisms of action may be the reason the drugs are effective. Furthermore, treatments commonly used to benefit the mitochondria and reduce inflammation also are effective in affective spectrum disorders, including depression.³⁶ Finally, the expansive list of mitochondrial-related diseases includes advanced macular degeneration (AMD). The retina is especially vulnerable to oxidative damage because it is among the most metabolically active tissues by weight in the body. It's also constantly bombarded by photochemical stress. Photoreceptors contain a large number of mitochondria and produce high levels of reactive oxygen species. When these ROS are inadequately controlled, tissue damage occurs, leading to AMD.³⁷

Protecting the Mitochondria

The first step in strengthening the health of our patients' mitochondria involves addressing lifestyle factors. Studies show that increasing physical activity improves mitochondrial function, so encouraging regular moderate exercise is essential.³⁸

To counteract insulin resistance, exercise combined with a diet rich in vegetables and healthy, organic, grass-fed meats and low in refined carbs and sugar will help stabilize blood sugar.

Encouraging our patients to reduce stress with practices such as meditation or yoga, as well as ensuring that sleep apnea is ruled out also is important. I make certain that I test all my patients with hypertension or heart disease for sleep apnea.

Additionally, detoxifying the body is critical. Recommend that your patients avoid all canned foods (with the exception of companies that have removed the BPA from their cans). Suggest patients wash their hands after touching cash register receipts. And also recommend a good detoxification supplement.

Several other supplements are important in any regimen designed to boost mitochondrial health.

One of those supplements is L-carnitine, which helps fatty acids cross the inner mitochondrial membrane. It also scavenges reactive oxygen and binds iron—a metal involved in mitochondrial oxidative stress that leads to mitochondrial damage.¹⁰ L-carnitine also has been shown to prevent the damaging effects of statins on the mitochondria.¹⁰

L-carnitine protects against mitochondrial dysfunction associated with oxidative stress caused by a series of conditions such as aging, ischemia reperfusion, inflammation, degenerative diseases, cancer and drug toxicity, in vivo or in vitro.¹⁰

L-carnitine also is essential for the detoxification of environmental pollutants, meaning it can protect the mitochondria on a number of levels.⁹

Coenzyme Q10 (CoQ10) is an essential cofactor involved in the mitochondrial electron transport chain. CoQ10 protects against beta-amyloid-induced mitochondrial malfunction.³⁹ Statin drugs are thought to cause mitochondrial damage in part by lowering levels of CoQ10, and supplementation with this important antioxidant has been shown to counteract some of the negative effects of statins.^{40, 10}

Finally, D-ribose is another excellent addition to a mitochondrial resuscitation regimen. D-ribose reduces markers of oxidative stress that can form after high-intensity exercise.⁴¹ It also boosts post-exercise ATP levels⁴² and has benefited heart disease patients.⁴³ D-ribose replenishes low myocardial energy levels, improving cardiac dysfunction following ischemia. Studies also have shown it can improve ventilation efficiency in patients with heart failure.⁴³

Conclusion

Mitochondrial dysfunction is involved in most, if not all, diseases. Therefore it's crucial to address our patients' mitochondrial health by encouraging a healthy lifestyle, along with optimal detoxification and nutrients such as L-carnitine, CoQ10 and D-ribose.

Action Steps

Mitochondrial Resuscitation

Supporting the health of our patients' mitochondria can result in excellent progress, no matter which disease they are battling. Here are some ways to incorporate mitochondrial support into treatment regimens.

- Encourage exercise. For patients who are in a lot of pain, emphasize that even small amounts of daily exercise can be beneficial.
- Recommend a low-glycemic-load diet with an emphasis on vegetables and grass-fed meat in order to balance insulin levels.

- Test patients for sleep apnea, especially those who snore or who have a heart condition or hypertension.
- Recommend a good detoxification supplement and advise patients to wash their hands after touching cash register receipts.
- Advise against consuming canned food, unless it's from a company who has declared their cans BPA-free, and remind patients to drink out of BPA-free water bottles.
- Suggest patients add L-carnitine, CoQ10 and D-ribose to their supplement regimens

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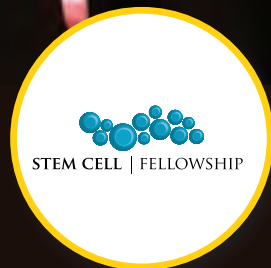
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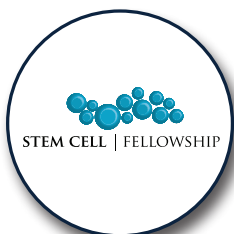
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Some Effects of Food Intolerance on Athletic Performance & Weight

By- Roger Deutsch



Age appropriate exercise confers numerous health benefits not least of which is often improvement in body composition and weight. However, even vigorous exercise and healthful diets, comprised of high nutrient density, diversity and quality, are sometimes insufficient to achieve healthy weight and performance objectives. A growing body of evidence suggests that even healthful foods can, based on idiosyncratic factors, trigger inflammatory cascades that negatively impact metabolic function. Hence, weight objectives based on diet and exercise regimens often fall short of hopes and expectations. The notion that immune activation may impede normal weight and body composition is not at odds with the sacrosanct notion that energy excess results in weight gain and energy deficiency results in weight loss. Rather, it highlights the role of immune activation on metabolism and points to a need for individualization of diet.

Following on from this, FOOD INTOLERANCE IS A RAPIDLY INCREASING PROBLEM THAT DIFFERS FROM FOOD "ALLERGY" IN THAT SYMPTOMS ARE USUALLY CHRONIC AND DELAYED, PERHAPS BY SEVERAL HOURS OR EVEN DAYS. HENCE THEY HAVE RECEIVED RELATIVELY LITTLE ATTENTION from the medical and scientific communities until recently when knowledge of gluten induced autoimmunity and auto inflammation has broadened awareness both in the professional and lay sectors of society. Clinical insight into specific FOOD intolerances is emerging as a POWERFUL TOOL FOR IMPROVING both ATHLETIC PERFORMANCE AND WELL BEING.

Eating healthful food is one thing, but eating in accordance with genetically (both host and micro biome) determined biochemical makeup, brings dieting to a personalized and more sophisticated level. One man's meat is another man's poison.

Foods that are compatible with the individual physiology will increase strength and energy. Consider the

upward trajectory of professional athletes' (like that of David Ortiz) performance once they have adopted a scientifically determined personalized eating regimen. Conversely, foods or added chemicals that the body perceives as harmful will result in chronic inflammation. Food intolerance induces the excessive generation of toxic free radicals and inflammatory chemicals. The damage can result in a wide range of health problems. The following are some of the effects of food intolerance and inflammation that are of strong interest to athletes and health conscious individuals:

- Direct damage to the mitochondria, the organelles where energy is produced. Intolerogenic foods activate the innate immune system and the free radicals produced can cause damage to the DNA, and other structures within the mitochondria, resulting in lower energy production. This is of special significance to athletes as intense exercise alone will generate damaging free radicals from normal metabolic process sped up somewhat by the intensity of the exercise.
- Inflammation also causes shortening of the ends of the strands of our DNA called telomeres. Telomeres, like the plastic pieces at the end of a shoe lace, hold the DNA strands together. Whenever the cell divides, and immune cells divide frequently, the telomeres are shortened. When they become too short the cells senesce prematurely. Shortened telomeres are associated with cardiovascular disease, generalized weakened immunity in old age, cancer and other degenerative processes.
- Auto-immunity can also arise from too much inflammation. Some immune cells die in the process of trying to neutralize a food. These dead cells release their own genetic material, DNA, which, if too great, may be seen as "foreign" bodies the immune system must attack. However, this time you are attacking the blueprint for you own proteins and auto-immune reacts may occur. Associations have been seen with Lupus and RA.

- Cytokines, like Interleukin 6, and tumor necrosis factor alpha, block insulin receptors. This is a recently discovered finding made by medical scientists which explains why food intolerance makes people fat. The muscles cells are starved of important energy material which is instead stored as fat in fat cells.
- Food intolerance can cause inflammation in the gut. An extreme form is celiac disease. This reduces the absorption of nutrients that are needed for proper energy production as well as all other biological processes. It also consumes serotonin in the gut. Serotonin deficits are related to sugar craving.
- Toxic immune chemicals, released by myeloid cells—such as elastase— damage muscle tissue.
- Food intolerance can cause inflammation in the upper and lower airways. Decreased air intake limits aerobic metabolic function.
- Chronic inflammation promotes fatigue. The same immune chemicals that fight viruses cause fatigue. The body wants you to rest, not exercise, so that energy can be used to fight the invader. The immune system that's inappropriately activated by the wrong food will thus cause you to be chronically fatigued to a lesser or greater extent. The immune system is now mistaking a food particle for a virus or bacterium.

The Solution

Many professional athletes are now turning to a new coach. It's not a conventional coach, it's not even a human coach; rather, it's a coach that instructs them in personalized dieting, a coach to help increase strength and normalize weight. It's a simple blood test for food and chemical sensitivity called the ALCAT Test.

Two of the most notable athletes to have taken the ALCAT Test are Minnesota Twins' First Baseman Justin Morneau and Boston Red Sox's Designated Hitter David Ortiz. Morneau is a former American League MVP and four time MLB All-Star. Morneau's experience was chronicled in the StarTribune wherein he states he was planning to 'do everything I can humanly do to make myself feel better.' The StarTribune also states: "While making the medical rounds last fall, Morneau also took the ALCAT Test to see if he had food allergies. He learned his body was sensitive, if not allergic, to sugars, gluten and dairy." Morneau then lost 14 pounds in just a few weeks by following his new diet.

Athletes are always seeking to enhance performance, and diet plays an essential role in their ability to do so. Morneau states, "It's not like I'll choke or die from eating those things, but my body reacts poorly to them. It treats them as something I'm allergic to and fights the inflammation instead of the inflammation from an injury."

The Red Sox David Ortiz was recently featured in a separate article titled, *The Skinny on Big Papi's Diet* featured on ESPN.com. Even though Ortiz took the ALCAT Test for different reasons than Morneau, he had a similar experience, dropping 20 pounds in two months. Ortiz took the test because his "cholesterol had risen to dangerously high levels." Since taking the test, and following its dietary guidelines, Ortiz said, "The results have been almost immediately noticeable." He goes on to say, "I feel great" and "I can feel the difference when I swing a bat and when I work out, I don't get as tired."

Studies have shown dietary modification and food sensitivity testing to be a viable treatment modality for a number of chronic health problems. Major League Baseball isn't the only professional sport that has utilized the ALCAT Test. Joe Rowgowski, Strength and Conditioning Coach for the NBA's Orlando Magic, has been quoted as saying, "I've seen very good results with the ALCAT Test and often recommend it to the athletes I work with." In addition, Juventus, historically the most successful Italian football club and one of the most important globally, was recently featured in a study titled *Rational Management of Food Intolerance* in an elite soccer club. This study was presented at the International Society of Sports Nutrition: 8th Annual ISSN Conference and Expo in 2011.

So what does all of this mean? This means your fork may actually be the best tool available for maximum performance. For the elite athlete, with all the available supplements and training methods perhaps it is indeed as simple as Hippocrates said, "Let food be thy medicine." For the elite athlete or the occasional weekend warrior the best medicine may actually be finding out what foods are right.

Author Bio:

Roger Deutsch has been working with the study of food intolerance for the past 27 years and is the author of the book series, *Your Hidden Food Allergies are Making You Fat*. He directs research at Cell Science Systems, Corp. RD@Alcat.com

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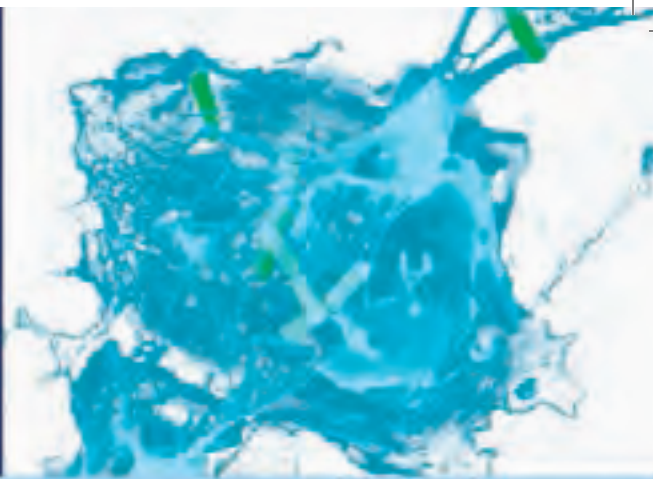
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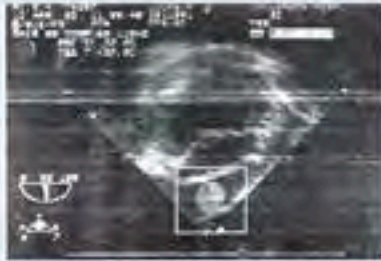
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Propitious Modulation of Fibroblast Quiescence Mediated by Cytokine Expression Mediated from Embryonic Stem Cells

By- Ron Shane, Ph.D., OMD; Andrew Crain; Andrei Golovko; Benjamin Morpurgo; Alexa Dillberger; Taylore Bonn; Amber Buz-Zard; Evan Snyder

ABSTRACT

The intent of this study is to ascertain whether a potpourri of cytokines derived from stem cells can actually impact the transcriptional status of older cell lines. The evidence from stem cell research indicates that the efficacy of these cellular systems is founded on the temporary effectiveness of their cytokine secretions. We are suggesting that growth factors from stem cells will have a dramatic effect on genetic quiescence in older cells.

INTRODUCTION

There have been a bevy of studies that have attempted to ascertain whether stem cells, either autologous or allogenic, can reverse age-related genetic cellular modifications.¹⁻⁴ These research endeavors, which have been executed on a diverse array of animal and human models utilizing stem cell therapies, have been less effective than anticipated. Furthermore, many kinds of stem cell regimes have been utilized by researchers without sufficient clinical therapeutic efficacy.⁵⁻⁷

Researchers have employed autologous progenitor cells, as well as, mesenchymal stem cells from bone marrow and allogenic stem cells for a plethora of therapies from repairing spinal cord injuries to that of the treatment of brain pathologies. Moreover, the result of many studies on older phenotypes, in terms of reversal of quiescence likewise, has not been clinically relevant. The use of stem cells, either IV or systemic, as well as regional titration, has not yielded any edifying results with respect to modulating genetic expression or the mitigation of programmed molecular aging. It is evident that the heuristic benefit of stem cells for ameliorating the genetic changes associated with cellular aging has been very nominal in all mammalian phenotypes; and this includes the various kinds of stem cell strategies.

Systemic injections of stem cells have consistently shown that these protocols do not provocatively adhere to tissue sites for a prolonged period of time. Moreover, systemic diffusion is quite problematic; and in general, stem cells are not incorporated in a diverse array of aging tissue

regions. Many researchers have speculated that stem cells may not necessarily repopulate specific tissues, but more importantly, they do not reprogram genetic aging as a result of optimizing transcriptional activities of older quiescent cell lines. Conversely, those stem cells which are incorporated into specific tissues become differentiated into adult cell lines. However, their transcriptional and translational proclivities will not be youthfully medicinal. These cell lines which have been differentiated from grafted stem cells become molecularly consistent in terms of their transcriptional activities with their aged extracellular milieu. These stem cells, which should express proteins concordant with a youthful phenotype, instead become remodeled in a fashion that is similar to the genetic profile of the other aging cell lines in a certain tissue environment. Thus, IV injections of stem cells will not reprogram genetic expression where older cell lines will then become robustly youthful. It is evident that this paradigm must be forsaken and researchers need to develop a qualitatively distinctive model that is more adaptive to mitigate programmed cellular aging. There is consistent speculation that the medicinal and therapeutic benefits of stem cells are related to their unique profile of cytokine production, which can significantly impact genetic transcriptional activities of compromised cell lines. Moreover, it has been theorized by the authors that cytokine activation of seemingly quiescent cells is a viable strategy to ameliorate compromised transcriptional genetic activities. Stem cells, in this new emerging paradigm, are now regarded as cellular factories for the production of edifying growth factors that can reverse age-related transcriptional dysfunctionality. There is some evidence for this paradigm based on Thomas Rando's work on heterochronic parabiosis.^{8,9} Dr. Rando has suggested that youthful circulating cytokines do amelioratively impact gene expression in the older rodent phenotype.

The intent of this study is to ascertain whether a potpourri of cytokines derived from stem cells actually can impact the transcriptional status of older cell lines.

The evidence from stem cell research indicates that the efficacy of these cellular systems is founded on the temporary effectiveness of their cytokine secretions. We are suggesting that growth factors from stem cells will have a dramatic affect on genetic quiescence in older cells. We will be demonstrating in this study, that stem cells are not necessary to mitigate the effects of genetic aging, but rather their paracrine secretions are far more efficacious.

We are interested in understanding which growth factor profiles are actually most effective at modulating cellular aging. Currently, there has not been any study to ascertain if stem cells differ with respect to the effectiveness of their growth factor matrixes on attenuating programmed cellular aging in human fibroblast. In this study, we will be comparing growth factors from autologous stem cells with cytokine secretions derived from embryonic stem cells (ESC). Furthermore, we will also be utilizing ESC that are not fertilized; and examining their effectiveness for impacting and improving transcriptional activities of older fibroblasts.

RESULTS

The results of this study indicate that treating older human fibroblasts with ESC culture media can impact gene expression, making the older phenotype more like the untreated younger control. Indeed, fibroblasts from the 63-year-old donor began to express a gene profile more consistent with the donor's 29-year-old daughter after being treated with the ESC culture media. We found that many genes were turned on when treated with ESC growth factors. In general, there are at least 300 genes either upregulated or downregulated as a function of the ESC cytokine treatment therapy. Moreover, we have not yet fully established with exacting clarity the significance of modifying the expression profile of 300 genes, or how this impacts overall morphological changes in the older fibroblast phenotype. Notably, in the dendrogram of the filtered expression data we observe that the 63_ESC sample (older fibroblast treated with ESC cytokines culture media) is clustering closely with the 29_fib samples (untreated younger samples). Hence, there is a strong likelihood that treating with ESC cytokines can change overall gene expression where the mother's older fibroblast's become closely akin to her daughters untreated fibroblasts (29_fib). In our view, it is evident that a greater sample of mother and daughter fibroblasts need to be used for a more comprehensive study in order to determine if this innovative treatment strategy is actually effective for changing age related transcriptional profiles in human fibroblasts. In our view this research in terms of our analysis of microarray data does indicate that cytokine therapy definitely impacts upon transcriptional behavior with respect to changing genes that are involved in a physiological process which has been defined as aging. Our results are intriguing and lead us to believe that cytokine interventions can improve and optimize gene transcriptional profile. Observational Cells were fed on day-2 and day-4, and harvested on day-5. Results are shown in *Table 1*.

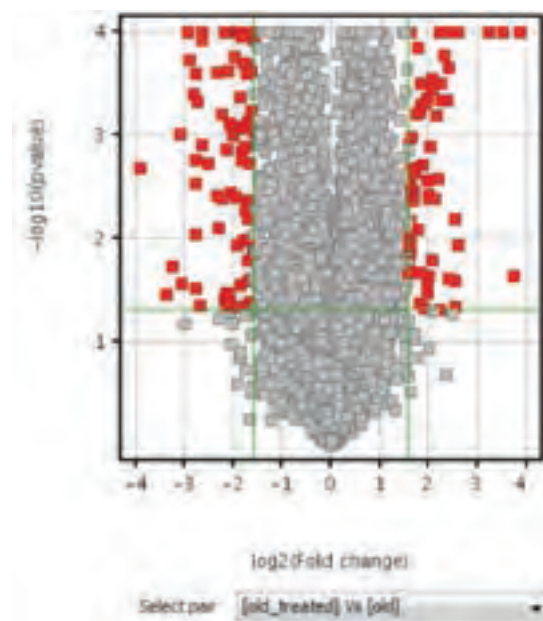


Figure 1. The global changes in gene expression between the old-treated and the old-untreated samples, illustrated by using a Volcano plot. The red squares represent the genes that satisfy the significant-change threshold; a greater than 2-fold expression change with a p-value < 0.05. Fold change is plotted on the x-axis and $-\log_{10}(pvalue)$ on the y-axis.

RESULTS

Fibroblasts, from a 63-year-old female (SC-0001) and a 45-year-old female (SC-0002), proliferation rates over 5-days in 6-well plates exposed to 4 different media conditions.

Cells were first thawed from cryopreservation in FibroLife basal media (2% FBS) and allowed to recover and proliferate for 5-days. Cells were then passaged using T/E and re-plated at about 3,000/cm² or 28,800 cell per well into the 4 media conditions as listed below in 6-well plates.

Media composition 75% (v/v) FibroLife basal media (FLb) + 25% (v/v) unconditioned or conditioned media listed below:

1. FLb + FibroLife basal media (2% FBS) = FL
2. FLb + Unconditioned PluriLife = UP
3. FLb + Fibroblast conditioned PluriLife = PL
4. FLb + H9 conditioned PluriLife = H9

SC-0001	None Endogenous	Oral Steroids	Topical Steroids	Vaginal Steroids	Troche Steroids
	Cell count	345,000	3,750,000	1,455,000	4,650,000
	Population doublings	3.6	7.0	5.7	7.3
SC-0002		FL	UP	PL	H9
	Cell count	665,000	6,120,000	8,010,000	21,540,000
	Population doublings	4.5	7.7	8.1	9.6

Table 1. Cell counts and population doubling on day 5.

Fibroblasts were growing in media that was suitable for proliferation in all cases; morphology in all 4 media conditions was normal and healthy after recovery from cryopreservation. Media #4 prepared by conditioning with H9 stem cells increased the growth of the fibroblasts more than standard media alone or media conditioned with fibroblast. Stem cell conditioned media increased both total number of cells and number of population doubling during the 5-day exposure.

The principal intent of this preliminary study was to determine if cytokine regimes of succinct stem cell sources affect gene transcription in human fibroblasts. We have found that culture media from adipose cells and unfertilized ESC have nominal effects with respect to changing transcriptional profiles in older fibroblasts. This connotes, in terms of modulating gene expression, that the older human fibroblasts did not become consistent with their daughters. Furthermore, it appears that the culture media or cytokines from ESC were only able to modify older fibroblasts in terms of their gene expression profile to where these cells then became similar to the untreated daughters fibroblasts.

In our view, the particular nature or matrix configuration of cytokines expressed by ESC seems to drive gene expression in a manner where these older fibroblasts are activated away from their age-related quiescence to where they exhibit transcriptional behavior to their daughters' cells. Thousands of genes were impacted from this treatment protocol; and hundreds of others displayed a 3-fold level change. It is difficult to know the multitude of proteins that would be affected from this kind of exogenous treatment strategy in humans. In brief, our results may represent an innovative paradigm shift away from stem cell therapies, and instead focus on their paracrine peptides as a medicinal strategy.

DISCUSSION

There is a kind of plasticity that is inherent in cellular behavior; and also in vivo systems, which may be possibly affected by sagacious cytokine interventions. Genetic aging is only partially regulated by biofeedback mechanisms of intercellular enzymatic activities; and they are only partially responsible for virulent changes to the extracellular milieu. Conversely, extracellular cytokine-like circadian modulation, which is not yet understood, mollifies robust genetic expression or overall transcriptional behavior from distinctive cellular systems. Furthermore, enzymatic and cytokine expression from cellular systems can further enhance the extracellular milieu's catabolic activities.

The intent of this study was to demonstrate that exogenous cytokine intervention can attenuate transcriptional dysfunctionality in older human fibroblasts treated with ESC cytokines. These older fibroblasts did exhibit transcriptional characteristics of

a younger phenotype after therapeutically being treated with these cytokines matrixes. Moreover, cytokine profiles may also propitiously affect overall protein expression; and this is contingent upon the fact that there is amelioration in these older fibroblasts where their genetic expression is more characteristic of their younger progeny. Cytokine dysfunctionality or aging can be optimally reprogrammed as a result of exogenous peptide intervention; and this was the principal focus of this study. The older quiescent fibroblast displayed transcriptional changes consistent with the untreated fibroblasts of the participant's 29-year-old daughter.

We believe that cellular aging is not an absolute program, but rather, should be regarded as a biological phenomenon with viable plasticity. This connotes that cellular aging can be mitigated when modulated by particular cytokine matrixes. We also determined that ESC cytokines are most efficacious for mollifying molecular programmed aging. Thus, we demonstrated in this preliminary paper that cytokines, not stem cells are the propitious agents that induce volitional plasticity where aged cells can once again express optimal molecular machinations.

Dr. William Swindel wrote the following regarding skin aging in the human phenotype: "Skin from older individuals, for instance, can lack regenerative capacity required for wound healing and may provide a weakened immune barrier against pathogens, while also exhibiting an increased burden of somatic mutations that heighten susceptibility to melanomas and squamous cell carcinoma. Senescent decline in skin function is simultaneous with normal aging which involves diminished capacity to maintain homeostatic and overall loss of physiological reserve."¹¹

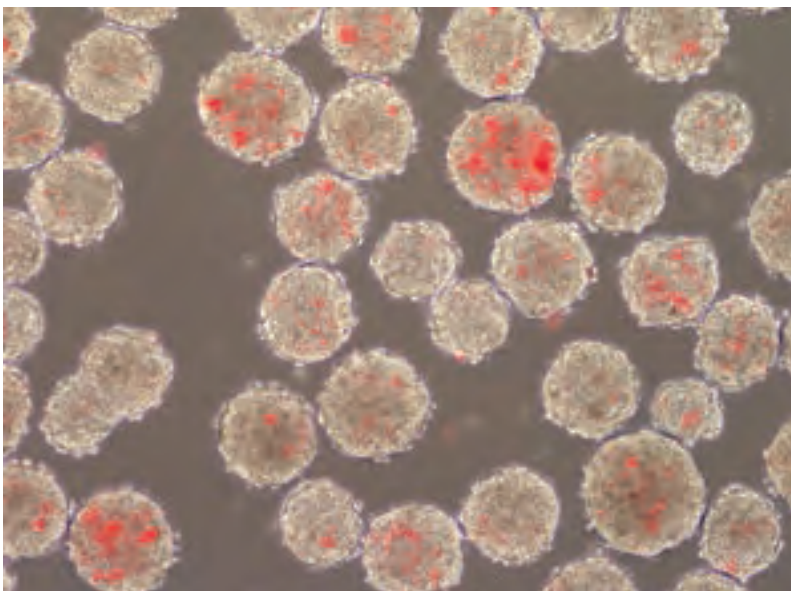
In the ensuing pages, we have outlined in terms of their functionality, what particular gene expression is equated with succinct protein secretion. Our cytokine treatment strategy appears to have ameliorated protein expression in the older human fibroblasts. It is evident that this cannot be empirically demonstrated in a cultured environment. Furthermore, as stated elsewhere in this article, particular genes that are characteristic of a younger phenotype were upregulated after cytokine therapy. Dr. Shane is currently involved in a sophisticated study employing cytokine therapy in the mouse model to see if this intriguing protocol actually reverses tissue and cellular aging. These results will be available in early 2013. Dr. Rando has shown that circulating cytokines from a younger phenotype do affect muscle genesis, cell density, and wound healing characteristic of a younger phenotype.⁸ In general, the results of this study are intriguing, as they suggest that cytokine intervention from ESC may reverse gene expression characteristic of a younger phenotype. This innovative protocol may turn on quiescent genes that have been silenced as a function of aging, and recapitulate protein expression in

a manner where tissue genesis is reflective of a younger phenotype. This is the first preliminary study to examine if cytokine intervention can ameliorate gene expression and tissue aging.

Cytokines expressed by ESC afford the most propitious source to affect gene transcription in fibroblasts compared to adipose progenitor cells. We did demonstrate in a limited sample that older human fibroblasts, when treated with culture media from ESC, do exhibit a gene expression profile characteristic of a younger phenotype, or her daughter's untreated fibroblasts. In general, many genes were affected that would translate into protein expression, which can then be concordant with regenerative robust peptides or what is depicted as reversal of aging.

ABOUT THE AUTHOR

Primary author Dr. Ron Shane is a multiple disciplinary PhD with post-doc training in Renaissance mysticism, western medicine, neuroscience, cross-cultural shamanism, and neuroendocrinology. He is an accomplished researcher in optimal and age management medicine as well as a Doctor of Oriental Medicine. An expert master practitioner in kundalini yoga, the energy arts, Dr. Shane holds several seventh degree certificates in Tae Kwon Do including one from grandmaster Hwang, the highest living Tae Kwon Do master. Dr. Shane is considered to be one of the world's leading experts in the spiritual aspect of martial arts. He has written hundreds of articles in various aspects of higher consciousness and optimal medicine. This energy master has over 15 books available online on topics ranging from optimal health to that of higher esoteric consciousness. Dr. Shane is a research faculty member in the area of Shakespeare and Mysticism at the University of California/San Diego; and an adjunct faculty member at several universities concerned with mind body consciousness as well as a leading researcher in regenerative medicine.



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Gene Symbol	Possible Function	Possible Implications in Aging
Upregulated Genes in Young Versus Old and Treated Versus Old		
ALDOA	Aldolase A deficiency has been associated with myopathy and hemolytic anemia	Upregulation could be a sign of a reversal of myopathy and hemolytic anemia.
ATP6V0C	These transporters play an important role in processes such as receptor-mediated endocytosis, protein degradation and coupled transport. They have a function in bone reabsorption and mutations in the A3 gene cause recessive osteopetrosis.	Upregulation could be a sign of a bone improvement, or reversal of osteopetrosis.
CDK7	This protein is thought to serve as a direct link between the regulation of transcription and the cell cycle	Upregulation may be a sign of more active cell cycle?
COL8A2	Defects in this gene are associated with Fuchs endothelial corneal dystrophy and posterior polymorphous corneal dystrophy type 2	Upregulation may be a sign of smooth muscle improvement.
DNM2	Has been implicated in cell processes such as endocytosis and cell motility, and in alterations of the membrane that accompany certain activities such as bone resorption by osteoclasts.	Upregulation may be a sign of muscle improvement.
ENO2	Has neurotrophic and neuroprotective properties on a broad spectrum of central nervous system (CNS) neurons.	Upregulation may be a sign of improved neuroprotection.
FBLN5	Promotes adhesion of endothelial cells through interaction of integrins and the RGD motif. Could be a vascular ligand for integrin receptors and may play a role in vascular development and remodeling	Upregulation might be related to reversing the aging process because the gene is involved in bone formation, fracture repair and vascular development.
PDLIM7	Involved in both of the two fundamental mechanisms of bone formation, direct bone formation e.g. (embryonic flat bones mandible and cranium), and endochondral bone formation e.g. (embryonic long bone development). Plays a role during fracture repair.	Upregulation might be related to reversing the aging process because the gene is involved in bone formation, fracture repair and vascular development.
GLS	This protein is primarily expressed in the brain and kidney plays an essential role in generating energy for metabolism, synthesizing the brain neurotransmitter glutamate and maintaining acid-base balance in the kidney.	Upregulation may be a sign of improved metabolism.
GPI	Mammalian GPI can function as a tumor-secreted cytokine and an angiogenic factor (AMF) that stimulates endothelial cell motility.	Upregulation could be a sign of a reversal of anemia and improved angiogenesis.
HK2	In addition to glucose metabolism, mitochondrial hexokinases have been implicated in anti-apoptotic and cell survival signaling.	Upregulation may indicate reversal of apoptosis
KRT7	Blocks interferon-dependent interphase and stimulates DNA synthesis in cells.	Upregulation may be a sign of more active cell cycle?
MET	In the adult, MET expression is limited to stem and progenitor cells and is necessary for wound healing and hepatocyte regeneration	Upregulation may indicate improving of the wound healing and hepatocyte regeneration
MKL1	Suppresses TNF-induced cell death by inhibiting activation of caspases. It may up-regulate anti-apoptotic molecules.	Upregulation may indicate reversal of apoptosis
PAK4	Serine/threonine protein kinase that plays a role in a variety of different signaling pathways including cytoskeleton regulation, cell migration, growth, proliferation or cell survival. Stimulates cell survival by phosphorylating the BCL2 antagonist of cell death BAD. Alternatively, inhibits apoptosis by preventing caspase-8 binding to death domain receptors in a kinase independent manner.	Upregulation may indicate reversal of apoptosis
PKM2	Plays a general role in caspase- independent cell death of tumor cells.	Upregulation may be a sign of tumor suppression
TBRG4	May play a role in cell cycle progression	Upregulation may be a sign of more active cell cycle.
TGFB11	May play a role in the processes of cell growth, proliferation, migration, differentiation and senescence	Upregulation may be a sign of more active cell cycle.
TUBB6, TUBB2A	Tubulin is the major constituent of microtubules. Microtubules form the spindle fibers for separating chromosomes during mitosis.	Upregulation may be a sign of more active cell cycle.
TXNRD1	Induces actin and tubulin polymerization	Upregulation may be a sign of more active cell cycle.
USP14	Serves also as a physiological inhibitor of endoplasmic reticulum-associated degradation (ERAD) under the non-stressed condition	Since USP14 represses protein degradation, maybe its upregulation signals reversal of apoptosis.
Downregulated Genes in Young Versus Old and Treated Versus Old		
BST1	The polyclonal B-cell abnormalities in rheumatoid arthritis may be, at least in part, attributed to BST1 overexpression in the stromal cell population.	Downregulation may be a sign of rheumatoid arthritis reversal.
NOA1 (C4orf14)	Plays a role in mitochondria-mediated cell death	Downregulation may indicate reversal of apoptosis
CCNB1IP1	Overexpression causes delayed entry into mitosis. Downregulation of CCNB1IP1 may result in increased mitosis, which may also be a sign of reversed aging.	Downregulation may be a sign of more active cell cycle.
CHEK1	May negatively regulate cell cycle progression during unperturbed cell cycles. It is required for checkpoint mediated cell cycle arrest in response to DNA damage or the presence of unreplicated DNA	Downregulation may be a sign of more active cell cycle.
HLA-F	Involved in the presentation of foreign antigens to the immune system.	Less inflammation?
SPSB3	May be a substrate recognition component of a SCF-like ECS (Elongin BC-CUL2/5-SOCS-box protein) E3ubiquitin-protein ligase complex which mediates the ubiquitination and subsequent proteasomal degradation of target proteins	Since SPSB3 is involved in protein degradation, maybe its downregulation signals reversal of apoptosis.
TRIB3	Disrupts insulin signaling. May play a role in programmed neuronal cell death	Upregulation may be a sign of improved neuroprotection.

Table 2. A selection of genes that were up or downregulated after treatment with the ESC cultured media. The analysis was performed using web portal GeneCards <http://www.genecards.org>.



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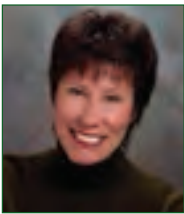
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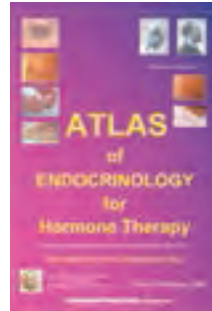
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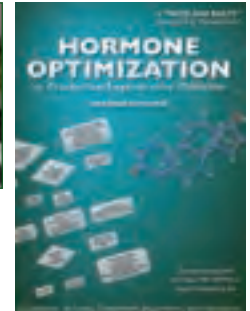
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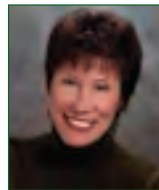
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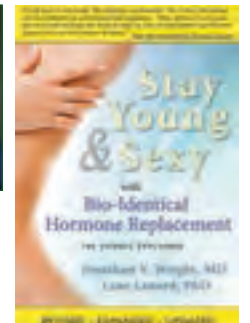
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A New Approach To Telomere Measurement

By- Dr. Marcelo Viegas, Ph.D.

From bacteria to humans all, living organisms are composed of cells which contain DNA (deoxyribonucleic acid), the molecules responsible for keeping the genetic information, the instructions for everything done directly or indirectly by the cells.

In eukaryotes (including man) the DNA molecules are linear and highly packed inside the nuclei of the cells along with a complex group of proteins forming structures known as "chromosomes." To protect vital genetic information, cells have to replicate their chromosomes before dividing into two daughter cells, and thus preserving these instructions from generation to generation. Chromosome replication is a highly accurate process that ensures faithful copies of the DNA sequence. There is, however, one exception: the

very tips of the chromosomes called "telomeres" which get shorter after each cell division (*Figure 1*).

In the scientific jargon, this limitation is known as the "end-replication problem" and derives from the intrinsic nature of the DNA replication mechanism which cannot completely replicate the ends of a DNA molecule (and hence, the chromosomal ends).

At the DNA level, telomeres are repetitive sequences at the ends of linear chromosomes that serve as essential protective structures that maintain the integrity of the chromosomes. The whole structure is capped by a dynamic protein complex called a "shelterin," which further protects the chromosomal ends by impeding chromosome fusion¹.

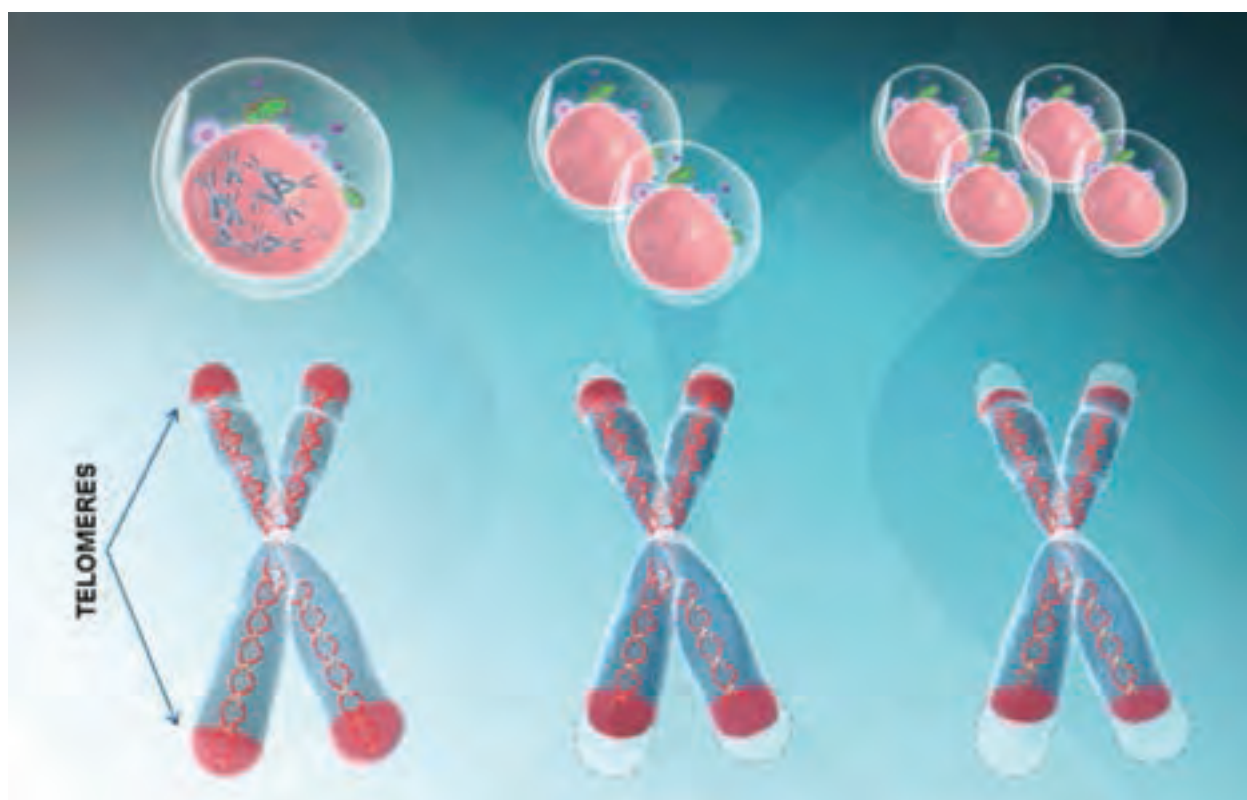



Figure 1 Liver detoxification pathways and supportive nutrients (used with permission from the Institute for Functional Medicine.)



It is believed that telomere sequences do not bear any genetic information but only support a structural function. Therefore, the progressive erosion of telomere ends does not imply any genetic loss. Nevertheless, telomeres are crucial for cells to live. So as cells replicate, telomeres shorten up to a point where the telomere length is critically short and the cells become senescent or apoptotic (i.e. they die). Therefore telomeres are like “biological timers” that count down after every cell division imposing a limit to the cell’s lifespan when they come to “zero”. This property of the telomeres links telomere length with aging, the time-dependent functional decline of the organisms until death².

Interestingly, telomere shortening and aging have some remarkable exceptions that actually reinforce the correlation between these two processes. There exists a special enzyme called “telomerase” whose function is to elongate telomeres, thus counteracting normal telomere attrition. This enzyme is synthesized only by a few cell types, mostly germ-cells, stem-cells and cancer cells. It is also expressed in the morula-to-blastocyst transition in embryos. The expression of telomerase in both germ-cells and early embryos explains why our children are born young and with the same life expectancy that we have despite our own age. Stem-cells also express telomerase albeit at low levels. In these cells, the presence of telomerase allows a high mitotic index. Yet, even these telomerase levels are not sufficient to completely counteract telomere shortening. As a consequence, although stem-cells can replicate and live longer than somatic cells do, they also end-up dying after a certain number of divisions. Cancer cells probably represent the most striking example of the correlation between telomere length and aging. Telomerase is highly expressed in over 80% of all adult cancers³. This explains why these cells do not exhaust their replication capacity. Thus, telomere length turns out to be a key factor in cell replication and aging.


In normal aging somatic cells though, as noted, telomeres shorten inexorably. Yet, usually cells age faster than they would if it were only for the end-replication problem. It is a common observation that certain people seem to age faster than others. It is as if the biological age of some individuals does not fully correlate with their chronological age. At the telomere level, there is increasing evidence that supports the idea that an unhealthy life style can accelerate the shortening process. For instance Valdes et al. carried out a study which concluded that obese women’s leukocytes had shorter telomeres compared to lean ones of the same age. In the same study smokers also had shorter telomeres than non-smokers and the relation between telomere shortening and smoking was dose-dependent. According to a second study⁵, sedentary behavior (lack of exercise) also accelerates telomeres shortening in leukocytes.

Even psychological stress seems to play a role on telomere length. Epel et al. compared healthy premenopausal women who were biological mothers of either a healthy child (“control mothers”) or a chronically ill child (“caregiving mothers”) who are subjected to significantly greater stress. While a direct comparison of these two groups did not show any significant difference in telomere length, the duration of the chronic stress (number of years spent as a caregiver) did have a significant correlation with telomere length. Indeed, within the caregiving group, the more years of caregiving, the shorter the mother’s telomere length was after correcting for the mother’s age⁶. Of greater concern, stress can also shorten telomeres in children according to a long-term study carried out in Romania that indicates that children who spent their early years in state-run orphanages have shorter telomeres than children grew up in foster care⁷.

All in all, these studies illustrate basically the same underlying concept; namely, that there are existing mechanisms of telomere attrition that work on top of the basal shortening given by cell replication. So the question becomes: can we do something to stop further telomere shortening?

There is mounting evidence pointing toward an affirmative answer. Intake of certain vitamins, antioxidants and minerals has shown a positive correlation with telomere length^{8,9}. Song et al. have demonstrated that duration of physical exercise correlates with telomere length. Exercise can reduce harmful fat and lead to reduced oxidative stress and preservation of DNA and telomeres¹⁰. Although not yet proved in humans, caloric restriction has a positive impact on telomere length in mice¹¹.

As appealing as these findings might be, a unifying model for the action of external factors on telomere length is still lacking. On one hand, some of these studies show only marginal statistical significance. On the other hand, there are also some publications that dispute the existence of any beneficial life habit on telomere health. For example, Shin et al. studied the changes of both oxidant and antioxidant levels with exercise training in obese middle-aged women. The association between telomere length and oxidative stress with exercise was also examined. While antioxidant enzyme activities were increased following long-term exercise training, the length of telomeres in leukocytes was not influenced by neither mid-intensity nor high intensity exercise stress¹². In yet another study, Kadi et al. compared telomere length in muscle of power lifters against that of a group with no history of strength training and reported no differences in mean telomere length¹³. Regarding low calorie diets, Smith et al analyzed skeletal muscle, fat, leukocytes, and skin of two groups of rhesus monkeys, one of them fed with a normal diet while the second one was calorie restricted. After controlling the individual’s muscle mean telomere length, differences between leukocytes and skin were significantly associated with age but not with calorie restriction¹⁴.



What is the cause of these discrepant results? Undoubtedly, one of the problems is the use of different biological models that precludes inter-study comparisons. Additionally, many times the studies are based on relatively small groups and while the results can be statistically significant, the statistical power of the test applied turns out to be small.

But on top of all these issues, the methodology used to measure telomere length is of paramount importance. In the vast majority of these studies, telomeres are measured either by TRF or qPCR. The TRF (terminal restriction fragment) assay is the application of the classical Southern-blot technique to measure telomere length. DNA is purified from cells and digested with one or more restriction enzymes that do not cut into the telomere sequence. After blotting, a smear representing the distribution of telomere lengths in the total cell population is visualized. The final outcome of this test is an estimation of the mean telomere length in that cell population. However results depend on the total DNA amount, DNA integrity and electrophoretic conditions. The latter is of critical importance and while it is possible to fix the conditions in advance, the resolution of the assay will vary depending on the telomere length of the population under study qPCR (quantitative polymerase chain reaction) is a biochemical technology to amplify single or a few copies of a piece of DNA several orders of magnitude. Due to its high sensitivity and high-throughput capability it has become the technique of choice to quantify gene expression and gene copy number. However, telomere amplification poses a challenge to PCR. Not only telomeres but also one or more single-copy gene has to be amplified to control for DNA load in the reaction. Since the telomere sequence is repetitive, partially degraded DNA samples will not affect telomere amplification; however, the amplification of the genes used for normalization will be impaired proportionally to the percentage of degradation of the samples. Consequently, telomere length measurement by PCR heavily relies on sample quality which is difficult to control in practice. Moreover, due to the repetitive nature of telomeres, several amplicons are amplified per target during the PCR reaction; additionally, PCR primers cannot fully complement the telomere sequence for the same reason. Both of these factors imply a low overall amplification efficiency which adds to the error in the measurement of telomere length.

The hurdles discussed so far are purely technical. There is, nevertheless, a more conceptual difficulty associated with TRF and PCR techniques. The outcome of both methodologies is a mean telomere length. There is a third technique called qFISH (Quantitative Fluorescence In Situ Hybridization) that provides the advantage of detecting individual telomeres within a cell (Figure 2a). A number of studies based on qFISH showed that the actual distribution of telomere lengths in cells have

a distribution similar to the one depicted in figure 2b (see ^{15,16} as examples). Far from being symmetrical, the distribution of telomere lengths is positively skewed. That implies that the mean is not a good estimation of the telomere length distribution for any small fluctuation in the tail of the distribution will affect the reading of the mean telomere length quite significantly.

Therefore the use of the median rather than the mean of the telomere length distribution should be preferred as it is a more robust and a representative estimator of telomere length. In addition, every study should include histograms like the one shown in figure 2b to be able to assess the quality of the data. A curve that strongly deviates from that of the figure could imply poor data (i.e. low number of telomeres tested) or some kind of artifact during the protocol. On the other hand, the whole distribution of telomere lengths is tremendously more informative than just one estimator. It is possible to analyze the percentage of short telomeres or the length of telomeres under a certain percentile to find effects on telomere length, not necessarily perceived by the median value. Moreover, the curve allows an analysis of the data dispersion as measured by the standard deviation. For instance, too high of a standard deviation could mean telomere lengthening through recombination rather than telomerase, which eventually could indicate deficient telomerase activity.

Life Length Inc. has been commercializing a powerful high-throughput qFISH test for a couple of years. This is the only company in the world that measures both short telomeres and the median telomere length in cells. Now the company has announced the launch of a new version of the test that also includes the whole frequency distribution of telomere lengths. The novel test will undoubtedly aid researchers in designing more robust studies to assess the impact of their subject matter on telomere length.

CONCLUDING REMARKS

Telomere biology is central to understanding aging. Telomere shortening (and aging) seems unavoidable at the present. However, some healthy behaviors may retard this process. More precise studies will clarify the impact of diet and exercise on our telomeres. Now a new commercial tool based on qFISH has emerged that will definitively assist in these investigations.

CONFLICT OF INTEREST

Dr. Viegas is Chief Scientific Officer at Life Length, Inc. (www.lifelength.com), Madrid, Spain.

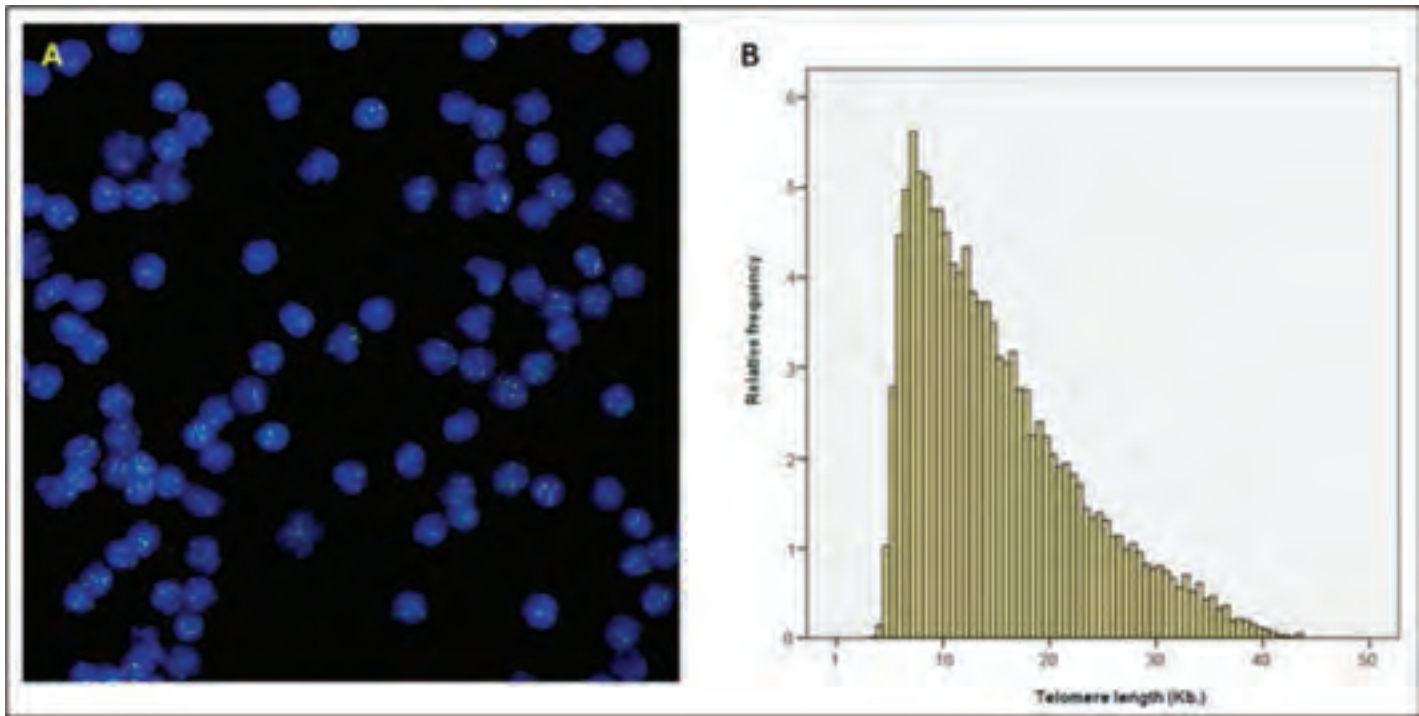


Fig. 2: qFISH image and frequency distribution of telomere length in cells. a) Image of qFISH done on purified lymphocytes. Nuclei were stained with DAPI (blue) and telomeres are visualized in green. b) Example of the distribution of telomere lengths in human lymphocytes.

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- **Discuss** the process of detoxification involving the mobilization, biotransformation and elimination of toxicants of exogenous and endogenous origin.
- **Review** how we can better cope with the toxicity stemming from the environment.
- **Discover** the important role of the ONDAMED Technology and its unique emotional Feedback to identify toxic, dysfunctional, and inflamed tissue enabling the practitioner to offer a focused therapeutic stimulation to these areas that potentially are the root cause of symptoms and chronic disease.
- **Learn** how ONDAMED helps uncover and stimulate emotional trauma/shockinduced toxicity stored as cellular memory.
- **Review** cases including Immune Deficiency Disorders, Allergies, Cardiovascular Disease, Depression, and even Blindness.



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How Nature-Throid® saved my life.



I was first diagnosed with hypothyroidism after my third child in 2009. I felt misunderstood by everyone, even my doctor. I was suffering from exhaustion, a sense of cold, weight gain and depression. Nature-Throid® not only saved my life, it helped me feel sexy again. Most synthetic thyroid treatments contain only T4 hormone. Nature-Throid® contains both T4 and T3 hormones, providing a more natural response. It's also hypoallergenic and delivers a consistent hormone replacement. Nature-Throid® is my lifeguard. Learn how it can become yours at nature-throid.com

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Your Medical Practice, Your Way.

What a journey it's been since I first started my fellowship training in anti-aging and preventive medicine in 2004. The fellowship training I received gave me a unique opportunity to help patients in ways I never dreamed of before. The fellowship also put me in an amazing position to start a cash-based practice and get away from the hassles of insurance billing and Medicare/HMO reimbursement rates.

A few questions I had for myself before I started my practice were, "Will I be able to make a nice living practicing this kind of medicine?" and "How will I find my patients and how will they find me?" and "How will I operate, build and market my practice AND see patients?"

Enter BodyLogicMD

When I first looked into joining BodyLogicMD, the company was fairly new with only a few practices in the network. I was nervous, yet excited, about a company that could possibly handle all the business worries I had.

First, BodyLogicMD started me off with a comprehensive business plan. What would have taken me months to develop on my own was readily available for me to implement immediately into my practice. Having a business "map" to follow when starting a new practice is key in helping any business succeed. Still, even if it looks great on paper, how can anyone be sure it actually works?

I took the chance, and was surprised to have my practice up-and-running within a few short months. It was a partnership every step of the way; from finding an office space to negotiating the lease, from finding office furniture to gaining an office staff. I now have a virtual staff that is in charge of finding and maintaining my patient base. Many different marketing strategies are regularly incorporated on my behalf and all prospective patients contact my patient services staff (who I don't pay for) to book their appointments. Finally!...A practice that consists of only seeing patients while not having to worry about the various business hassles.

Since opening my practice nearly six years ago, BodyLogicMD has duplicated my success for approximately 60 other physicians, while still improving their process.

Ongoing Education for Ongoing Opportunities

Other advantages of becoming a physician of the BodyLogicMD network are the numerous educational opportunities. BodyLogicMD physicians participate in educational webinars, hosted by the elite in preventive and functional medicine, as well as presentations

hosted by experts in the field of technology and lab testing, which is constantly changing in the preventive arena. These free webinars are exclusive to BodyLogicMD physicians and are an invaluable resource to continuing my education beyond the fellowship. An elite group of strategic partners also help to open doors for physicians of the BodyLogicMD network because they know we are the highest trained doctors of the most successful anti-aging network in the world.

Another tremendous benefit is the annual business retreat hosted by BodyLogicMD and their lab, pharmacy and supplement partners. There is nothing quite like spending 3-4 days networking with all of my BodyLogicMD colleagues discussing the "biology of business" and new strategies to help me grow my practice.

A Network of Experts at Your Fingertips

One aspect I have found to be very beneficial to my practice is being able to easily communicate with my BodyLogicMD colleagues. Each fellowship-trained doctor is only an email away. We can contact one another on a daily basis to review challenging cases together or just get input from fellow physicians, who are specially-trained in different areas of medicine. Because all BodyLogicMD physicians are required to be board eligible in a field of medicine before completing the fellowship and A4M board certification, the network of physicians I work with are incredibly knowledgeable and a great resource.

The network of specially-trained physicians isn't the only support I have for my practice. BodyLogicMD also provides me with excellent marketing support. The marketing team provides me with materials specifically designed for my practice. Many of us have been published and quoted in countless local and national magazines and newspapers, best-selling books and influential websites like Foxnews.com, Oprah.com and national television programs including Dr. Oz.

The Time to Join is Now

BodyLogicMD has the know-how to put you into a successful bioidentical hormone replacement therapy and functional medical practice. You can purchase your own BodyLogicMD practice or join an existing practice. If you're interested in working with the most highly-trained and experienced physicians in the country, visit join.bodylogicmd.com or email Julie Seymour at jseymour@bodylogicmd.com.



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**Dementia: You
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6:30 pm – 8:30 pm**



Eric Braverman, M.D. is the Founder and President of PATH Foundation NY, current Assistant Professor of the Department of Psychiatry at the University of Florida College Of Medicine and the McKnight Brain Institute and previous Assistant Professor in the Department of Neurosurgery at Weill Cornell Medical College during 2008-2012. He is also Director of PATH Medical.

A majority of individuals have an average brain speed of 300 milliseconds (msec) + their age (i.e., a 40-year-old has a brain speed of 340 msec) or P300, the test that measures brain age and waves. Individuals with brain speeds slower than 340 msec show progressive signs of cognitive decline and low brain metabolism on PET scan (Positron Emission Tomography). A healthy brain is at its peak performance processing information at 300-320 msec. Loss of even 40 milliseconds correlates to lowered brain performance and cognitive decline. The loss of brain processing speed is the best marker of memory and attention decline and the P300 is as easy to perform as an EKG

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The Next Generation of Omega-3 Anti-Aging Supplements



Everyone, regardless of their age, current state of health, or gender, needs omega-3 supplementation as their first line of defense in anti-aging. We know that the all too familiar diseases that occur with aging, such as Alzheimer's disease, cardiovascular disease, and osteoarthritis are all caused by inflammation. An informed decision for good quality omega-3 supplementation can control this inflammation and protect your body from the ravages of these disorders. However, the trick is to understand the qualitative differences between Omega-3 supplements, in order to make the most informed decision for yourself, your family, and your patients. All omega 3 supplements are definitely NOT created equal and yet they are of vital importance to all of us.

Omega-3/Omega-6 Balance

Omega-3 supplementation is critical for metabolism function and balance. Our bodies were designed by nature to have an equal balance of essential fatty acids from our diets to protect blood vessels, the heart, joints, and nerves from inflammation. There are two important classes of fatty acids which control the inflammatory process in the body: Omega 3 fatty acids from marine oils, walnuts and flax seeds, which reduce inflammation; and Omega 6 fatty acids from corn, soy, vegetable oil, and livestock, which trigger inflammation. When these two types of fatty acids are balanced in your body, the immune system is able to do its job and protect the body from infection and injury. Uncontrolled or exaggerated inflammation can cause pain, injury, and disability in virtually every organ system throughout the body. Table 1 outlines some of the degenerative diseases that are rooted in inflammation.

Table 1:

Inflammation is the Basis of Most Degenerative Disease

- Asthma
- Alzheimer's disease
- Cardiovascular disease
- Metabolic syndrome
- Osteoarthritis
- Rheumatoid arthritis
- Inflammatory bowel disease
- Dysmenorrhea
- Delayed onset muscle soreness

Over the past forty years, changes in our food manufacturing practices have resulted in huge quantities of omega-6 fatty acids in our food supply, in the form of hydrogenated vegetable oils, high fructose corn syrup, and genetically engineered corn and soy products. These trans fatty acids decrease the amount of omega-3 fatty acid available for metabolism. At the same time, our consumption of Omega-3 fatty acids has dramatically reduced, creating a dangerous imbalance of omega-6 to omega-3 fatty acids in the body.

The ideal ratio of omega-6 to omega-3 fatty acids is 1:1. Studies show that the majority of us have a 20:1 ratio of omega-6 to omega-3 fatty acids, which results in chronic inflammation. This chronic inflammation is causative in most of the prevalent degenerative disorders that occur with aging, including cardiovascular diseases, osteoarthritis, autoimmune disorders, diabetes, cancer, and pulmonary diseases. In the secondary prevention of cardiovascular disease, a ratio of 4:1 omega-6 to omega-3 fatty acids was associated with a 70% decrease in total mortality (Lyon Heart Study).¹ A ratio of 5:1

has been shown beneficial for those suffering from asthma and respiratory distress.³ Studies by James and Cleland showed the benefits of maintaining an omega-6 to omega-3 fatty acid ratio of 3-4:1 for the treatment of rheumatoid arthritis.⁴ This promising research substantiates the premise that nutritional medicine does indeed impact degenerative disease. This in turn, makes the quality of the omega-3 fatty acid supplement a determining factor in its effect on disease. From there, the source, purity, and fatty acid content are important considerations. The omega-3 supplement marketplace is fraught with variability in terms of these characteristics, as well as the overall health benefit.

Source and Purity

A majority of omega-3 supplements are derived from fish oil, although this may prove not to be the best source for a variety of reasons. Many fish used to produce fish oil supplements live in polluted waters and eat other contaminated fish, resulting in the accumulation of harmful toxins, such as heavy metals, polychlorinated biphenyls (PCB's), pesticides, and radiation which may not be removed during the fish oil manufacturing process. This poses a potential health risk, and is the main reason pregnant women are advised to limit fish intake and avoid omega-3 supplements. Additionally, most fish oil supplements are not created from fish fillets, but rather the leftover parts such as the scales, eyes, and fins, resulting in an inferior product.

Recently, there has been a preponderance of omega-3 supplements derived from krill, which are tiny crustaceans at the bottom of the food chain. While krill do not consume other fish and compound toxins as with other marine life, there is controversy regarding the eco-responsibility of harvesting krill grown in open water, as it is the primary food source for the ocean's inhabitants. A number of US retailers refuse to carry krill oil due to this sustainability issue.

The most important and healthful source of omega-3 products comes from the green-lipped mussel oil extract. These mussels are farmed in a special part of New Zealand, known as the Marlborough Sound, an area known for its pure waters. The mussels feed on antioxidant-rich plankton exclusive to these waters for eighteen months until they reach maturity. These farming conditions produce the purest, natural source for omega-3 fatty acids, free from heavy metals and solvents.

Potency and Bioavailability

Another important factor in evaluating the quality of an omega-3 supplement is the manufacturing process used to extract the marine oil. Marine oils are extracted by three processes: cold press, distillation, or supercritical fluid extraction. The first two processes result in bound forms of essential fatty acids, such as triglycerides or

ethyl ester forms. These bound forms need to undergo multiple metabolic processes before they can be absorbed efficiently in the body. Green-lipped mussels are harvested, their meat is freeze dried, and their oil (PCSO-524™) is extracted using a patented process known as carbon dioxide super critical fractionation, which dissolves nonpolar lipids from the freeze-dried mussel powder using a combination of pressure and low temperature.

This process is important for three reasons. First, it removes any impurities and heavy metals from the oil, making it toxin free. Second, it removes the marine proteins from the oil, resulting in a reduced risk of allergic reactions. Third, and most importantly, it delivers the oil in the free fatty acid form, which allows for easy absorption into the bloodstream and use in the body. Large doses of traditionally manufactured fish oils can cause both bleeding problems and increases in harmful cholesterol, in addition to digestive challenges. When analyzing product for potency, free fatty acid content is relevant.

Many supplement manufacturers compare fish oil supplements based on their EPA (eicosapentaenoic acid) and DHA (docosahexanoic acid). Many market the superiority of their product based solely on the amount of EPA/DHA without regard to the free fatty acid content or side effects associated with the total dose of oil.

EPA and DHA content have received the most of the spotlight because they are both essential fatty acids, meaning they are substances that are necessary for optimum health that must be obtained from dietary sources. This trend originated over thirty years ago with the discovery that Greenland Eskimos, who despite consuming large amounts of fatty fish, had fewer heart attacks and strokes. At that time, the cellular pathways for this observation had not been fully elucidated, so it was assumed that EPA and DHA alone accounted for a reduction in cardiovascular risk. It is now known that there are other vital, biologically active fatty acids in marine oils involved in cardiovascular protection, as well as many other health benefits. Analysis of the green-lipped mussel oil (PCSO-524™) reveals thirty different fatty acids that work synergistically to reduce inflammation 100 times better than traditional fish oils. PCSO-524™ contains EPA and DHA, as well as twenty-eight more fatty acids that create a significant anti-inflammatory effect, modifying fifty-three inflammatory pathways.

Omega-3 fatty acids exhibit natural anti-inflammatory properties as a COX-2 (cyclooxygenase) and LOX-5 (5-lipoxygenase) pathway inhibitors in lipid metabolism. These enzymes catalyze the oxygenation of polyunsaturated fatty acids (PUFA), preferably arachidonic acid, to form biologically active prostaglandin and

leukotriene metabolites, which regulate body functions such as heart rate, blood pressure, and clotting, and immune function and inflammation.

Supplementation with omega-3s is one of the most crucial preventative health measures we can take, but a pure, potent, and complete supplement is key to optimal health. When choosing an omega-3 supplement, it is important to know the amount of essential fatty acids, such as EPA and DHA, are available in the free bioactive form.

More popular fish oil supplements contain fatty acids in the triglyceride form, requiring additional metabolic steps in order for the micelles (which contain the free fatty acids) to be freed from the triglycerides so that the free fatty acids can cross the intestinal barrier. Not only does this take longer, but a majority of the free fatty acids are lost along the way, with only a small amount making its way to its final destination. Much larger quantities of omega-3 oil are required to get the same benefit.

The higher concentration of free fatty acids in green-lipped mussel oil PCSO-524™ makes them more readily available in the bioactive form, so they do not need to be metabolized or “digested” by the body. This means the supplement is more effective in smaller doses when compared with other fish oil products. So in most instances, it is not how much omega-3 fatty acid the supplement contains, but how readily available it is to your body that determines effectiveness. More free form omega-3 fatty acid translates into more benefits and faster results.

Clinical Benefits

Inflammatory Joint Disorders

Omega-3 fatty acids are very effective in treating the pain and symptoms of inflammation. It helps with pain and swelling associated with arthritic joints and back pain—the types of conditions that cause disability and difficulty enjoying what life has to offer.

Osteoarthritis affects 27 million people in the US and its treatment costs amount to \$42.3 billion per year. Osteoarthritis begins as a small injury in the joint that creates inflammation. That inflammation degrades the cartilage in the joint, making it unstable, causing pain. Omega-3 fatty acids affect the same chemical pathways as NSAIDs (non-steroidal anti-inflammatory drugs), making it a natural anti-inflammatory but without the risks and side effects of NSAID medications. Studies conducted with omega-3 fatty acid from *Perna canaliculus* (PCSO-524™) were shown to reduce joint stiffness and pain, increased grip strength, and improved walking pace.⁵

Cardiovascular

A potent omega-3 will also work on the subtler, silent diseases such as cardiovascular disease. Researchers propose that omega-3 fatty acids influence cardiovascular disease via anti-arrhythmic properties; regulating blood pressure; lowering triglyceride levels; elevating HDL levels; stimulating endothelial-derived nitric oxide production that increases vasodilation; decreasing platelet aggregation; increasing prostacyclin-2 production; and decreasing pro-inflammatory eicosanoids.⁶

Arrhythmia, or irregular or abnormal heart rate, affects nearly 10 million Americans and is the leading cause of sudden cardiac death. Clinical evidence showed that after 3.5 years, subjects in the Italian GISSI prevention trial of post-myocardial infarction, patients receiving 850 mg/day of omega-3 fatty acid supplementation experienced a 20% reduction in overall mortality, and a 45% reduction in sudden death.⁷

Asthma

Asthma is a chronic inflammatory disease of the airways mediated, in part, by leukotrienes and other lipid mediators. Experimental studies have shown that the lipid extract of the New Zealand green-lipped mussel PCSO-524™ *Perna caliculus*, is effective in inhibiting 5-lipoxygenase and cyclo-oxygenase pathways responsible for the production of eicosanoids, including leukotrienes and prostaglandins. Forty-six subjects with atopic asthma received two capsules of PCSO-524™ (50 mg omega-3 polyunsaturated fatty acids and 100 mg olive oil) or placebo (150 mg olive oil) twice daily for eight weeks. Results showed a significant decrease in daytime wheeze, the concentrations of exhaled hydrogen peroxide, and an increase in morning peak expiratory flow.⁸

Inflammatory Bowel Disease

More than 1.4 million adults in the US have reported to suffer from some form of inflammatory bowel condition. Increasingly, therapeutic interventions for gastrointestinal disease are based on the role of different inflammatory mediators and reactive oxygen and nitrogen-free radicals. Nitric oxide activates COX-2 enzymes. Prostaglandin E2 negatively modulates nitric oxide synthase induction, effecting gastric mucosal integrity via the arachidonic acid cascade. Prostaglandins have pro-inflammatory and anti-inflammatory effects on the immune system. Polyunsaturated fatty acids and eicosanoids may modulate inflammatory processes and immune cells involved in irritable bowel disease as LOX inhibitors, inducible nitric oxide synthase activators, and natural antioxidants.

In an animal model of inflammatory bowel disease, researchers found that *Perna caliculus* PCSO-524™ treatment, when compared to fish oil, improved gastrointestinal and digestive inflammation.⁹

Delayed Onset Muscle Soreness (DOMS)

Delayed onset muscle soreness is a common condition that presents as a combination of muscle pain, soreness, or stiffness 24 to 72 hours after exercise. It is thought to be caused by microtrauma to the muscle fibers during exercise, and more specifically, eccentric muscle contractions. Little is known about the underlying physical trauma that causes the pain. Distance runners given 400 mg PCSO-524™ daily for eleven weeks experienced reduced pain symptoms compared to placebo following a 30 km run. This effect was greater in less experience runners.¹⁰

Omega-3 for Anti-Aging

A novel marine oil from the green-lipped muscle of New Zealand, PCSO-524™ marketed as OMAPREM strictly to the medical profession, has been found to be the purest and most potent omega-3 fatty acid to reduce inflammation via COX-2 and LOX-5 inhibition and to improve the aforementioned specific disorders, as well as overall quality of life. OMAPREM provides these benefits in a natural, pure, potent supplement contained in a tiny capsule that is easy to swallow and is free from the side effects of the average fish oil product. PCSO-524™ has been shown to reduce pain and swelling as effectively as many anti-inflammatory medications (such as non-steroidal anti-inflammatory drugs, or NSAIDs), without the side effects and drug interactions associated with these products. NSAIDs cause approximately 16,500 consumer problems yearly, due to their tendency to increase gastric bleeding as well as renal and hepatic function problems. Analysis has shown that there are up to twenty-two times higher levels of omega-3 fatty acids in the PCSO-524™ green-lipped mussel oil extract than in regular fish oil.

An omega-3 fatty acid supplement is an important step for optimal health. This valuable, natural substance helps prevent many of today's medical conditions by controlling inflammation. The ability to rebalance our fatty acid metabolism works to prevent the onset of osteoarthritis, cardiovascular disease, asthma, and other afflictions caused by inflammation. A pure, potent viable omega-3 supplement should be a mandatory component of the anti-aging armamentarium, and the marine oil from the green-lipped mussel provides an effective and safe source of fatty acids to combat inflammation.

Author: Sharon McQuillian, MD is a board certified physician who has been practicing Anti-Aging and Aesthetic Medicine for over 14 years. She is current medical director of the Ageless Aesthetic Institute, Ageless Regenerative Institute, and director of the A4M Aesthetic Fellowship.

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EXHIBITORS DIRECTORY

Product Announcements	75
-----------------------	----

Exhibitors by category

Aesthetic Medical Treatment	99
Alternative Therapies	101
Associations/Education	104
Compounding Pharmacies	105
Computers/EMR	108
Cosmeceuticals	112
Dental	117
Dermatology	117
Diagnostic Service/Laboratories	118
Hair Rejuvenation	125
Holistic Medicine	126
Hyperbaric/Oxygen Therapies	127
Insurance/Finance/Practice Management/Marketing	127
Med Spa/Equipment	130
Medical Equipment/Supplies	134
Medical Publications/Directories	138
Mesotherapy/Hormone Therapy	139
Miscellaneous	140
Nutraceuticals	141
Pharmaceuticals	157
Sports/Fitness/Physical Therapies	158
Stem Cell Therapies	158
Web Design	159
Weight Management	159

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Supplement Facts

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Servings Per Container 60, 120, 180

1 soft gel capsule contains	Amount Per Serving	% Daily Value
Calories	15	
Calories from fat	15	
Total fat	1.5 g	2%*
Cholesterol	<5 mg	<2%*
Total Omega-3s (as Triglycerides)	950 mg	**
EPA (Eicosapentaenoic Acid)	430 mg	**
DHA (Docosahexaenoic Acid)	390 mg	**
Omegas-3s (additional)	130 mg	**

* Percent Daily Values are based on a 2,000 calorie diet.
** Daily Value not established

ID# 440060 60 Soft Gel Capsules

ID# 440120 120 Soft Gel Capsules

ID# 440180 180 Soft Gel Capsules

SUGGESTED USE: 1 soft gel capsule per day or as recommended by your health care professional.

These statements have not been evaluated by the Food and Drug Administration. This product is not intended to diagnose, treat, cure, or prevent any disease.



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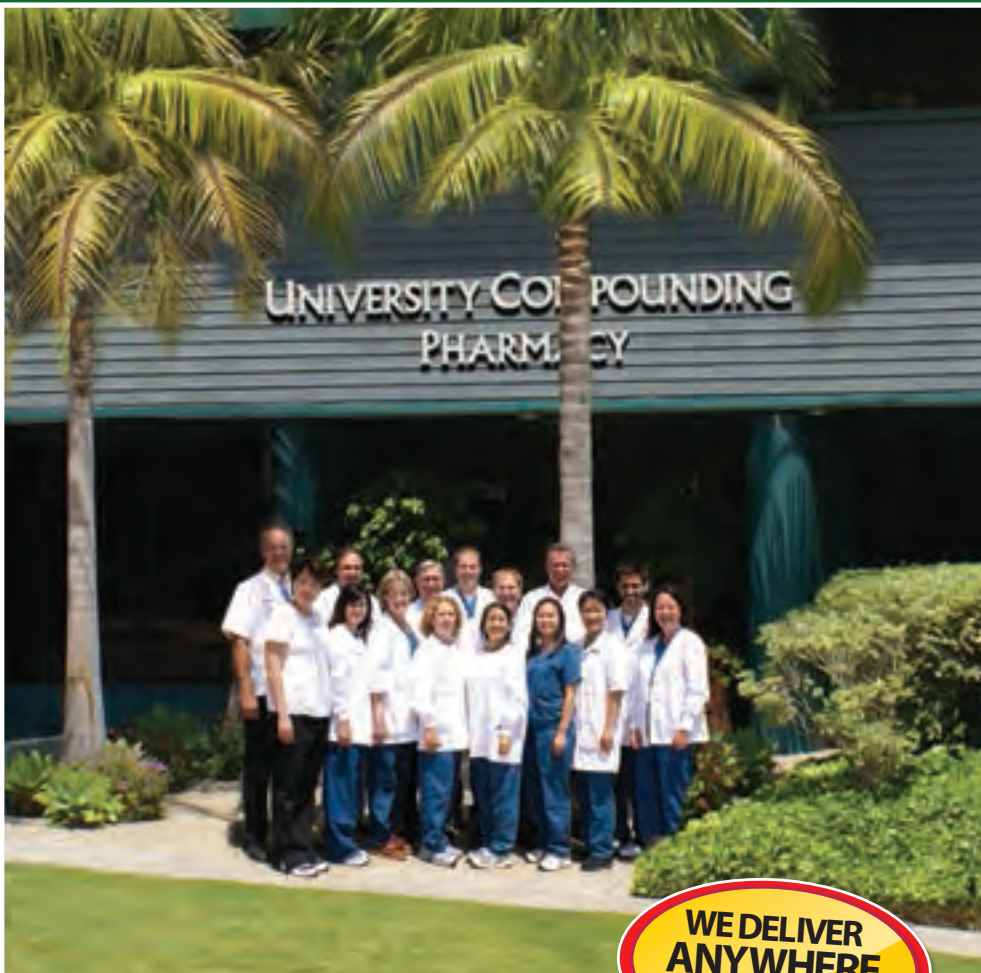
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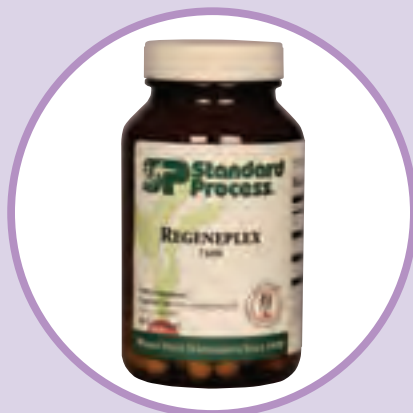
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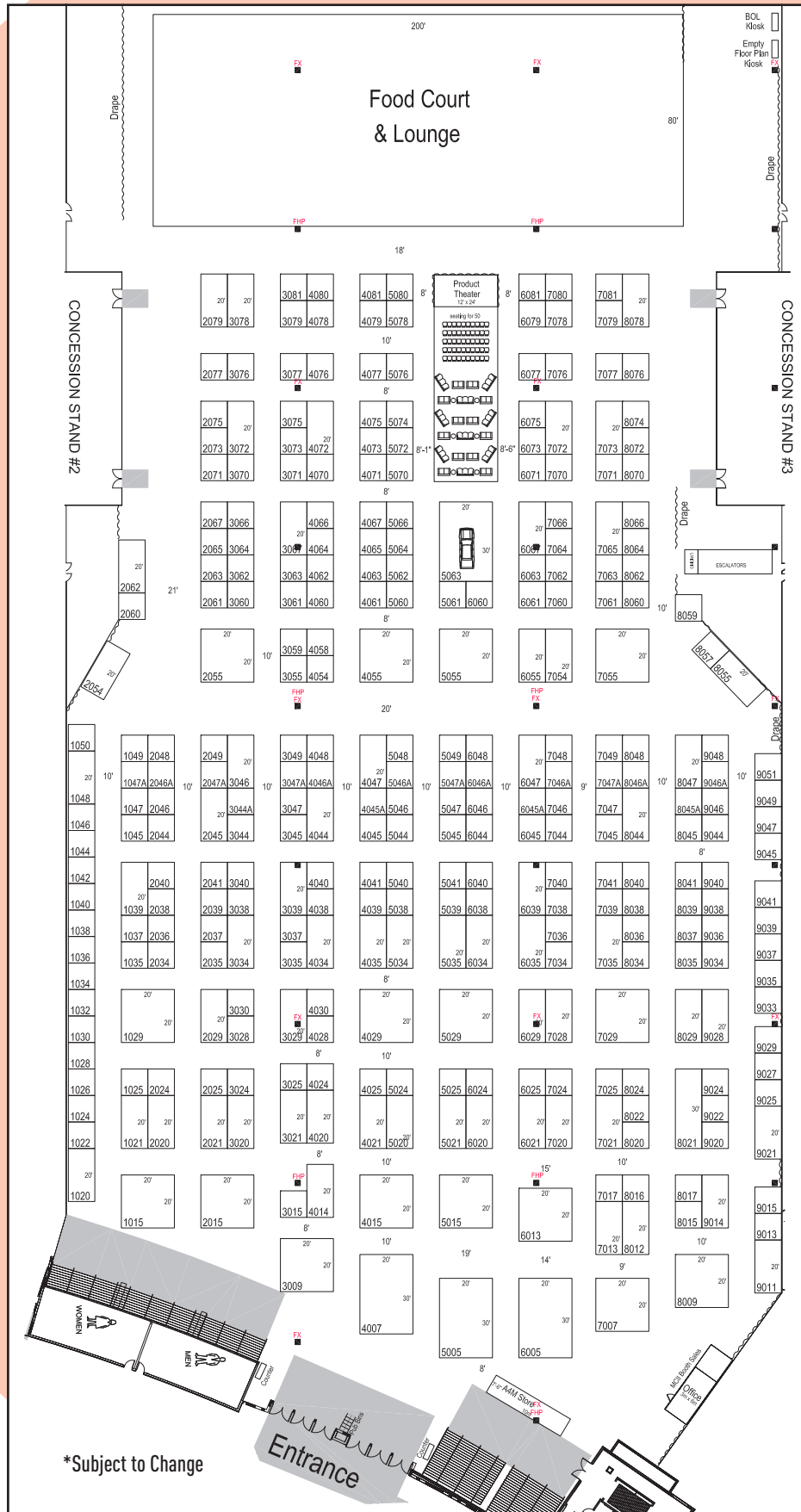
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A Fashion Hayvin, Inc.	2060	Boston Heart Diagnostics.	9045
ABL Medical.	4044	BrainMaster Technologies, Inc.	2061
Abrams Royal Compounding Pharmacy	5015	BTL Aesthetics	5074
A.C. Grace Company	7040	Calvin Scott, Inc.	7038
Access Medical Labs, Inc.	2055	Canada RNA Biochemical, Inc.	8060
Advanced Naturals	3044	Canfield Imaging Systems	6048
Ageless Regenerative Institute	3055	Cascades Clinical Systems	7070
The Ageless Secret	9014	Cell Science Systems, Corp.	7013
Ageology	3038	Central Drugs Compounding Pharmacy	8009
Aidan Products	8022	Chi's Enterprise, Inc.	4041
Ajinomoto North America	6055	ClarityRx Clinical Skin Care	7078
Allergy Research Group®	5035	Clear Mind Center	5024
Alletess Medical Laboratory	1035	Clearlight Infrared Saunas	6067
AlmsBio	5064	Cleveland HeartLab, Inc.	5066
American Biotech Labs	4047	CNS Vital Signs	1044
American Custom Compounding Pharmacy	7031	College Pharmacy.	4040
American Express OPEN	3078	Colonic.net	3039
American Healthcare Foundation	9037	Complementary Prescriptions	4007
American Herbal Labs / Nano Vita Water.	4025	CompliMed	1045
American Metabolic Laboratories	8020	CryoProbe - US	5041
American Pharmaceutical Ingredients	7031	CryoSurgery, Inc.	7048
Amiea Med, Inc.	3060	CS Health, LLC	3059
AML Diagnostics	8012	CustomVite	7020
AnazaoHealth Corporation	4055	Cutting Edge Aesthetics, LLC	2062
AnteAGE MD	4070	Cynosure	3049
APS Pharmacy.	6061	Cyrex Laboratories, LLC.	3034
Argentyn 23 by Natural-Immunogenics Corp.	2025	DaVinci Laboratories	3040
ARKRAY, Inc..	1046	The Delgado Protocol for Health	9041
Artisan Bistro Pro	6035	Demandforce®	5046
ASEA Redox Signaling Molecules.	2067	Derme & Co. / Physiodermie8024, 8029
Axia Medical Solutions	3071	Deseret Biologicals, Inc.	8040
Bank of America Practice Solutions	7060	Designer Drugs	4048
BerryBreeze	7056	Designs For Health	2029
Bio-Botanical Research, Inc.	4060	Diplomat	6034
Bio-Tech Pharmacal, Inc.	6039	Double Helix Water	9028
Biologix Hair, Inc.	5070	Douglas Laboratories	4029
BioMat Wellness Center	3067	Dr. Emma's Corporation	6046
Bionutrigen Co., Ltd.	7045	Dr. Fuji / ACIGI.	3029
BioPharma Scientific	5025	Drug Crafters Compounding Pharmacy	4066
BioProtein Technology	1024	Dunwoody Labs.	9022
BioRegenerative Sciences.	6060	Eclipse Aesthetics.	4078
BioSpace, Inc.	7025	Equipmed USA / Dermapen World	2054
Bios Rx, Inc.	1028	Elsevier	8066
BioTe Medical	4054	Emage Medical	7066
Biotics Research Corp.	1025	eMedical Fusion, LLC	7065
BodyLogicMD.	6013	Enhancement Medical LLC	5080
Boiron.	8045	enVy Pillow Co.	1026



THE FUTURE OF MEDICINE TODAY

EXHIBITOR Index

EXHIBITOR	BOOTH #	EXHIBITOR	BOOTH #
Erchonia Corporation	8044	Itamar Medical	9020
Essential Formulas	6063	Jan Marini Skin Research, Inc.	2035
Essential Pharmacy Compounding	3021	Kirkman Group, Inc.	8036
Excelerol	4071	KMI Diagnostics.	8055
Fairway Physicians Insurance Co.	8037	Korr Medical Technologies	4034
Foothills Professional Pharmacy	8064	Krave Blocker	1030
Forever Health	7073	Labrix Clinical Services	3045
FotoFinder Systems, Inc.	9047	Lenyosys / Nestatek.	7061
Freedom Gold Group	8070	Liability Insurance Solutions, Inc.	5062
Freedom Pharmaceuticals	4061	Life File LLC	1038
Fry Laboratories, LLC	3079	Life Length	7017
Gavée Gold	3035	Lifeline Skin Care	3066
Genova Diagnostics, Inc.	4015	LifeWave	1020
Gentech Pharmaceutical	1021	Liquid Smile.	9011
German Society for Thymus Therapy.	3064	Living Younger Preventive-Aging Medicine & Business System	8034
Global Compounding Pharmacy	7072	LivOn Labs	7041
Global Life Rejuvenation	8016	Lumiere Medical, Inc.	6079
Green PolkaDot Box	7054	MarcPro.	8078
Hair Science, LLC	9027	MD-Ware Software	4030
Hallandale Pharmacy	8021	Med-Chem Labs, Inc.	8038
Happy Hormone Cottage	5038	Medaus Pharmacy	6005
Harbor Compounding Pharmacy, Health & Wellness.	7035	MedEsthetics Magazine	7039
Harmonic Medical Devices, LLC.	9025	MedicalMine, Inc.	2073
Harvest Technologies Corp.	2020	Men's Wellness Centers.	9051
Hawaiian Moon.	5048, 7071	Meridian Valley Lab.	4028
Health & Beauty Connection	4075	Metagenics, Inc.	5029
Health Diagnostic Laboratory, Inc.	1029	Michael Marcus Cosmetics	7044
HealthSpanMDs.	9046	MitoQ Ltd.	2041
Healthy Habits Management Co.	4080	MorNuco & Hanua Holistic Care	8080
Heartland Products, Inc.	4058	MushroomScience	9021
Hevert Pharmaceuticals, LLC	2075	Mushroom Wisdom, Inc.	5049
High Tech Health International, Inc.	9024	Natural Partners, Inc.	9038
Hormonal Clarity Diagnostics (HCD)	2038	Natural Pharm Source, LLC	3072
Hormone Treatment Centers of America	9039	Nature-Throid.	6021
HydraFacial MD®- Edge Systems LLC	5044	Neogenis Labs	7021
i-Lipo / Chromogenex.	4065	NeoGraft	4063
ICA Health.	4045	Nerium International	9048
Identity HRT / Afton Medical	2037	NES Health	7049
Ilhwa NA, Inc.	1032	NeuroScience, Inc.	7036
illumiWave	4039	Newtropin, Inc.	9035
Incredible Marketing	5078	NEW YOU MEDIA	5055
InfiniteAloe	9034	Next Healthcare, Inc.	9040
Infinite Therapeutics	9044	Novavit, Inc.	1036
InMode	2040	Novus Optimum	7024
InnoVision Professional Media	3062	NuMedica, LLC	3020
International Anti-Aging Systems (IAS)	8048	NuSkin / Pharmanex	7055
Isagenix, International	8059	Nutritional Frontiers	3070



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EXHIBITOR	BOOTH #	EXHIBITOR	BOOTH #
Oceanus Insurance Company	8072	Standard Process, Inc.	2024
Oligoscan International.	6081	Stem MD	8017
OMAPREM	5055	Storey Marketing	2048
Ondamed	2015	Sun Chlorella USA.	3025
Optimum Hormone Balance	1049	T.A. Sciences, Inc.	7007
Ortho Molecular Products, Inc.	5020	Tavger Ltd.	1050
OxyHealth, LLC	2021	Terra Biological, LLC	4064
Pacific Herbs	9029	Texas Biostetic Instruments.	7046
Panasonic Healthcare Company of North America.	4072	The Global Stem Cell Group	8015
Park Compounding	6024	Therasauna	2039
PATH Medical	4014	Thermi Aesthetics.	5072
Patient One MediNutritionals	3028	Thorne Research, Inc.	7079
PCCA	1015	TimeSlip Solutions / RG-Cell®	6071
Perigee Medical.	8062	TouchMD	4079
Pharmacy Solutions.	6073	True Star Health Portal	4073
PhysioAge Systems	6029	United Pharmacy	3047
Precision Analytical, Inc.	9015	Universal Healthcare Lending.	2081
Prevention Pharmaceuticals, Inc.	2063	University Compounding Pharmacy	5005
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Pure Encapsulations	6040	Vitamix	2044
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Quincy Bioscience.	6038	Wellesley Therapeutics, Inc.	5060
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Researched Nutritionals	6044	XYMOGEN.	6020
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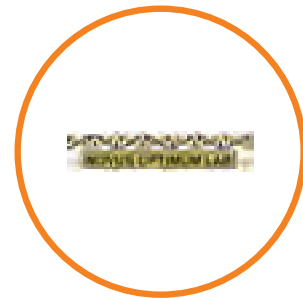
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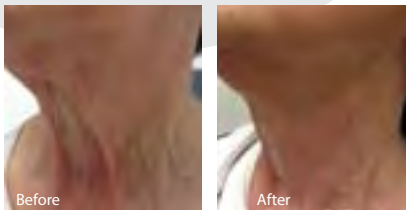
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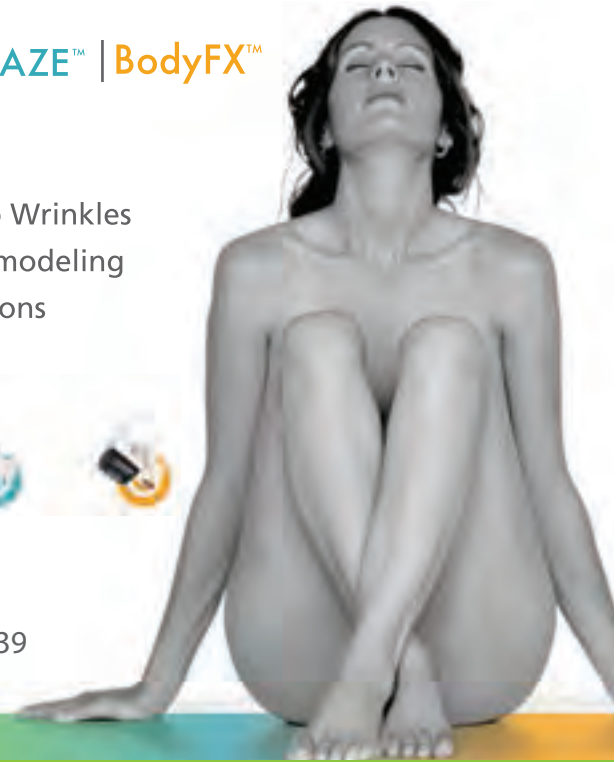
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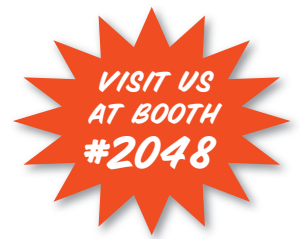
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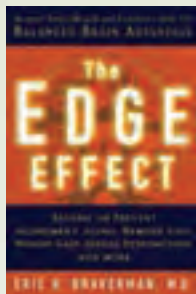
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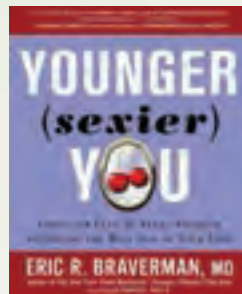


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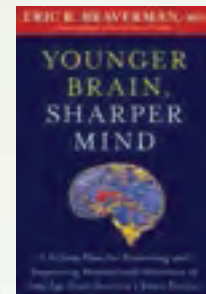
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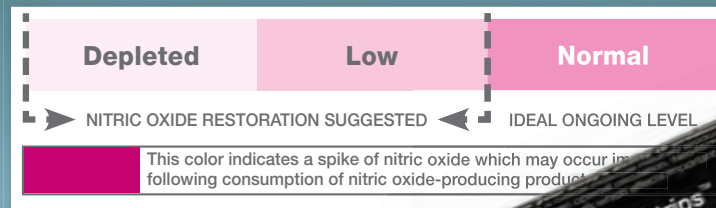
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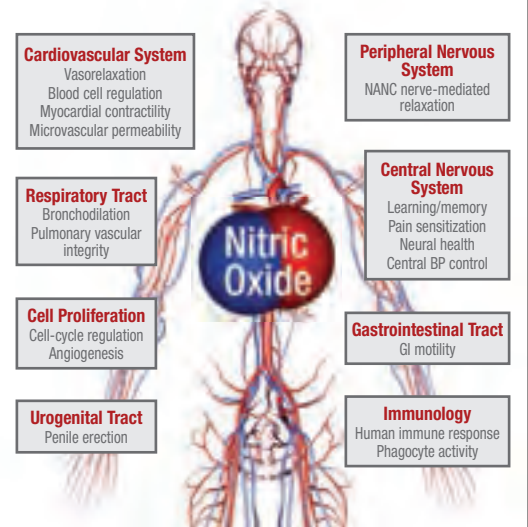
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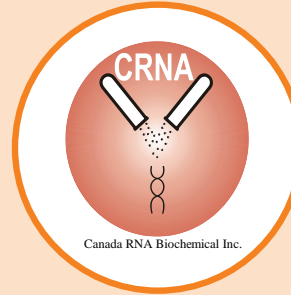
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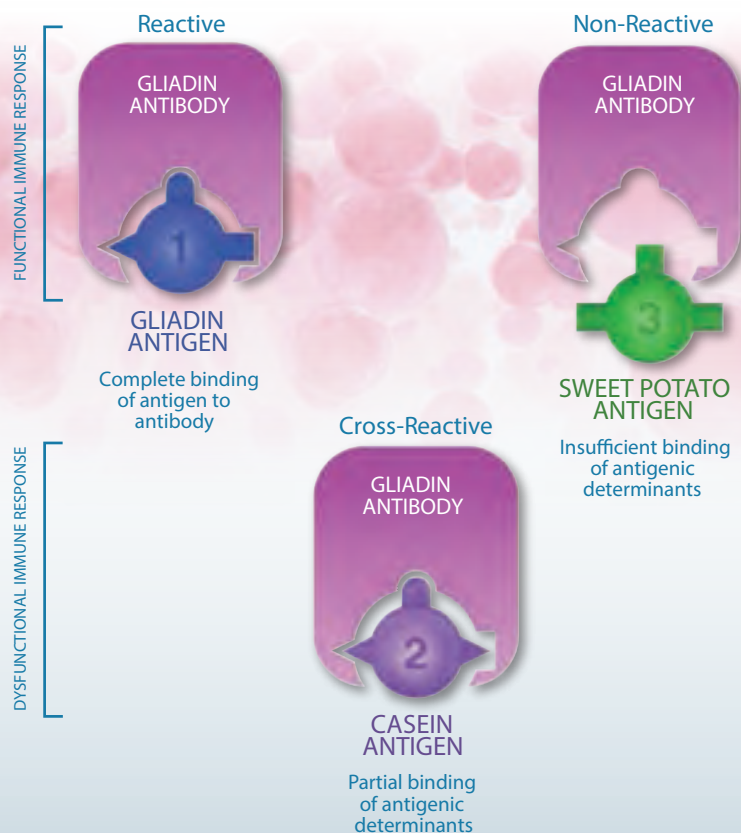
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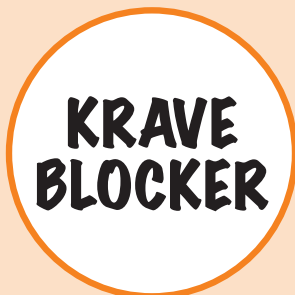
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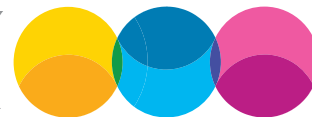


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Sangeeta Pati, MD, FACOG

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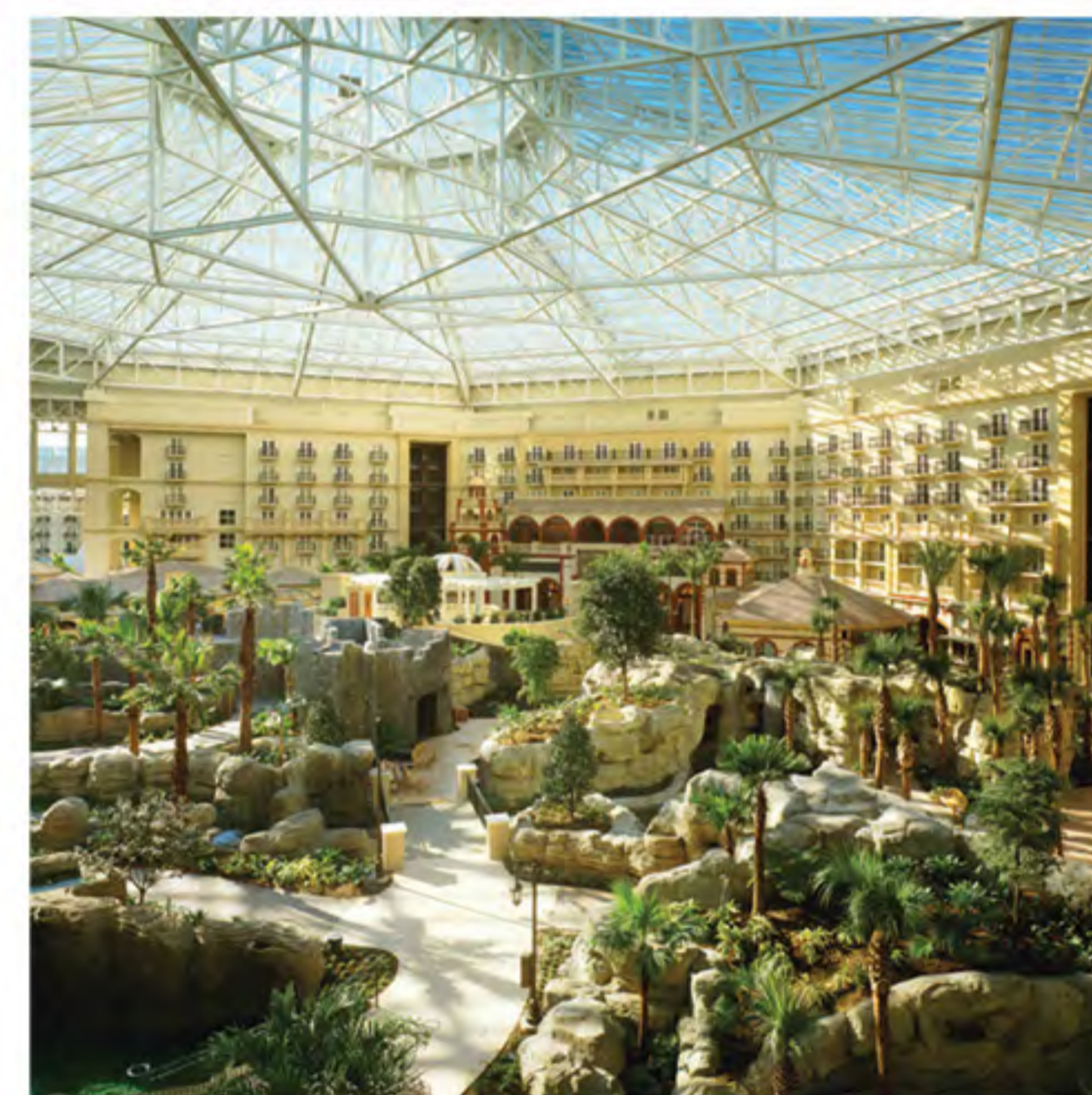
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