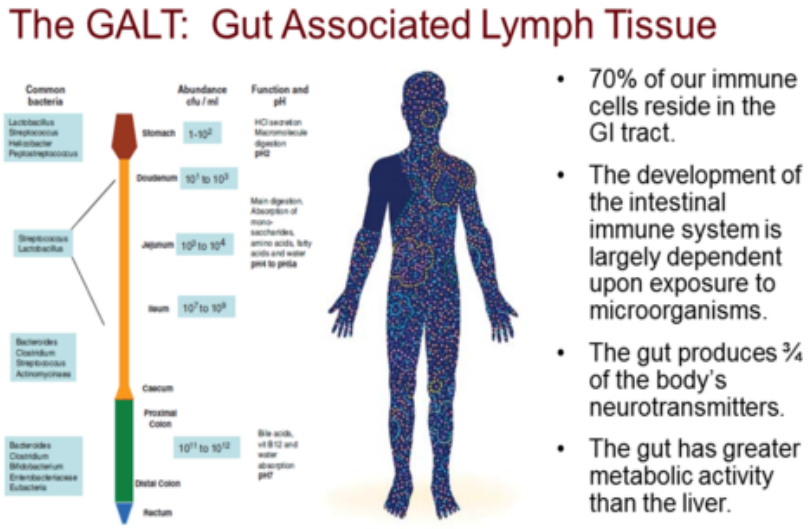
Support Your Digestive System *.

Physiology Highlights and Vital Force Technology (VFT) Formulation

This paper presents the current scientific data regarding the gut-associated immune system, the gut microbiota and its influence on the central nervous system, as well as nutritional support of the microbiota. In the last part, we explain how to combine specialized VFT energy formulas to support gastrointestinal structure and function.

1. The Role of Gut Associated Lymphatic Tissue (GALT)

As mentioned above, the GI tract/gut is continuously exposed to both potential pathogens and beneficial commensal microorganisms. This fact creates a requirement for the homeostatic balance between tolerance and immunity, which poses a unique monitoring challenge to the mucosal immune system (MacDonald & Monteleone, 2005).



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Homeostatic State of the Gut's Immune System

The commensal microbiota, intestinal epithelial cells, and intestinal immune cells all engage in a complex form of crosstalk (Kim & Ho, 2010; Selleri et al., 2008; Abraham & Cho, 2009). The gut microbiota provides a strong selective pressure to the host to evolve adaptive immune responses required for the maintenance of the local and systemic homeostasis. The continuous antigenic presence in the gut forces a dynamic remodeling of gut-associated lymphoid tissues

(GALT) and the selection of multiple layered strategies for immunoglobulin (Ig) A production (Abraham & Cho, 2009; Fagarasan et al., 2010).

The lamina propria normally contains a diverse array of immune cells and secreted cytokines. These include anti-inflammatory mediators that down-regulate immune responses, as well as pro-inflammatory mediators from both innate and adaptive immune cells (Artis, 2008). Dysfunction of this homeostasis probably results not only in various intestinal inflammatory diseases, like Crohn's disease and ulcerative colitis, but may also contribute to diseases such as dermatitis eczema and type1diabetes (Wen et al., 2008).

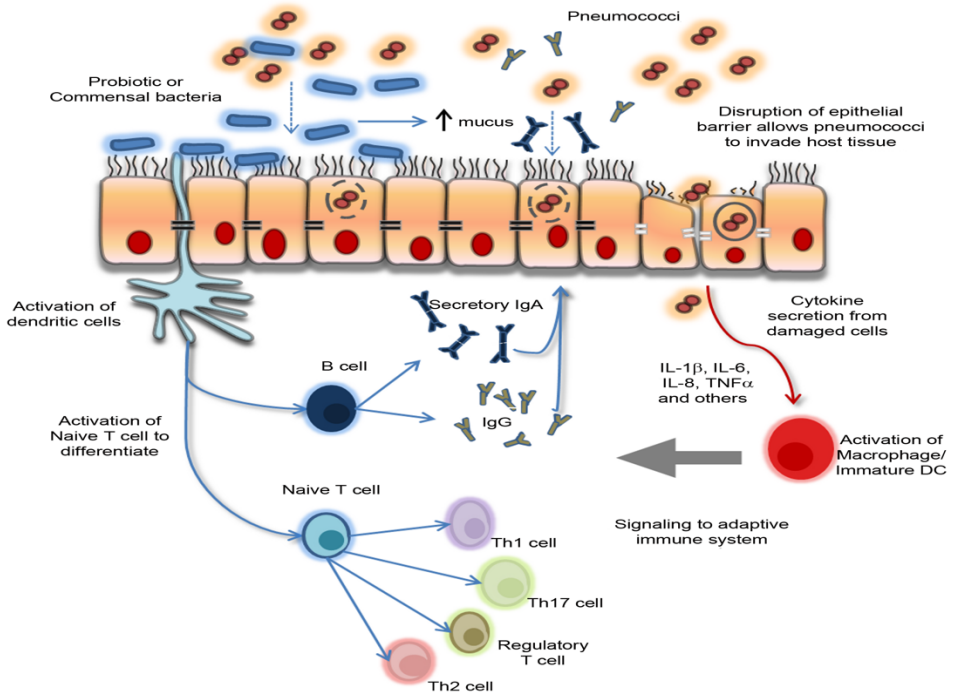
Gastrointestinal Inflammation State

In a person with intestinal inflammation, several events contribute to the increased bacterial exposure, including a disturbance of the mucus layer, dysregulation of epithelial tight junctions, increased intestinal permeability, and increased bacterial presence in epithelial cells. In the case of inflammatory bowel disease, innate cells produce increased levels of the tumor necrosis factor α (TNF-α), interleukin-1β, interleukin-6, interleukin-12, interleukin-23, and chemokines. There is obvious enlargement of the lamina propria with increased numbers of CD4+ T cells, which also secrete increased levels of cytokines and chemokines. These events result in the enrollment of additional leukocytes, causing a cycle of inflammation (MacDonald & Monteleone, 2005; Selleri et al., 2008; Fagarasan et al., 2010).

Innate and Adaptive Immune Responses of the Intestinal Immune System

The innate division of the immune system provides an initial, rapid response to microorganisms. The cells of the innate system display receptors that recognize general microbial patterns, in contrast to the antigen-specific recognition by receptors of the adaptive immune system. The intestinal epithelial layer has various types of the innate immune receptors that mediate defenses against luminal microbiota, as well as epithelial and antigen-presenting cells for stimulating tolerance mechanisms that maintain an immune homeostasis in the intestine (MacDonald & Monteleone, 2005; Kim & Ho, 2010; Artis, 2008).

This microbial testing occurs by the translocation of microbes across epithelial cells and the M cells of the epithelium of Peyer's patches by immunoglobulins and dendritic cells. Activated antigen-presenting cells, notably dendritic cells, then present peptide antigens to T cells in secondary lymphoid organs of the gut, such as Peyer's patches, mesenteric lymph nodes, and isolated lymphoid follicles. This interaction initiates an adaptive immune response, after which memory lymphocytes are developed (Kim & Ho, 2010; Selleri et al., 2008; Fagarasan et al., 2010).



From Licciardi PV, Toh ZQ, Dunne E, Wong S-S, Mulholland EK, Tang M, et al. (2012) Protecting against Pneumococcal Disease: Critical Interactions between Probiotics and the Airway Microbiome. PLoS Pathog 8(6).

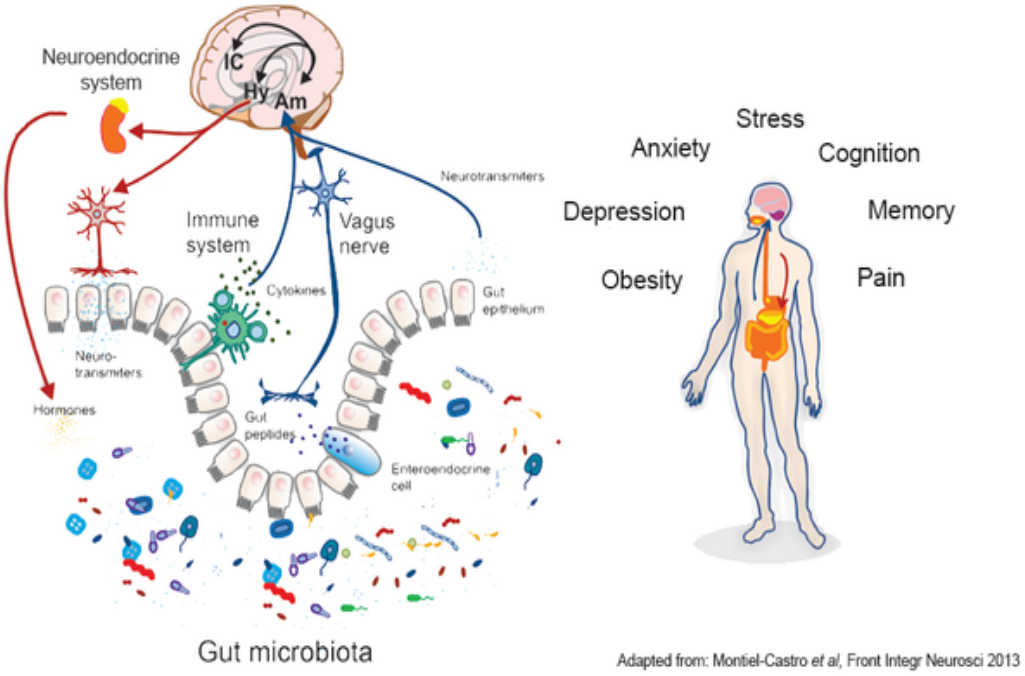
Special helper T cells (Th1, Th2, and Th17) and regulatory T cells (subgroups of CD4+ T cells) secrete several types of cytokines. Regulation of these subgroups must be continually adjusted to maintain the intestinal immune homeostasis (Artis, 2008; Abraham & Cho, 2009). Intestinal B cells produce IgA antibodies, which contribute to the immune protection without increasing inflammation (Abraham & Cho, 2009).

2. The Microbiota-Gut-Brain Axis

The gut of humans is characterized as one of the most tightly inhabited microbial networks on Earth with an average 40,000 bacterial species, 9 million unique bacterial genes and 100 trillion microbial cells (Yang et al., 2009).

The important role of the human microbiome was confirmed by multiple past and current researchers and can be expressed via the following words: “intra-familial and subsequent intergenerational transmission of a gut microbiome could shape the biological features of humans within a relationship, contributing to differences in the structures and operations of their innate and adaptive immune systems, and together with their *H. sapiens* genotypes, modulate their risks for immunopathology states, as well as other diseases” (Garrett et al., 2010).

Recently, researchers have confirmed many species of bacteria that reside within our gastrointestinal system cooperate with the rest of the body through the ruling of the immune system and secretion of factors such as short-chain fatty acids (SCFAs), which influence physiological processes throughout the whole body.



From www. optimoz.com.au/blogs/ews/81659079-the-gut-and-the brain-work-together-to keep-us-healthy

As an example, gram-negative bacteria can motivate the production of pro-inflammatory cytokines, such as interleukin IL-6 and IL-1β, via binding of the lipopolysaccharide (LPS) section of their cell walls to toll-like receptors (TLRs), which communicate to monocytes, macrophages, and microglia. The cytokines can then signal to the brain via the vagus nerve, or they can access the brain directly via the circumventricular organs (regions of the blood–brain barrier that are comparatively permeable) (El et al., 2014; Dinan et al., 2015).

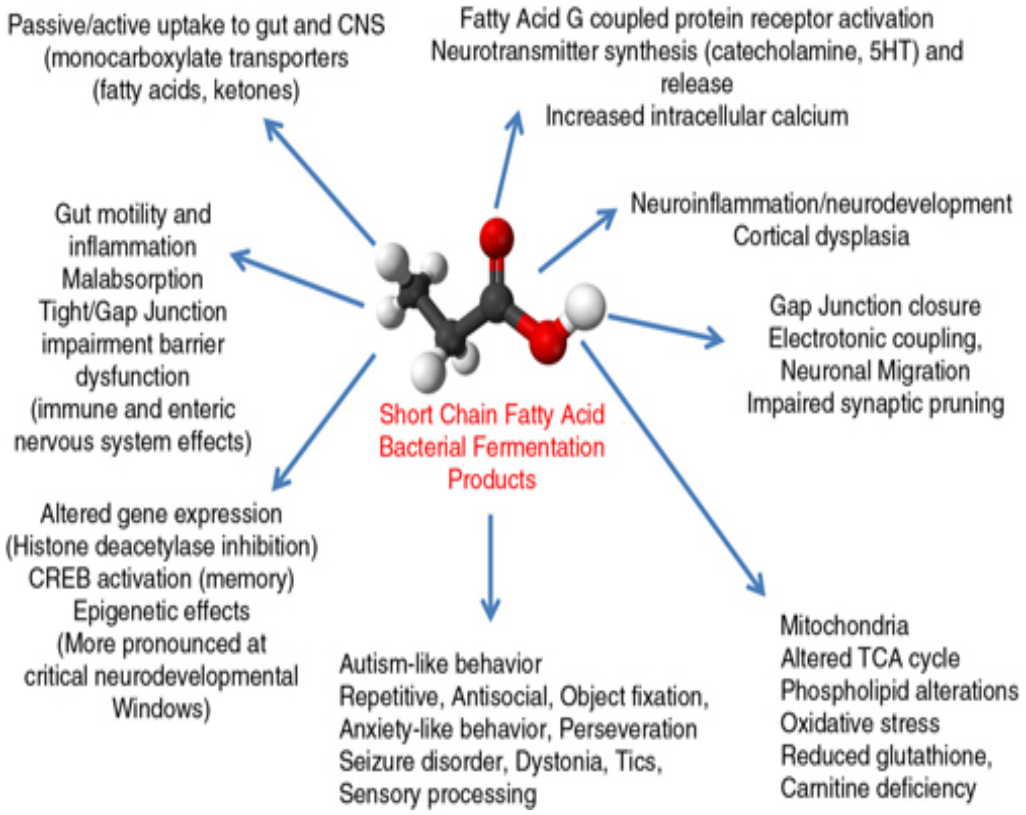
Within a bi-directional communication network with the brain, called the microbiota–gut–brain axis, the bacterial commensals help to maintain homeostasis of the central nervous system (CNS) and influence our behavior and mood, as well as play an important role for the gut microbiota in neuropsychiatric conditions, such as depression, autism and schizophrenia (Daulatzai, 2014; Kelly et al., 2015).

Activation of the Afferent Pathway via Microbiota

The vagus nerve represents the main afferent pathway from the abdominal cavity to the brain, and there are sufficient scientific data confirming the gut microbiota is capable of activating this pathway to mediate their behavioral and physiological effects on the brain (Bravo et al., 2011; Dinan & Cryan, 2015). For instance, the vagus nerve is responsible for mediating the beneficial effects of probiotics on physiological mechanisms, such as wound healing. The probiotic bacteria, *Lactobacillus reuteri,* was shown to enhance wound healing in mice by increasing the oxytocin release from the hypothalamus (Poutahidis et al., 2013).

Signaling the Brain via Short-Chain Fatty Acids

Acetic acid, propionic acid, and butyric acid are the basic short-chain fatty acids (SCFAs), metabolites produced by the bacterial population in our gut. These SCFAs act as another mediator between the gut microbiota and the brain, and they represent an additional mechanism through which gut bacteria can influence brain physiology and behavior (Tan et al., 2014).



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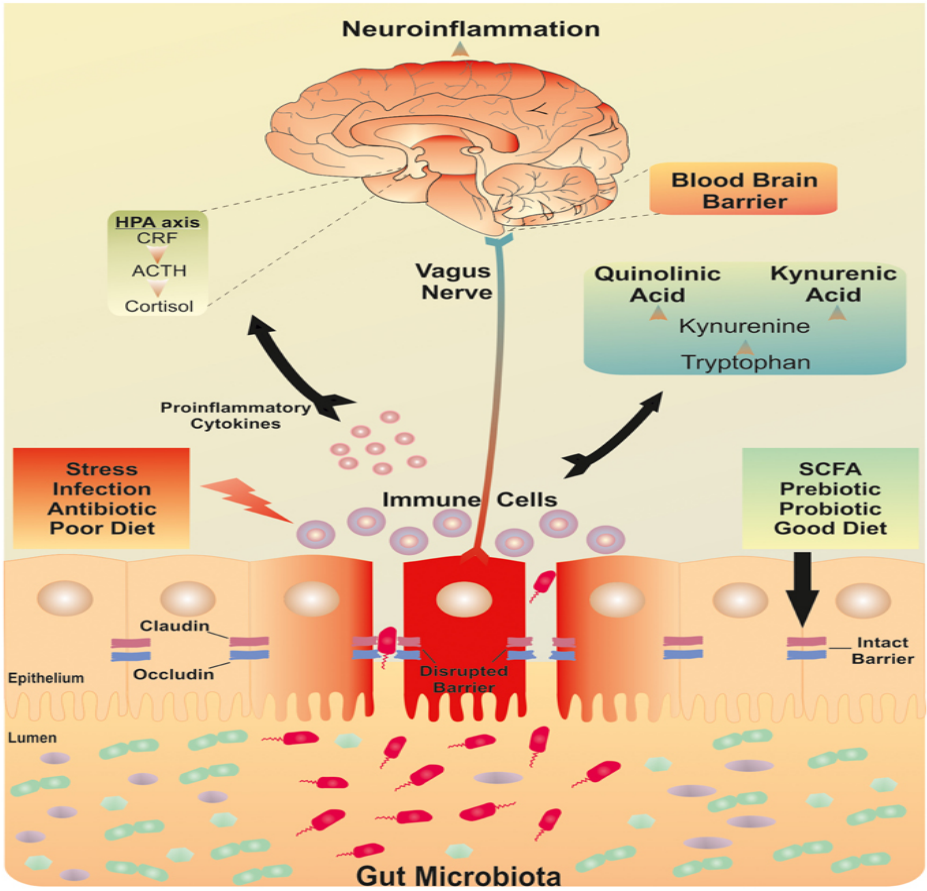
Recent research has shown propionic acid provides helpful effects upon the body's weight control and glucose metabolism by initiating the FFAR3 receptor upon nerve fibers of the portal vein (De Vadder et al., 2014). Experimental data have shown butyric acid and propionic acid increased expression of tyrosine hydroxylase, the enzyme involved in dopamine and noradrenaline synthesis and capable of regulating the serotonergic neurotransmission (Nankova et al., 2014; Valladares et al., 2013).

Researchers have demonstrated the gut microbiota can regulate the metabolism of a key dietary amino acid tryptophan and thus coordinate kynurenine and 5-H production (Wikoff et al., 2009; Clarke et al., 2013; Desbonnet et al., 2008). 5-HT is an essential neurotransmitter that regulates physiological activities such as mood, appetite, aggression and sleep (O'Mahony et al., 2015). It should be noted increased peripheral and central kynurenine have been documented in autism, schizophrenia, depression and neurodegenerative diseases, such as Alzheimer's and motor neuron disease (Schwarcz et al., 2012; McFarlane et al., 2008).



From https://www.linkedin.com/pulse/gut-microbiome-microbiota-neuroinflammation-why-does-taylor-hanna

Further, butyric acid has been shown to generate antidepressant effects in preclinical tests, while excessive doses of propionic acid are associated with insufficiencies in cognition and friendliness (Schroeder et al., 2007; Gundersen & Blendy, 2009; Macfabe, 2012; Kratsman et al., 2016).

Effect of SCFAs on Glia and Astrocytes Homeostasis

The current data suggest an increase in SCFA-producing bacteria in the gut may have an effect upon the neuro-inflammatory response controlled by microglia and synaptic regulation mediated by astrocytes. Propionic acid has been reported to increase expression of the astrocyte marker glial fibrillary acidic protein (GFAP), while butyric acid was found to reduce its expression (de Almeida et al., 2006; Shultz et al., 2009; Kanski et al., 2014).

Microbiota and the Enteroendocrine Signaling System

Enteroendocrine cells (EECs) are specific cells of the intestine that generate the signaling molecules 5-HT, cholecystokinin (CCK), glucagon-like peptide (GLP)-1 and peptide YY (PYY). They operate on the GI tract, on the pancreas, and on the brain, as well as aid energy homeostasis (Latorre et al., 2015). It has been found proteins secreted from *Escherichia coli* are capable of stimulating discharge of GLP-1 and PYY from EECs (Breton et al., 2016).

3. The Gut Microbiota and the Central Nervous System Homeostasis

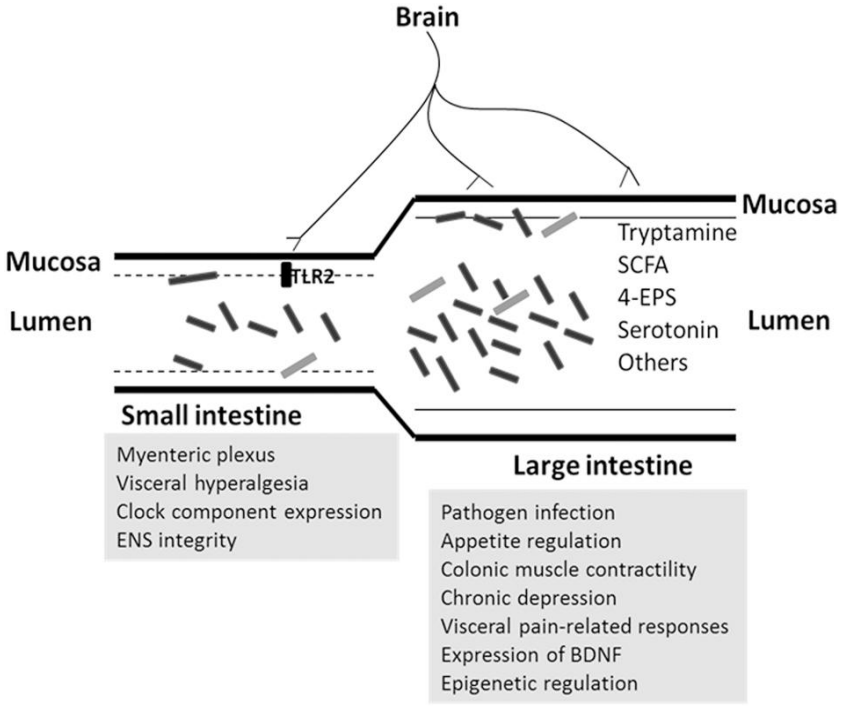
Several recent studies have demonstrated the bacterial population in the gut may regulate neurotransmission, neurogenesis, inflammatory states of the brain and activation of the hypothalamic–pituitary–adrenal (HPA) axis (Nishino et al., 2013; Ait-Belgnaoui et al., 2012; 2014).

GABA, Dopamine, Noradrenaline and Histamine

GABA is the main inhibitory neurotransmitter in the CNS, and dysfunctions in this neural system are associated with depression, anxiety, and autism (Möhler, 2013). The bacterial commensals in our gut, mainly lactic acid-creating bacteria, also produce GABA (Barrett et al., 2013; Hiraga et al., 2008; Komatsuzaki et al., 2008).

Dysfunction of noradrenaline and dopamine neural circuits has been associated with many neurological and psychiatric disorders. Researchers have demonstrated the microbiota generates a regulatory consequence upon catecholaminergic transmission in the brain and that the lactic acid–producing bacteria produced dopamine in culture (Nishino et al., 2013; Kuley et al., 2012).

Histamine can act as a neurotransmitter and is associated with central brain activities, such as circadian rhythms, food intake, learning and pain perception (Brown et al., 2001). Scientific data suggests the microbiota is capable of synthesizing this molecule in the gut, which may have an impact upon brain function (Thomas et al., 2012).



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Neurotrophic Factors and Microglial Activation

Several researchers have recently confirmed manifestation of brain-derived neurotrophic factor (BDNF) is under the effect of the gut microbiota (Savignac et al., 2012). The data also suggests the gut microbiota affect the expression of other neurotrophic factors, such as glial-derived neurotrophic factor (GDNF) and nerve growth factor (NGF) (Aguilera et al., 2016; Brun et al., 2015).

Recent works have shown the microbiota affect myelination in the prefrontal cortex, and it was suggested the microbiota might be considered as a potential treatment for multiple sclerosis and other neurodegenerative conditions (Hoban et al., 2016; Ochoa-Repáraz et al., 2010; Wang et al., 2014; Lavasani et al., 2010).

Microbiota and the Hypothalamic–Pituitary–Adrenal (HPA) Axis

Probiotics have been shown to modulate activation of the HPA axis, which may link to their beneficial effects upon behavior, as well as was proven that the certain species of gut bacteria can modulate central oxytocin levels (Tomova et al., 2015). An effect of the probiotic supplementation on corticosterone levels looks to be bacterial strain-specific, as *Lactobacillus salivarius* and *Lactobacillus farciminis* have both been shown to decrease a stress-induced corticosterone secretion (Ait-Belgnaoui et al., 2012; 2014).

Facts About Depression, Schizophrenia and Autism

Several pieces of evidence have shown "chronically depressed patients displayed increased circulating levels of immunoglobulin IgA and IgM antibodies raised against LPS expressed upon Gram-negative Enterobacteriaceae" (Maes et al., 2012,), and demonstrated the beneficial effects of a *Lactobacillus* and *Bifidobacterium* probiotic supplementation on mood in healthy individuals (Steenbergen et al., 2015).

The increase of urinary excretion of phenylalanine metabolites derived from *Clostridium* species was observed in schizophrenic patients (Shaw, 2010). Also, dysregulation in the composition of the gut microbiota by *Toxoplasma gondii* infection may create the ability of the protozoan to induce psychoses via production of bacterial metabolites that have that effect (Bhadra et al., 2013; Fond et al., 2014).

Research evidence suggests it is possible to estimate the severity of autistic behavior by the level of gastrointestinal dysfunction (Finegold et al., 2012). The levels of microbial metabolites in fecal samples from autistic children have been shown to be increased, as well as levels of acetic acid, butyric acid, and propionic acid, compared to the control group (Wang et al, 2012;2014). This excessive production of SCFAs by the gut microbiota in autistic children may cross the BBB and have a damaging effect on neuronal development and functioning (Parracho, 2005).

4. Nutritional Support of the Gut Microbiota

Prebiotics

Prebiotics are described as non-digestible fibers that support the proliferation of beneficial *Lactobacillus* and *Bifidobacterium* in the gut (Candela et al., 2008). The main prebiotics contain fructans (FOS, inulins, oligofructose) and glucans (GOS) (Slavin, 2013). It was recognized that prebiotic supplementation may help to support the healthy gut microbiota in elderly individuals and alleviate the risks of age-related gastrointestinal and psychiatric disorders (Bindels et al., 2015). Prebiotics have also been shown to reduce intestinal permeability and improve tight junction integrity (Sherwin et al., 2016).

Probiotics

The main bacterial species used as probiotics in both animal and human studies are the *Lactobacillus* and *Bifidobacterium* classes (Butel, 2014; Tillisch et al., 2013; Savignac et al., 2014). The ability of probiotics to reduce intestinal permeability may help to stop the bacterial translocation that appears in IBS, depression, and autism (Qin et al., 2005). Other beneficial effects of probiotics "include enhanced wound healing. *Lactobacillus reuteri* enhances wound healing by increasing secretion of oxytocin, which in turn activates T regulatory cells to promote healing" (Sherwin et al., 2016).

"Probiotics may suppress or enhance the growth of other bacterial species in the gut, leading to global changes in the composition of the microbiota. The mechanism of action or efficacy of probiotics may vary from one individual to another, because of variations in the composition of the gut microbiota from person to person" (Akkasheh et al., 2016).

5. Vital Force Technology (VFT) Energy Formulation to Support Intestinal Health

Clearly, we have in our hands enough scientific evidence (as well as a large number of testimonials) that VFT energy formulas are a vital part of what is being called "the New Medicine" (http://www.pbs.org/thenewmedicine/), bringing a wide range of benefits to humanity, largely because these formulas enhance the internal healing energetic mechanism that penetrates the human body through all levels: physical, emotional, and mental. Most importantly, these energetic formulas help our bodies to generate and support conditions that enhance both the purity and intensity of our vitality and life force, thereby increasing our potential to move toward our own optimum health.

Practitioners can use these specific combinations of ETI formulas, as well as fine-tune dosage, frequency and proportions of each formula, to create a personalized approach for an individual client's support to improve the gastrointestinal health.

1. Fatigue, low GI functionality

Description:
May help to overcome fatigue caused by frequent or sustained stress, followed by digestive function dysregulation.

Formulation and Dosage:

Combined dosage of Adrenal Support (10 drops) and GI Aid (10 drops) in 2-4 oz. of water.
Drink 2-3 times per day, continue up to two weeks.
Precautions: No precautions or side effects if used with the recommended dosages.

2. *High nervous state accompanied by GI structural and functional dysregulation*

Description:
May decrease symptoms of nervousness by more efficiently supporting regulation of homeostasis in the Microbiota-Gut-Brain axis, and may help with travel constipation, fight-or-flight reactions, physiologically induced diarrhea.
Formulation and Dosage:
Combined dosage of Master Brain (5-8 drops) with GI Aid (5-8 drops), Stress Relief (5-8 drops) in 2-4 oz. of water.
Drink 2-3 times per day, continue up to two weeks.
Precautions: No precautions or side effects if used with the recommended dosages.

3. General support of GI function

Description:
May be helpful with symptoms of sluggishness and slow digestion function.
Formulation and Dosage:
Combined dosage of GI Aid (10 drops) and Liver/pancreas/spleen (5 drops) in 2-4 oz. of water.
Drink after food intake, up to two weeks or whenever it seems necessary.
Precautions: No precautions or side effects if used with the recommended dosages.

4. *Overall improvement in digestion and absorption*

Description:
May be helpful with compromised GI functions and low thyroid function.
Formulation and Dosage:
Combined GI Aid (10-15 drops) and Thyroid (3-5 drops) in 2-4 oz. of water.
Precautions: No precautions or side effects if used with the recommended dosages.

5. *Gut dysfunction associated with any inflammatory state*

Description:
May be helpful in the spectrum of GI conditions associated with the increase of permeability and inflammation. This may be enhanced by using an oligo-antigenic diet, anti-inflammatory agents, and factors that improve microbial balance.
Formulation and Dosage:
Vital 360 (10-15 drops) in 2-4 oz. of water.
Drink 3 times per day after food intake, up to two-three weeks.

Precautions: Always increase the amount of water up to 4-6 oz. for individuals with GI sensitivity to minerals in the solution.

∗ Disclaimer: These statements have not been evaluated by the Food and Drug Administration. This text is for educational purposes only and not intended to diagnose, treat, cure, or prevent any disease.

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