



## Quantum Medicine Update

by Paul Yanick, Jr., PhD

### Activating the Longevity Gene to Restore Insulin Sensitivity and Immune Reciprocity in Patients with Metabolic Syndrome

Metabolic Syndrome due to insulin resistance and aberrations in visceral-abdominal fat generates a cluster of physiological abnormalities: high triglycerides, high blood pressure, high fasting blood sugar, and low HDL Cholesterol. While insulin injections help control diabetes, they are by no means a cure. Diabetics commonly have elevated risks of severe neurological and vascular complications, primarily due to autoimmune destruction of the insulin-producing beta cells. When the body lacks insulin, cells starve and glucose levels soar, causing blindness, kidney failure, and a wide spectrum of diseases. No treatment on the market has been proven to correct insulin resistance by addressing immune and genetic mechanisms underlying the death of pancreatic beta cells.

Recently SIRT1, also known as the *longevity gene*, has been shown to promote adaptation to caloric restriction (CR) by regulating the genetic programs for gluconeogenesis and glycolysis in the liver.<sup>1</sup> SIRT1 is a pivotal sensor of glucose and nutrient availability, a central metabolic regulator in the liver, muscle, and fat cells, and a regulator of cell proteins, apoptosis (cell death), and glucose metabolism.<sup>1-4</sup> Since insulin resistance causes a buildup of visceral-abdominal fat that induces unwanted inflammation and oxidative stress, we may need to ask if SIRT1 activation is necessary to reverse these processes. The answer, based on recent research, appears to be a resounding yes.

Excessive abdominal fat is a strong predictor of heart attacks in young men and chronic heart failure in older people and a predictor of high blood pressure. Excessive abdominal fat is even implemented in the development of Alzheimer's disease, colon cancer, gallstones, ovarian cystic disease, breast cancer, and sleep apnea. Unlike other kinds of body fat, visceral-abdominal fat can become dysfunctional and produce

a stew of menacing molecules that can expand to the point of rupturing. Ruptured fat cells trigger immune cells (macrophages), interleukin-6 (IL-6), and tumor necrosis factor-alpha, which adhere to the endothelium of the blood vessels, causing atherosclerosis. Indeed, elevated levels of IL-6 and C-reactive protein (CRP) predict the development of type 2 diabetes and support the role for inflammation in diabetogenesis. Baseline levels of CRP and IL-6 were significantly higher in 188 diabetic women vs. 362 matched "normal" controls. And large-scale studies (the Physician's Health Study and the Women's Health Study) revealed high CRP levels to be a risk predictor of myocardial infarction or stroke in men,<sup>5</sup> cardiovascular events in women,<sup>6</sup> and cardiovascular events in patients with the metabolic syndrome.<sup>7,8</sup> A cross-sectional study revealed that CRP levels were related to insulin resistance, obesity, endothelial dysfunction, hypertension, and diabetes,<sup>9-12</sup> and excessive visceral-abdominal fat.<sup>13,14</sup> These studies raise the prospect that doctors might forestall autoimmune disease by restoring immune function.

In autoimmune disorders, immune reciprocity is lost, due to an insufficient population of commensal cells and a disruption of the pH gradients in the digestive tract. Besides diabetes, autoimmune disorders cause rheumatoid arthritis and more than forty other conditions. In these disorders, components of the body's immune system cause extensive damage to the energy-producing mitochondrion in commensal cells. Commensal cells account for 90% of the body's cells and possess a rich genetic diversity. With 235,000 more genes than human cells to express, they can activate huge amount of SIRT1 and mimic the positive health effects of caloric restriction.<sup>15-16</sup> In my February-March 2007 column, I defined how the use of synbiotic-prebiotic nourishment, rich in bipolar energies (polarities), provided the best way to nourish SIRT1-



generating commensals.<sup>17</sup> In choosing nourishment for these cells, we need to consider avoiding man-made, synthetic "USP" vitamins or pharmaceuticals, as they all carry positive ionic charges that have the opposite effect on SIRT1.

Since maldigestion (gastro-duodenitis) causes constrictions in what I term the body's primary *Excretion-Secretion Channel*, it needs to be addressed clinically. Gastric acid production is regulated by reciprocity of the Autonomic Nervous System (ANS) and hormones. The vagus nerve is directly involved in this process, and stress depresses vagus function. The duodenal release of digestive fluids depends on the volume of fluids being secreted via the sphincter of oddi. Gastric acid is neutralized by pancreatic sodium bicarbonate-enzymes and alkaline bile. It is important to understand that low levels of gastric acids lead to a major reduction in hepatic cell production of bile, causing infectious gastritis. In these cases, the disinfecting properties of gastric acid can no longer act as a chemical barrier to food-borne infections as explained in Table 1.

When bile production is reduced, bile becomes toxic and acidic, burning the duodenum and increasing the body's toxic burden. An inflamed duodenum interferes with detoxification (excretion) and digestion (secretion). Toxic bile fractions cause extensive mucosal damage, breaching gut barrier functions even further. As Figure 1 illustrates, gastric-Biliary pH disorders cause insulin resistance and cause commensal cells to starve and die from a lack of synbiotic nutrients, because synbiotic nutrients cannot be produced in the gut when digestive fluids are diminished or gastric or bile pH is abnormal.

### Harnessing the Therapeutic Benefits and Rich Genetic Diversity of Commensal Cells

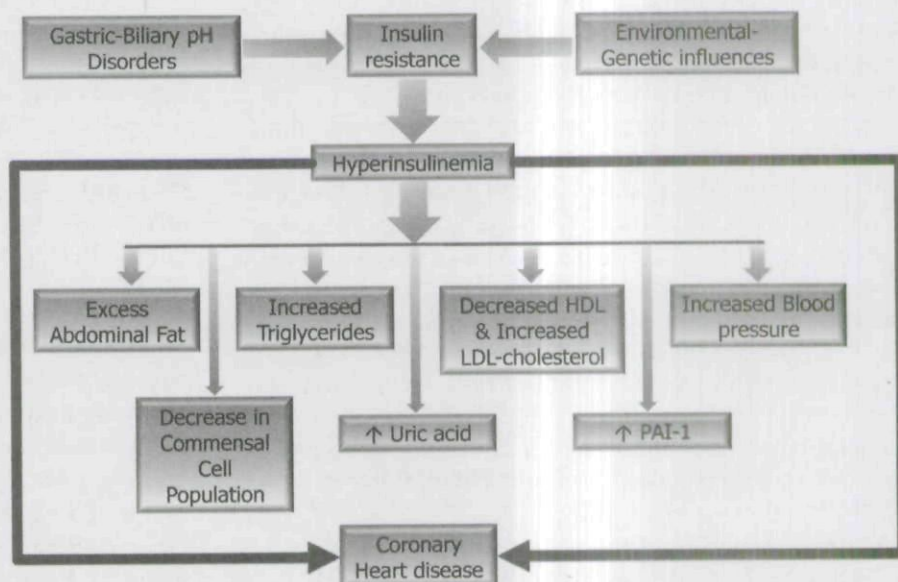
Commensals – more than a trillion per every gram of intestinal tissue – are immunological peacekeepers that promote the digestion of food and destroy dangerous microbes. Two Nobel laureates have exploited commensal flora as a realistic therapeutic strategy for infectious, inflammatory, and neoplastic disorders.<sup>18-20</sup> Commensals are essential health assets that confer protection against infections, offer prime mucosal immunity, produce a rich repository of nutrients and metabolites, restore the reciprocity of immune mechanisms, and activate genetic mechanisms (SIRT1). They have confirmed efficacy in acute enteric infections, post-antibiotic

syndromes, colitis, and irritable bowel syndrome, and they are critical for restoring immune reciprocity.<sup>17-29</sup>

Probiotics, defined in operational terms as commensal cells, exert their beneficial effects by mimicking the competitive interactions, antagonism of pathogens, and production of synbiotic nutrients and anti-microbial factors. Commensal flora is the most adaptable and renewable metabolic organ of the body, demonstrating a level of metabolic activity comparable to the liver. The metabolic repertoire of the flora includes the synthesis of active synbiotic compounds that create a gut immunological barrier. A synbiotic is defined as a mixture of probiotics and prebiotics that activates SIRT1 and has proven efficacy in a wide spectrum of clinical disorders.<sup>30-39</sup>

Progress with the clinical use of probiotic and synbiotics has been delayed because of a failure to achieve the following:

- **Correct Digestive Incompetence:** Our research shows that correcting the pH gradients of the gut, especially gastro-duodenitis, requires symbiotic 200:1 cultures.
- **Eliminate Mycotoxins:** Our research and that of other scientists has documented high levels of mycotoxins (mold) in a high percentage of cultured or fermented probiotic or synbiotic products.<sup>40-43</sup> For example, in one university study of 49 samples, 41 had dangerous levels of fungal isolates.<sup>40</sup>
- **Maintain Quantity Control:** A bewildering array of soft claims and standardized verification of product bioactivity, composition, stability, and shelf life has resulted in formulations with microbe-microbe competition and formulations that do not mimic host-flora signaling pathways.



**Figure 1: Gastric-Biliary Dysfunction as a Precursor to Insulin Resistance and Coronary Heart Disease**



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## • Exploit Host-Commensal Signaling Pathways:

According to a leading commensal researcher at the University of Ireland, "mucosal homeostasis requires continual signaling from bacteria within the lumen of the gut. It is a question of mimicking the flora and exploiting host-flora signaling pathways." Researchers now understand that host-flora signaling is a function of riboswitches and pattern recognition receptors that attenuate inflammatory responses and allow commensals to take residence in the gut. Therefore, a mineral-ligand matrix – critical to riboswitch signaling of commensals – should logically be included in probiotic formulations. Since Yale University research shows that commensals make synbiotic nutrients with riboswitches,<sup>44,49-51</sup> finding ways to exploit these signaling pathways in clinical practice may yield superior results with probiotics.

- **Mimic Gut Flora:** Competitive microbe-microbe interactions are neglected in probiotics. Finding ways to mimic human commensal flora should be explored in greater detail. By accomplishing this feat, probiotics can become commensals and will not be transient in nature. They stay in the gut long enough to maintain the physiological normal state of inflammation and activate gut-associated lymphoid

tissue (GALT). GALT is the largest immune organ in the human body, resulting in the stimulation of T- and B-cells and the establishment of the cytokine networks. It is critical to understand that once a person has taken an antibiotic or a natural anti-infective, they only have a ten-percent (instead of 90%) population of commensals. For over a decade, I have studied how different probiotics can disrupt the reciprocity of the TH-1 and TH-2 cytokine responses and looked carefully at how some can compete against others to diminish their total effectiveness. Borrowing further from nature's design, I fermented foods to produce mineral-ligands that could turn "on" the toggle-like riboswitches for a greater range of bioactivity or turn "off" in pathogenic bacteria, starving them to death.<sup>44-48</sup>

- **Nourish Commensals:** Commensal cells make proteins – the wheels, cogs, chutes, and conveyor belts that transport the synbiotic nutrients to cells – and co-enzymes in small, "nano-sized" molecules that cooperate with proteins and enzymes to ignite powerful biochemistry in the body. Epic metabolic pathways are activated in the construction of these nutrients, which can occur in the fermentation tank or in the body as commensals; they construct nutrients from prebiotics and synbiotics and strictly control nutrient levels by shutting synthesis down when nutrients are ample and the cell's infrastructure is optimal.<sup>30-39</sup>

**Table 1: Comparison between hepatic cell production of bile during healthy digestion and digestion affected by pH disorders**

Digestive Organ	Healthy Digestion	Digestive pH Disorders
Stomach	6 pints of HCl	¾ pint of HCl
Liver	2 pints bile salts	¼ pint of bile salts (acidic pH)
Pancreas	3 pints pancreatic juices	1 ½ pints of pancreatic juices

**Table 2: Specific Strains and Their Effects**

<i>Lactobacillus acidophilus</i>	Immune enhancer, important commensal, prevents diarrhea or constipation, anti-inflammation effects, supports GALT <sup>8-20,58-62,67</sup>
<i>Lactobacillus Casei</i>	Immune enhancer, positive anti-diarrhea effects, positive immune effects with cancer <sup>52,55-57</sup>
<i>Lactobacillus plantarum</i>	Inhibits bacterial translocation and secondary septic responses <sup>52</sup>
<i>Lactobacillus Salivarius</i>	Minimizes abdominal fat storage, anti-clostridium effects; attenuates GI inflammation <sup>6</sup> and prevents tumor formation <sup>63-66</sup>
<i>Lactobacillus Rhamnosus</i>	Prevents eczema and has anti-allergenic effects. <sup>54,58-62</sup>
<i>Bifidobacterium bifidum</i>	Prevention of acute infectious diarrhea, anti-clostridium effects <sup>23-29,68</sup>

While pharmaceuticals and natural anti-infective herbs can save lives, they are indiscriminate killers of all commensal cells. Antacids and digestive enzymes destroy commensals or alter commensal cell metabolism, creating a favorable environment for opportunistic yeast and fungal infections. In addition, excessive alcohol and sugar, non-steroidal anti-inflammatory drugs (NSAIDs), radiation, chlorine, or fluorine, and inorganic, non-covalent ionic minerals are extremely harmful to commensal cells. In the face of a global epidemic of metabolic syndrome, none of these stopgap measures address the essential question: what kind and balance of commensals can diminish autoimmune reactions and restore immune reciprocity?

To answer this question, it is important to understand that immune reciprocity is contingent on commensal nourishment and that commensals are the immune system's primary weaponry against unwanted microbial invaders. When depleted or wiped out from antibiotics or anti-infectives (herbal, silver, hydrogen peroxide, or stabilized oxygen) or synthetic, man-made chemicals, commensals are unable to proliferate and take residence in the gut. The result: the immune system is driven into exhaustion from long-term immunological warfare and battles that cannot be won. This exhaustion, called immunosuppression, explains



why runaway viral infections can't be controlled and why so many patients struggle with cyclic yeast, fungal, and bacterial infections.

Table 2 illustrates the specific strains used in our clinical studies designed to exploit signaling pathways, improve acid and bile tolerance, and eliminate unwanted microbial competition. Our preliminary research shows that powerful antagonistic actions against a wide spectrum of microbial pathogens may be achieved with the correct probiotic balance.

### Nourishing Commensals and Beta Cells to Activate SIRT1 and Restore Glucose Homeostasis

As Figure 2 illustrates, a loss of the reciprocity of immune TH-1/TH-2 cytokine responses causes *autotoxicus*, or aberrant autoimmune behavior, which causes destruction of the insulin-producing beta cells of the pancreas. This destructive process eventually destroys enough beta cells, causing excess abdominal fat and diabetes. When the immune system attacks proteins made by the pancreatic beta cells, the offending proteins, or "autoantigens," destroy the beta cells. To that end, synbiotics were developed from spirulina and green barley to mimic the effects of balanced commensal flora on restoring beta cell and other cellular functions. Again, our preliminary clinical outcome studies reveal positive changes in the toughest, treatment-resistant cases with synbiotic nourishment.

Since synbiotics are rich in organic, non-covalent chromium and zinc mineral-ligands formed by nature via a controlled, mycotoxin-free fermentation process, they contain thousands of known and unknown

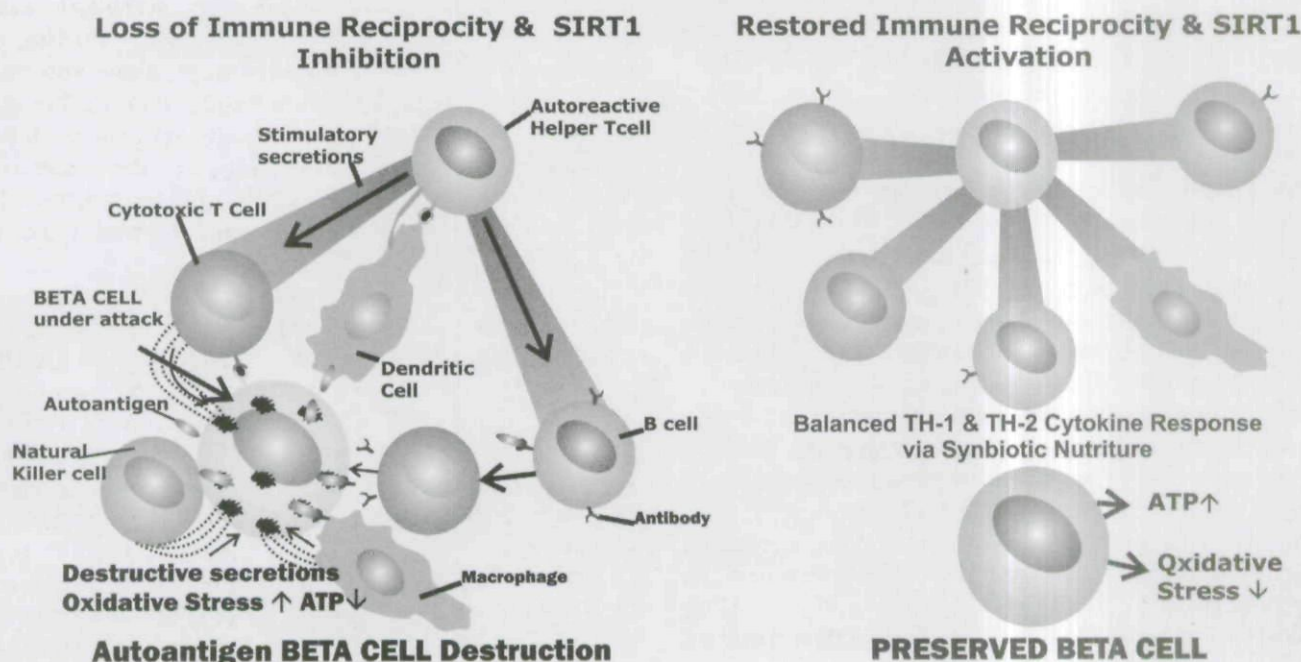
## Quantum Medicine

nutrients in co-protein formats. As such, they can penetrate malnourished cells thousands of times faster than eating whole foods and/or taking conventional supplements. This common-sense approach is based on the recognition that, as cells become less efficient, so do the tissues and organs they compose. Cellular inefficiency causes a loss in reciprocity on a multi-dimensional anatomical basis. Thus, the body can't resist infections, reject tumors, inhibit carcinogenesis, detoxify pollutants, and heal itself.

Nature's recipe for restoring insulin sensitivity is vastly different than the man-made creations of nutritional and pharmaceutical science which lack polarity and carry a positive ionic charge. Positive ionic toxicity disrupts the body's reciprocal, harmonic polarities, causing a loss of immune and endocrine reciprocity, and intensifies oxidative stress. In turn, free radicals damage proteins, lipids, and the ATP-synthesizing machinery of mitochondrial DNA, causing deficits in ATP production. Since ATP production decreases with age in the brain, heart, and skeletal muscle, it may help to explain why degenerative diseases of the nervous system and heart are common in diabetes.

Other than caloric restriction, treatments to reverse pancreatic beta cell function are limited and ineffective. Since a great deal of research implements glucose metabolism in regulating lifespan,<sup>18,19</sup> the use of synbiotic, nanoscale nutrients as a caloric restriction

Figure 2: Beta Cell Function and Immune Reciprocity





mimetic to activate SIRT1 may allow cells to function more efficiently. Maintaining the harmonic polarities of healing and repair mechanisms in the quantum domain allows the electron transport chain to stabilize and minimize oxidative stress. Clearly, more efficient functioning of the cells' ATP-producing machinery has the potential to improve cellular health on many levels.

Exploiting nature's reciprocity to create bipolar nourishment, reduced in molecular weight from 60,000 daltons in a whole food to 250 daltons, is what makes a synbiotic nutrient so special. Synbiotic nutrients are loaded with carrier proteins and ligands, which are proven to improve cell receptor responsiveness<sup>17-19</sup> and can yield dramatic results in studies of patients with severe acute pancreatitis,<sup>20</sup> chronic hepatitis, abdominal surgery,<sup>21</sup> and liver transplantation.<sup>21,22</sup>

Regrettably, most nutritional or pharmaceutical prescriptions disrupt commensal and beta cell functions and cannot quell the cellular misbehavior of renegade immune cells, tame oxidative stress, or correct the deficiency states underlying insulin resistance. Instead, their entry into the bloodstream stimulates energy artificially, causing chronic nutrient deficiencies. Acting as inhibitors or agonists of reciprocity, these prescriptions act as stressors to decrease an organism's ability to withstand stress.

In summary, a steady chorus of skeptics continues to cast doubt on the massive peer-reviewed scientific literature that forms the cornerstone for a consensus that nature's recipes for nourishment are vastly superior to man-made synthetic nutrition. The healing wisdom of nature is found primarily in the genes of commensal cells, which have the remarkable ability to "heighten one's disease fighting capacity" and produce co-enzymes, proteins, essential fatty acids, vitamins, and compounds that heal, disinfect, and repair the body.<sup>28</sup>

The genetics of these cells can transform the body into a *super-organism*, with an ability to do things far in excess of its innate healing abilities. These powerful cells don't just complete us; they offer us the ability to improve the quality our lives dramatically. A world-renowned leader in probiotics, Dr. Shanahan, calls them "a virtual organ with a metabolic activity in excess of the liver" and states, "intestinal bacteria outnumber cells in the human body 10-fold, account for 400-500 species...and have the collective metabolic activity of a virtual organ - the "neglected organ."<sup>30</sup>

Finally, in earlier columns I alerted the alternative medicine community regarding the existence of gastro-duodenitis and pH disorders.<sup>75-76</sup> Healing this excretion-secretion channel is the first priority

in restoring immune and endocrine reciprocity. Antibiotics, synthetic vitamins, and processed "calorie-condensed" foods with positive-ion charges kill commensal cells and aggravate gastro-duodenitis.<sup>31</sup> Instead of treating the patient with synthetic vitamins, antimicrobials, anti-yeast, and/or anti-viral botanicals, doctors of Quantum Medicine™ emphasize nature's own cleansing and nutritive agents and respect the fact that nourishment is the ultimate way to ignite reciprocal, bipolar healing energies that activate and enhance repair and regenerative functions in the body. Applying these concepts in our patients could make the difference between having patients die miserably at 65 or live in good health until 95.

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The author is supported in part by American Academy of Quantum Medicine, a non-profit foundation dedicated to frontier research in Quantum Medicine. The author is affiliated with QuantaFoods, LLC, a firm that develops and researches probiotics and fermentation to develop novel forms of mineral-ligand-synbiotic nutrients and is a research consultant for several independent, university-based laboratories. The content of this article was neither influenced nor constrained by these facts.

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