



CONVERGENCE

News, Links, and Insights
by JEFFREY BLAND, PHD



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PERSONALIZING NUTRITION THERAPY
IN THE AGE OF LIFESTYLE MEDICINE:

Compelling Evidence, Breakthrough Science,
and a **New Era** of Clinical Care

OCTOBER 11 - 12, 2019 Seattle, Washington

Hosted by
Jeffrey Bland, PhD

Early-Bird Registration
is Now Open!

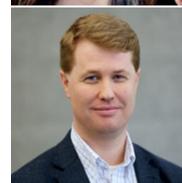
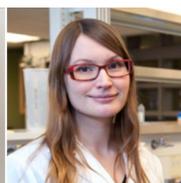
www.plminstitute.org



The Seventh Annual Thought Leaders Consortium

See you in Seattle this October? Early-bird registration is now open for Dr. Jeff Bland's signature event. More than 20 expert speakers will focus on **INNOVATION** and **IMPLEMENTATION**: evidence-based science, new assessment tools, and clinical approaches to personalized therapeutic nutrition.

Click [HERE](#) to learn more.



Clockwise from upper left: Albert-

Clockwise from upper left: Sara

Clockwise from upper left: Molly

Click [HERE](#) to view the current program schedule.

Click [HERE](#) to register for the 2019 Thought Leaders Consortium.

Silymarin Can Strategically Modulate Autophagy and Apoptosis



Long appreciated as a sovereign healer and protector of the liver, extensive research has discovered that silymarin (a phytonutrient complex from milk thistle, *Silybum marianum*) also cooperates with antioxidant/detoxification enzyme systems and shows considerable influence over cell life-growth-senescence-renewal-death cycles, especially in injured, pre-cancerous, and cancer-transformed cells. Silibinin is the major component of silymarin, which can interact with an [unusually broad array of cell signaling networks](#), including those involving telomerases, caspases, microRNAs, the master metabolic regulator AMPK, growth factors, matrix metalloproteinases, cyclins, kinases, pro-inflammatory cytokines, and others.

A remarkable quality of silymarin is that it modulates cellular apoptosis and autophagy differentially in different cell types under different conditions of perturbed function. Here are a few examples:

- Through upregulating brain-derived neurotrophic factor (BDNF) expression, silibinin limited excessive autophagy in [hippocampal cells](#) stressed by amyloid-beta exposure
- Silibinin protected [pancreatic beta cells](#) from inflammation in diabetes by selectively modulating autophagy and apoptosis and encouraging estrogen receptor expression
- Silymarin has been reported to reduce mitochondrial apoptosis in [dopaminergic neurons](#) of the substantia nigra and engage estrogen receptor-beta in the central nervous system
- Silibinin induces apoptosis in [prostate cancer cells](#), and was seen to potentiate some chemotherapies
- Silibinin activates autophagy in [endothelial cells](#) damaged by glucose toxicity
- In [synovial cells affected by rheumatoid arthritis](#), silibinin inhibited autophagy and, with Sirtuin 1, increased apoptosis; it also induced M2 macrophage differentiation and inhibited Th17 cell development
- Silibinin induced apoptosis in [breast cancer cells](#) having high expression of estrogen receptor-alpha, and enhances autophagy induced by antagonism of estrogen receptor-alpha
- In [skin cells exposed to ultraviolet B radiation](#), silibinin reduced apoptosis in those with mild injury while potentiating it in those with extensive photodamage
- In this way-ahead-of-its-time FMU interview, Dr. Bland discusses functional genomics, how silymarin can help [normalize androgen receptor activity](#) in the prostate, and how balance among estrogenic substances affects health and function with Thomas Klug, MD, who collaborated with eminent cancer researcher Dr. Leon Bradlow

This evidence of silymarin's profound regulatory effects on cell cycles suggests that it may be a candidate for adjunct application with the [Fasting-Mimicking Diet](#) and chemotherapies in cancer treatment.

Eating Habits Can Create "Start Up" Economies in the Gut



Diet constitutes a kind of mission for the digestive tract, ordering digestive fluids, enzymes, and harmonious microbial communities that can unlock all the nutrients in food, including those humans cannot directly metabolize. Resistant starch is a good example of this: it poses a challenge in that some of its amylose linkages make its energy relatively unavailable to humans. This thrown gauntlet encourages the emergence of bacteria that can take advantage of the situation through having specialized enzymatic capacity to transform 'indigestible' into 'nutritious.' In turn, this cultivates other cooperative microbial species that can make use of metabolites produced, effectively generating a micro-economy in the utilization of a particular food or nutrient.

The *Ruminococcus* genus, which comes from the same Clostridial cluster (within the Firmicutes phylum, yet separate from clusters of well-known Clostridial pathogens) of commensal butyrogens as *Faecalibacterium prausnitzii* and *Roseburia*, is an interesting case in point. [*Ruminococcus bromii*](#), considered by some an ancestral gut species in humans, has recently been discovered to specialize in metabolizing the RS3 type of resistant starch, found in foods like plantain bananas and potatoes. *R. bromii* produces both butyrate and acetate, and thus [nourishes the intestinal epithelium](#) as well as other bacteria that thrive in the gut environment of individuals who regularly consume complex starches. For survival, these functionally related species compete in addition to cooperating, and their carbohydrate metabolism makes use of B vitamins and related nutrients provided by the host or by complementary members of the micro-economy.

Like humans, members of microbiome communities are more than just species names and numbers—each has a particular set of needs and skills that interweave with those of others in creating a living environment. Despite flying under the radar of most microbiome research to date, *R. bromii* possesses such a diverse portfolio of amylases that it is [increasingly considered a keystone species](#) within the human gut microbiome, characterizing diets rich in fermentable fibers.



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](#)

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