



CONVERGENCE

News, Links, and Insights
by JEFFREY BLAND, PHD



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Thank you for subscribing to Dr. Jeffrey Bland's newsletter. Enjoy and share this information, which is for educational purposes only and is not intended to be a substitute for professional medical advice, diagnosis, or treatment. Always consult with a qualified healthcare professional when you are in need of advice regarding a medical condition.

In this issue: The Broad Immunomodulatory Spectrum of Bifidobacteria; EPA Decisions on Chlorpyrifos (video blog); Gut Microbes Remodel the Vasculature; SNIppets: Zinc Transport; Balancing Inputs for Sustaining Brain Outputs

But first, a quote from the FMU Knowledgebase!

"Ten pounds of weight loss is more effective than any drug I could possibly give you."

Diabetes Specialist Philip Kern, MD
University of Kentucky
October 2014 Interview
Functional Medicine Update

Find a link to the October 2014 issue of Functional Medicine Update at the end of this newsletter, and learn more about how to explore Dr. Bland's extraordinary audio archive.

The Broad Immunomodulatory Spectrum of Bifidobacteria

Considering that Bifidobacteria are members of the Actinobacteria phylum that includes potential pathogens like Gardnerella, Corynebacteria, Actinomyces, and Propionibacteria, it is not surprising that they have strong immunomodulating potential in both protective and antiinflammatory modes. How is it that bifidobacteria can stimulate immunity in infants and elders and yet soothe colitis and other inflammatory states? Bifidobacteria are well-known butyrogens, producing this short-chain fatty acid

(SCFA) to feed hungry cells lining the intestines, but *B. bifidum* have also shown the surprising ability to metabolize the intestinal proteoglycan mucin into oligosaccharides that feed other bifidobacteria and allow them to produce even more SCFAs. These SCFAs then signal through G protein-coupled receptors to promote [production of regulatory T cells that rein in](#) the immune response. However, in the presence of intestinal pathogens, [bifidobacterial structural proteins](#) can interact with resident intestinal immune cells to trigger specific protective responses. In addition, at least four strains of *B. bifidum* can produce interleukin-17 to help [heighten mucosal defense](#), yet each of these strains differentially influences multiple cytokines associated with Th1/Th2 immune balance, providing for greater variability in the overall immune response.

Dr. Bland's Latest Video Blog

A Biochemist's Perspective:

Actions by the Environmental Protection Agency are Cause for Concern

Are you following the chlorpyrifos controversy that has been making headlines in recent days? Dr. Bland is monitoring the situation from multiple perspectives--scientist, citizen, and grandfather to young children. He shares his thoughts in this new video.



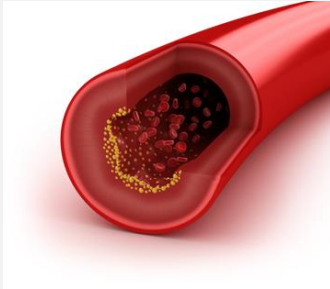
Video is one of Dr. Bland's favorite communication tools. Subscribe to his [YouTube channel](#) to never miss an update, and also find many additional videos on the Personalized Lifestyle Medicine Institute [Vimeo page](#).

The Human Longevity Project Film - You Can Register Now to Watch



Earlier this year, Dr. Jeff Bland was pleased to sit down for an interview with the producers of the Human Longevity Project film. This team has traveled all over the world to capture stories of longevity—from the scientists who study it to the people who have achieved it. These interviews are candid, authentic, enlightening, and inspiring. This 9-part docuseries begins online May 8th. Registration for the free viewing period is now open: <https://humanlongevityfilm.com/thlp-fm/?pid=5ad674d91e403>

Gut Microbes Remodel the Vasculature



Imbalance between tissue-building and -cleansing substances in the diet directly affects circulating levels of lipids as well as how they are stored in the body, but it also exerts a subtler (yet not less profound) effect through the garden of beneficial, commensal, and pathogenic bacteria it cultivates in the intestines.

Dysbiosis can promote [changes in vascular structure and function](#) when diets regularly provide rich sources of saturated fats, cholesterol, and precursors of the pro-inflammatory metabolite trimethylamine-N-oxide (TMAO). Dysbiosis impairs normal bile salt metabolism, reducing cholesterol excretion, and a microbiota skewed towards greater abundance of *Prevotella* species can increase TMAO formation. In the gut, these dietary substances promote formation of endotoxins that interfere with intestinal barrier integrity while lowering levels of bifidobacteria and other antiinflammatory butyrogenic species. Upon entering circulation, these endotoxins provoke vascular irritation, and in response, the vessels try to sequester the offending particles by forming localized deposits, foam cells. Even before atherosclerotic plaques are formed, vascular inflammation results in oxidation of circulating LDL cholesterol, reducing the formation of nitric oxide within blood vessels and causing vasoconstriction. At this point, the vasculature and microbiota are signaling each other their biochemical distress, maintaining a protective inflammatory response that is appropriate under these conditions. Unless blood levels of fats and endotoxins decrease, these foam cells thicken and calcify, ever recruiting more immune cells and eventually forming an unstable, wound-like structure.

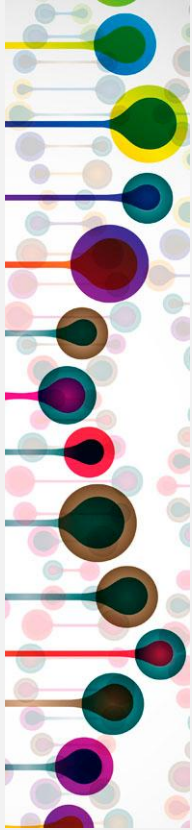
Though this sequence of events forms a complex knot, untying it is simple: modify biological input. Beyond replacing pro-inflammatory fats and TMAO sources, supporting butyrate formation and normalizing bile and cholesterol metabolism is recommendable. Inulin is well-known for promoting the growth of butyrogenic gut flora, but preclinical research suggests that [it may also improve bile acid turnover](#) and nitric oxide metabolism. Furthermore, it was shown to increase secretion of glucagon-like peptide-1 (GLP-1), a potent insulin secretagogue that triggers postprandial reductions in glucose and triglycerides.

SNiPpets

How significant to health are certain single nucleotide polymorphisms, also known as SNPs? SNiPpets is an ongoing exploration of this topic. This column is produced by Jeffrey Bland, PhD and the Personalized Lifestyle Medicine Institute.

With this SNP, Extra Zinc Improves Immune

Balance



Zinc is a crucial mineral for building a strong yet properly targeted immune response and aiding wound healing. Among healthy elderly individuals, carriers of the rs2234632 SNP of a gene coding for zinc transport in the blood show lower cellular levels of zinc along with higher blood concentrations of the pro-inflammatory mediators tumor necrosis factor- α (TNF α) and RANTES (a signaling molecule that recruits more immune cells and whose full name is Regulated on Activation, Normal T-cell Expressed and Secreted chemokine). However, [with zinc supplementation](#), levels of TNF α and RANTES, as well as the immune activator MCP-1 in these SNP carriers, suggesting overall more favorable immune balance and reduced susceptibility to inflammatory disorders.

Balancing Inputs for Sustaining Brain Outputs



In brains affected by Alzheimer's disease, impairments in energy metabolism coupled with deposition of beta-amyloid proteins were recently found to relate to how different areas of the brain connect to and communicate with one another. Over time, tissue atrophy occurring in one brain area was found to spread into other regions characterized by what scientists called 'specific connectivity' (connection patterns of one grey area to

other specific grey areas), whereas changes in metabolism spread through networks in a different way, affecting areas of what was termed 'high global connectivity' (connections between one grey area and all other grey areas). These results suggest that loss of function in [one area of the brain may differentially affect other areas](#) according to the quality of communication between them. And since hypometabolism and protein misfolding can occur to a lesser degree even in 'normal' brains, the findings echo the notion that maintaining a high level of coordination among neural networks in the brain (through activities like learning, exercise, problem-solving, and mind-body practices) may be key to preserving overall cognitive function.

While this study represents an early foray into better understanding the brain's complexities, it supports the idea that feeding healthy brain energy metabolism, redox homeostasis, and plasticity through lifestyle inputs is crucial for preventing the functional alterations seen in cognitive impairments. In this FMU interview, Dr. Bland and [NIH award-winning researcher Suzanne Craft, PhD](#) discuss how dietary choices significantly impact cerebral insulin signaling, immune activation, and detoxification to limit the hypometabolism that presages formation of brain plaques and cognitive dysfunction. Dr. Craft further explains that insulin and beta-amyloid compete for a metalloprotease that degrades both, that insulin affects production of this enzyme, and that beta-amyloid influences insulin receptor function, impeding long-term energy dynamics throughout the brain.



Where in the World is Dr. Bland?

Every year, Dr. Jeff Bland speaks in front of audiences around the world.

Will this be the year your paths cross?

[View Appearances Calendar](#)



Check out what's happening at the **Personalized Lifestyle Medicine Institute**

With Dr. Bland at the helm, PLMI is growing and expanding its educational outreach. Visit the [PLMI website](#) to learn more about:

- The 2018 Thought Leaders Consortium in Tucson, AZ October 12-13, 2018
- Past video presentations that are free to watch in PLMI's online Education Portal
- Other leadership activities and initiatives

FMU KNOWLEDGEBASE

For more than three decades, Dr. Jeff Bland recorded and self-published a monthly audio journal called Functional Medicine Update (FMU). Although he is no longer recording new issues, an archive of content spanning 1997-2016 is free to explore on Dr. Bland's website, and this extraordinary collection is now known as the FMU Knowledgebase. This newsletter began with a quote by Dr. Philip Kern, who was interviewed in October 2014. To access that issue, click [here](#). To explore the full archive, visit the [FMU Knowledgebase](#).

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Newsletter Team

Jeffrey Bland, PhD - Publisher

Cheryl Kos, ND - Content Developer and Writer

Trish Eury - Content Editor

Annette Giarde - Subscription Manager