FOOD IS FUNCTION

THE FOOD-IMMUNE CONNECTION

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What we will discuss today…

FOOD AS INFORMATION
• Power to heal or harm your immune system

IMMUNE SYSTEM BASICS
• What is inflammation?
• Food allergy vs food sensitivity

NUTRIENTS GOOD FOR IMMUNE FUNCTION
• Anti-inflammatory diet
• Vitamin D, Vitamin A, Zinc, Essential fatty acids
FOOD AS INFORMATION
Food is Information

Food imprints our cells with information

- Healthy food: cells work properly
- Unhealthy food: poor cellular function that can damage the body, such as inflammation
- Chemicals, additives and toxins change cell function
Dietary Signal molecules

Nutritional Anti-inflammatory

Dietary Proinflammatory Signal (trauma, stress)

Food Allergen

Signals Sent Through Cell

Protein Activation

DNA

mRNA

Anti-inflammation

Protein

Inflammation

mRNA

Free Radicals

Receptor Complex

p50

IκB-α

p65

Receptor Complex

p50

IκB-α

p65

p50

IκB-α

p65

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Functional Nutrition

Food is much more than calories

“All calories are not created equal”

Look at food and nutrients in terms of function

Immune cells are especially sensitive to dietary compounds
Phytonutrients

- Phyto = Plant
- Example: Resveratrol activates longevity gene
- Example: Berry components such as anthocyanins protect your genes from damage
Bottom line:
A component of caramel food coloring found in beer and barbeque sauce, suppresses the immune system.

What we eat is arguably linked to how robust our immunity is, but confusion still reigns as to the molecular details of how various nutrients and components in our diet can specifically affect our complex immune system. An exciting paper by Schwab et al. on page 1735 in this issue (1) shows that 2-acetyl-4-tetraydroxybutylimidazole (THI), a component of caramel food colorant III used in food products including beer and barbeque sauce, suppresses immunity by increasing the amount of a lipid called sphingosine 1-phosphate in tissues of our immune system. Sphingosine 1-phosphate has attracted much attention recently as a regulator of the immune and cardiovascular systems (2). This study is of considerable interest as it draws attention to dietary modulation of immune function by the regulation of lipid metabolism.

Many studies have focused on the pharmacological regulation of the cell surface receptors for sphingosine 1-phosphate (G protein–coupled receptors called S1P1–5; (3)). In contrast, our knowledge of sphingosine 1-phosphate metabolism is scant (4). The lipid is present in all eukaryotes, and although it is produced intracellularly during sphingolipid metabolism, it is also secreted by some of the cells that produce it. In mammals, a steep concentration gradient of sphingosine 1-phosphate exists between the vasculature and tissues, thereby exposing resident vascular and immune cells bearing a cognate receptor to excess ligand (5). As lymphocytes traffic in and out of the vascular system, they must navigate this steep sphingosine 1-phosphate gradient. Schwab et al. discovered that THI attenuates the activity of sphingosine 1-phosphate lyase in lymphoid tissues. This intracellular enzyme irreversibly degrades sphingosine 1-phosphate into phosphoethanolamine and 2-hexadecenal. Thus, by neutralizing the concentration gradient of sphingosine 1-phosphate, THI perturbs T cell egress, causing a logjam in lymphoid tissue. Immunosuppression ensues (5).

Naive lymphocytes circulate through lymphatic and vascular conduits (see the figure). In addition, they traffic in and out of peripheral tissues by crossing the vascular and lymphatic endothelial cell barriers.

Modulation of lymphocyte trafficking is a new approach to control immune pathology. Recent work with the experimental compound FTY720, which binds to the extracellular domain of sphingosine 1-phosphate receptors, has revealed an unexpected role of sphingosine 1-phosphate in the control of lymphocyte traffic (6). Expression of the sphingosine 1-phosphate receptor S1P1 (formerly known as EDG-1) in T cells is critical for their proper egress from lymph nodes, Peyer’s patches, and the thymus (7). This receptor is expressed on the surface of T cells when the extracellular concentration of sphingosine 1-phosphate is low; however, the receptor is rapidly internalized by cells upon exposure to the higher concentration of the ligand (8). The concentration of sphingosine 1-phosphate is extremely high in the blood (0.4 to 1.5 μM)—about 2
The effect of food on genetic expression and cell function is a major cause of our epidemic of Chronic disease.
IMMUNE SYSTEM BASICS

Immune cells, Inflammation, and Food Sensitivities
Immune System Basics

• An army of cells that defend your body from invaders of any kind

• Microbes: bacteria, yeast, parasites, spirochetes like lyme

• Toxins

• Anything that looks foreign, including food
Immune System Basics

The immune system is divided into 2 parts:

1. **The innate immune system**: First line of defense, ready to go

2. **The adaptive immune system**: Is primed by the innate immune system, needs time to respond (hours to days), and has memory
Innate Immune System

• **Physical barriers**: tight junctions in the skin and mucus membranes.

• **The intestinal lining**: most important barrier for preventing food sensitivities.
Adaptive Immune System

**B lymphocytes**: antibody producing cells

- Antibodies are like “bullets” to kill invader
- 4 kinds of antibodies: IgG, IgE, IgA, IgM

**T lymphocytes**: cytotoxic killer cells

- “hand to hand combat”
- Creates lots of inflammation
What is Inflammation?

• Release of chemicals and proteins from immune and other cells
• Role: Defend the body and attack the foreigner
• But also irritates and can cause damage to your tissue
• Brief: supposed to turn on and then off again when “danger” is over
Chronic Inflammation

• Effects every cell in your body

• Local: pain in muscles (Fibromyalgia) and joints (Arthritis)

• Systemic inflammation:
  • Symptoms: brain fog, fatigue, feeling puffy
  • Example: Fat cells
  • Find the source: rule of tacks
Can Food Cause Inflammation?

• You bet!

• Food triggers release of:
  • Inflammatory molecules
  • Antibodies IgE, IgG, IgA (“bullets”)

• Food allergy vs food sensitivity: different kinds of immune reactions
Food Allergy

- Very specific immune response that is measurable by a conventional allergy doctor
- Elevated IgE levels
- Histamine release from mast cells
- Obvious symptoms immediately after eating the food: sniffles, tongue swelling, hives, anaphylaxis
Food Sensitivity

• You feel better when you don’t eat the food, and feel worse when you do.

• Can be any symptom:
  • Gut: gas, bloating, constipation, diarrhea
  • Systemic inflammation: feeling puffy, brain fog and fatigue  Skin rashes or headaches
  • Arthritis or muscle pain
Testing for Food Sensitivities

• The best way is with an elimination diet
  • Remove the top 5: gluten, dairy, soy, corn and eggs for 3 weeks.
  • Reintroduce 1 at a time every 4 days, to see if there is any reaction
  • When you reintroduce, eat the food several times a day for 2-3 days and observe. If you have a reaction right away, no need to keep eating it.
Lab Testing for Food Sensitivities

• Don’t have tests for all the possible immune reactions
• IgG food allergy testing is the most commonly used
  • Good option for children because hard to do elimination diet
  • Not good for gluten
• In adults I always do elimination diet, too.
HOW DO YOU DEVELOP A FOOD SENSITIVITY?
Let’s start with the food…

- All food has protein, even vegetables.
- Protein has amino acid sequences.
- Immune cells recognize self vs not self by reading the amino acid sequences on the surface of every cell, microbe or compound.
- I call these “name tags”
Name Tags on Food

• All the cells in your body have name tags (“self”) that your immune cells recognize.
• This is good so you don’t attack yourself.
• Any foreign name tag will cause an immune reaction.
• Every food has a name tag, too
• Your digestive system is designed to destroy the name tag on the food and prevent it from entering your body.
Healthy Gut Scenario

- Food is completely digested in the stomach
- All of the “name tags” that make the food recognizable by the immune system are destroyed by enzymes and acid
- **Healthy intestinal lining and barrier:**
  - The immune cells inside your body are not exposed to the “name tags” from the food
- Low risk of developing food sensitivity
Problem: Weak Intestinal Lining

• The cells that line the intestines are supposed to be connected tightly together
• Instead the “glue” is destroyed and spaces open up between the cells
• Large pieces of food, microbes and toxins can get into the body
• Called **Leaky Gut Syndrome** or Increased Intestinal Permeability
Nadine Cerf-Bensussan & Valérie Gaboriau-Routhiau
The immune system and the gut microbiota: friends or foes?
Nature Reviews Immunology 10, 735-744 (October 2010)
Leaky Gut Syndrome Scenario

• You have poor digestion
• You have a leaky gut
• Large pieces of food, with name tags intact, leak through the intestinal wall into your body
• Immune cells react to these name tags
• Symptoms typical of a food sensitivity
Treating Food Sensitivities

• When someone has multiple food sensitivities, assume they have Leaky Gut syndrome
  • Restore healthy intestinal flora
  • Support digestion of the “name tags”
  • Remove problem foods for at least 6 months, usually longer because immune cells have memory.
Food to Heal the Intestinal Lining

• Cultured foods: yogurt, kefir
  • With live active cultures of lactobacillus, bifidobacteria and saccharomyces boulardii
• Fermented foods: kimchi and sauerkraut
• Prebiotics: vegetables and fiber.
  • Fructo-oligo-sacharides (FOS), which are compounds found in onions, garlic, leeks, rye, chicory, blueberries, and bananas.
  • Inulins, which are found in chicory and artichokes.
Food to Heal the Intestinal Lining

• Ghee and Coconut oil

• Glutamine:
  • Can be found in all animal protein, such as chicken, beef, and dairy
  • Also in beans, cabbage, beets, spinach, and parsley, so don’t focus only on animal sources.
The important role of intestinal flora in maintaining healthy immune function and in reducing inflammation.
Leaky Gut and Autoimmune Diseases

Alessio Fasano

Abstract

Autoimmune diseases are characterized by tissue damage and loss of function due to an immune response that is directed against specific organs. This review is focused on the role of impaired intestinal barrier function on autoimmune pathogenesis. Together with the gut-associated lymphoid tissue and the neuroendocrine network, the intestinal epithelial barrier, with its intercellular tight junctions, controls the equilibrium between tolerance and immunity to non-self antigens. Zonulin is the only physiologic modulator of intercellular tight junctions described so far that is involved in trafficking of macromolecules and, therefore, in tolerance/immune response balance. When the zonulin pathway is deregulated in genetically susceptible individuals, autoimmune disorders can occur. This new paradigm subverts traditional theories underlying the development of these diseases and suggests that these processes can be arrested if the interplay between genes and environmental triggers is prevented by re-establishing the zonulin-dependent intestinal barrier function. Both animal models and recent clinical evidence support this new paradigm and provide the rationale for innovative approaches to prevent and treat autoimmune diseases.

Keywords

Antigens · Autoimmunity · Gut permeability · Immune response · Tight junctions · Zonulin

Introduction

The intestinal epithelium is the largest mucosal surface providing an interface between the external environment and the mammalian host. Its exquisite anatomical and functional arrangements and the finely-tuned coordination of digestive, absorptive, motility, neuroendocrine, and immunological functions are testament of the complexity of the gastrointestinal (GI) system. Also pivotal is the regulation of molecular trafficking between the intestinal lumen and the submucosa via the paracellular space. The dimensions of the paracellular space are estimated to be between 10 and 15 Å, suggesting that under physiological circumstances, solutes with a molecular radius exceeding 15 Å (~3.5 kDa) will be excluded from this uptake route. Macromolecule trafficking is dictated mainly by intestinal paracellular permeability, whose regulation depends on the modulation of intercellular tight junctions (TJ). A fast growing number of diseases, including autoimmune diseases, are recognized to involve alterations in intestinal permeability related to changes in TJ competency.

Classical Theories on the Pathogenesis of Autoimmune Diseases

Soon after autoimmune diseases were first recognized more than a century ago, it was believed that their development was associated with viral and bacterial infections. The connection between infection and autoimmune disease is often explained by a mechanism known as "molecular mimicry," whereby microbial antigens are postulated to resemble self-antigens [1]. The induction of an immune response to the microbial antigens results in a cross-reaction with the self-antigens and the induction of autoimmunity.
The importance of the intestinal barrier in regulating the innate and adaptive immune system

The intestinal epithelium is a single-cell layer that constitutes the largest and most important barrier against the external environment. It acts as a selectively permeable barrier, permitting the absorption of nutrients, electrolytes, and water while maintaining an effective defense against intraluminal toxins, antigens, and enteric flora. The epithelium maintains its selective barrier function through the formation of complex protein-protein networks that mechanically link adjacent cells and seal the intercellular space. The protein networks connecting epithelial cells form 3 adhesive complexes: desmosomes, adherens junctions, and tight junctions. These complexes consist of transmembrane proteins that interact extracellularly with adjacent cells and intracellularly with adapter proteins that link to the cytoskeleton. Over the past decade, there has been increasing recognition of an association between disrupted intestinal barrier function and the development of autoimmune and inflammatory diseases. In this review we summarize the evolving understanding of the molecular composition and regulation of intestinal barrier function. We discuss the interactions between innate and adaptive immunity and intestinal epithelial barrier function, as well as the effect of exogenous factors on intestinal barrier function. Finally, we summarize clinical and experimental evidence demonstrating intestinal epithelial barrier dysfunction as a major factor contributing to the predisposition to inflammatory diseases, including food allergy, inflammatory bowel disease, and celiac disease.

(J Allergy Clin Immunol 2009;124:3-20.)

Key word: Intestinal epithelium

The intestinal epithelium is a single layer of cells lining the gut lumen and has 2 critical functions. First, it acts as a barrier to prevent the passage of harmful intraluminal entities, including foreign antigens, microorganisms, and their toxins. Its second
Case Study

• Amy is a 52 year old woman who came to see me because she was told she had fibromyalgia and rheumatoid arthritis and didn’t want to take medication

• All of her RA labs were normal

• She had lots of digestive symptoms: gas/bloating after eating, constipation

• She had been slowly gaining weight for 5 years and was now 20 pounds above her normal
Case Study (continued)

- She had terrible chronic pain in her feet and hands, worse in the morning, she could barely stand when she woke up
- At our first visit, her instructions were to
  - Begin an elimination diet: remove gluten, dairy, soy, corn and eggs for 3 weeks, with instructions on reintroducing each one to see if it caused symptoms
  - Send a CDSA (stool analysis) to the functional lab
Case Study Continued

• She came back 4 weeks later, ecstatic because the pain in her feet were gone:
  • She could get out of bed and stand right away without pain.
  • She did not reintroduce food yet because she felt so good. This is common.

• Stool test showed severe dysbiosis

• I sent her home with a dysbiosis treatment protocol, which includes herbs, probiotics and digestive enzymes
Case Study Resolution

- She returned 6 weeks later, and had lost 15 pounds
- She discovered:
  - When she ate corn or gluten, her pain came back the next morning
  - Her chronic sinus drip was gone and came back when she ate dairy
  - Soy gave her gas and bloating
  - Eggs were ok
Case Study Resolution

• She maintains a gluten, dairy, soy and corn free diet.
  • It’s been about 1 year.
  • When she cheats, the reaction is mild but still present.
• She never needed medication and never received a medical diagnosis of RA or any other disease.
NUTRIENTS THAT EFFECT IMMUNE FUNCTION

Sugar, fat, vitamin D, Vitamin A, selenium, zinc, probiotics
Factors Affecting Development of Immune Cells

- Genetics

- Environment:
  - Toxins
  - Inflammatory food: Sugar, saturated/trans fats

- Nutrient deficiencies:
  - Vitamin A and D, Zinc
  - Essential Fatty Acids
Optimizing Immune Function

• We certainly can’t change our genetics

• We can’t always control our environmental exposures

• What we can do is eat foods and nutrients that keep the immune system in good balance
“Look, you’re just going to have to cut back. Times have changed... Today’s kids are full of hydrogenated oils, trans fats, corn syrup, and all kinds of preservatives.”
“How are you not seeing this? Of course doughnuts are a hole food!”
Anti-inflammatory Diet: Sugar

• Sugar in the blood stimulates your immune cells to actively release inflammatory molecules that travel throughout your body causing damage and irritation.

• Avoid high glycemic foods like soft drinks, juices, foods made with white flour and processed sugar, candy, cookies, ice cream, etc.
High blood sugar causes release of inflammatory chemicals from immune cells

High glucose concentrations induce TNF-alpha production through the down-regulation of CD33 in primary human monocytes

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Anti-Inflammatory Diet: Fats

• Trans fats and saturated animal fats turn into inflammatory molecules in the body
• Omega 3 and 6 fats are turned into anti-inflammatory molecules
• Dietary fat fills every cell membrane in your body and influences function
  • Important for communication between cells, and this is critical for immune cells
Fatty acids work directly on the T cells and regulate function and inflammation.

**PROSTAGLANDINS LEUKOTRIENES AND ESSENTIAL FATTY ACIDS**

**Fatty Acids, Inflammation and Immune Responses**

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**ABSTRACT** Evidence obtained from experiments in vitro and in vivo suggests that certain unsaturated fatty acids (FA) may be safe and effective anti-inflammatory and immunomodulatory agents. Generation of a unique eicosanoid profile with different biological effects by administration of FA precursors other than arachidonic acid is one approach under investigation. In addition to their role as eicosanoid precursors, FA are of major importance in maintaining cell membrane structure, are key determinants of membrane bound enzyme activity and receptor expression. FA can exert these functions directly and therefore may themselves be important regulators of immune responses. For example, certain FA influence cytokine production and proliferation of human T lymphocytes in a manner that is direct and not due to their conversion to eicosanoids. The observations indicate that FA can modulate immune responses by acting directly on T-cells and suggest that alteration of cellular FA may be a worthwhile approach to control of inflammation.

Essential fatty acids (EFA) are essential not only because of their physiological importance, but because they must be derived in either direct or partially elaborated form from the diet. Thus, these acids may be classified as vitamins (indeed they were once called vitamin F).

Two groups of fatty acids (FA) are essential to the body: the ω6 (n6) series, derived from linoleic acid (18:2 n-6) and the ω3 (n3) series, derived from α-linolenic acid (18:3 n-3). FA provide energy, are an integral part of cell membranes, and certain ones are precursors for prostaglandins (PG), thromboxanes (TX), and leukotrienes (LT), collectively termed eicosanoids. Abundant experimental evidence supports the view that eicosanoids participate in development and regulation of immunological and inflammatory responses (1). Because rheumatoid arthritis (RA) is characterized by inflammation, disordered immune regulation and tissue injury, there is much interest in the role of eicosanoids in regulation of host defenses in RA patients. As the detrimental effects of therapy for RA may be more difficult to manage than the disease itself, there is a need for new, safe approaches to the treatment of these patients.

Generation of a unique eicosanoid profile with different biological effects by administration of FA precursors other than arachidonic acid (AA) is one approach under investigation. Although changes in eicosanoid production owing to alteration of FA intake form the basis of the current hypothesis for the anti-inflammatory effects of this type of treatment, it is likely that the precursor FA themselves alter immune responses. The n-3 FA eicosapentaenoic acid (EPA; 20:5) and docosahexaenoic acid (DHA; 22:6), prominent in fish oil lipids, inhibit formation of cyclooxygenase and lipooxygenase products derived from AA (2, 3). Diets enriched in fish oil reduce generation of platelet activating factor by peripheral blood monocytes (4) and reduce production of interleukin-1 (IL-1) and tumor necrosis factor (TNF) by stimulated peripheral blood mononuclear cells (5). Fish oil supplements have therefore been used with modest success to suppress inflammation in experimental animal models (6-7) and in patients with RA (8, 9).

Evidence obtained from experiments in vitro and in vivo suggests that other novel FA may be safe and effective anti-inflammatory and immunomodulatory agents. For example, certain botanical lipids, notably those extracted from seeds of the evening primrose and borage plants, contain relatively large amounts of gamma-linolenic acid (GLA; 18:3 n-6) which can be converted rapidly to dihomo-gamma-linolenic acid (DGLA; 20:3 n-6), the FA precursor of the monoenoic PG, PGE1, is such an eicosanoid and it has known anti-inflammatory and immunoregulating properties (10-12). These include suppression of diverse T lymphocyte functions such as proliferation, cytotoxicity and IL-2 production, PGE1 also suppresses polymorphonuclear leucocyte and monocyte activation.

An approach to PGE1 therapy, first suggested by Willis (13), is provision of PGE1 precursors such as GLA or DGLA. The extremely short half-life of natural
Anti-inflammatory diet: Antioxidants

- Your immune cells are constantly exposed to toxins, microbes, chemicals.
- Need a constant supply of antioxidants to quench the free radical fire
  - Vitamin C, E, Alpha Lipoic Acid
  - Glutathione which is made from sulfur foods containing cysteine, phytonutrients in fruits and vegetables
Foods Rich in Antioxidants

• Vitamin C:
  • Berries, cantaloupe, grapefruit, honeydew, kiwi, mangoes, nectarines, orange, papaya, strawberries, broccoli, Brussels sprouts, cauliflower, kale, red, green or yellow pepper, snow peas, sweet potato, tomatoes

• Vitamin E
  • Broccoli, carrots, chard, mustard and turnip greens, pumpkin, red peppers, spinach, nuts and sunflower seeds, mangoes, papaya
Foods Rich in Sulfur

• Sulfur rich food:
  • Contains cysteine to make glutathione
  • Cysteine is found in most high protein foods:
    • Animal sources: pork, chicken, turkey, duck, deli meat, eggs, milk, whey protein, ricotta, cottage cheese, yogurt
    • Plant sources: red peppers, garlic, onions, broccoli, brussels sprout, oats, granola wheat germ, sprouted lentils
Vitamin D

- There are Vitamin D receptors in all immune organs and cells
- Vitamin D influences development of all your immune cells and keeps them in balance
- Most people are deficient unless they supplement
- Food sources: Not many
  - Naturally occurring: Cod liver oil and other fatty fish
  - Added to dairy products
Good review of the effects of Vitamin D on the immune system and the importance in autoimmune diseases

Review
Insights into endocrine-immunological disturbances in autoimmunity and their impact on treatment

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Abstract
The neuroendocrine immune (NEI) system is regarded as a fundamental network for the maintenance of health status (homeostasis), and it plays an important role in several systemic diseases, including autoimmune disorders. Among the major players of NEI pathways are steroid hormones of the adrenal (cortisol) and gonadal glands (sex hormones), neurohormones such as melatonin, and more recently the vitamin D endocrine system. Estrogens, melatonin and chronic stress (inducing decreased adrenal glucocorticoid release over a long time) strongly modulate the NEI system and stimulate the immune response. The vitamin D endocrine system is regarded as a potential immunosuppressive factor. Consequently, estrogens (especially in patients affected by B-cell-driven immunity) and melatonin should be avoided, and glucocorticoids (as replacement therapy) and vitamin D are allowed in the treatment of autoimmunity.

Introduction
The neuroendocrine immune (NEI) system is generally considered a fundamental network and its integrity is essential for the maintenance of health status in humans [1]. As a consequence, several systemic diseases, including autoimmune disorders, originate from the altered balance/activation of the NEI system.

Modulators of the immune system include different hormones, and major players of NEI pathways are steroid hormones of the adrenal and gonadal glands, as well as neurohormones such as melatonin (MLT) [1]. Steroid hormones are not stored in endocrine glands in the form of the final bioactive hormones, but their precursor cholesterol is metabolized by different enzyme steps leading to cortisol, the bioactive highly antiinflammatory endogenous glucocorticoid. The enzyme steps are regulated by microenvironmental factors such as cytokines. In the tissue, cortisol is degraded to cortisone, which can be reactivated by the 11β-hydroxysteroid dehydrogenase type 1 [2]. In addition, the adrenal glands produce the major androgen precursors dehydroepiandrosterone and androstenedione, which can be converted into active sex hormones such as testosterone and estrogens in peripheral tissues.

Gonadal glands mainly synthesize sex hormones such as testosterone (in the testicles) and estrogen (in the ovaries), again from the precursor cholesterol. Testosterone can also be viewed as a precursor of estrogens in tissue with high aromatase activity (that is, rheumatoid synovial tissue).

Interestingly, another hormone arising from cholesterol, namely vitamin D and its endocrine system, is involved in various biological processes that modulate immune responses (mainly immunosuppressive), and has an important role in autoimmune diseases [3].

Role of glucocorticoids in autoimmunity and inflammation
Several factors are involved in the pathogenesis of autoimmune rheumatic diseases, including genetic aspects, chronic infections, sex hormones (estrogens) and stress (Figure 1).

In particular, stress (that is, interpersonal, severe surgery, chronic infections) is now recognized as an important risk factor [4]. Patients with insufficient stress response axes demonstrate paradoxically decreased stress responses and, consequently, proinflammatory side effects [5]. The loss of adequate stress responses is reflected by low serum levels of cortisol and also low concentrations of norepinephrine in the tissue (nerv fiber loss) [1,6]. Minor stress and probably also
Finally realizing the importance of Vitamin D in chronic disease.
Vitamin A

• Chemical name: Retinol, retinoic acid, retinyl palmitate (synthetic), retinoids

• Beta Carotene is sometimes called Vitamin A because it is converted to Vitamin A in the body

• Required for normal immune cell function
Foods High in Vitamin A

• Foods high in vitamin A
  • Beta carotene is in fruits and vegetables:
    • Asparagus, beets, broccoli, carrots, green peppers, kale, turnips and collard greens, pumpkin, squash, spinach, sweet
  • Preformed Vitamin A:
    • Liver and other organs: Cod liver oil
    • Fortified Dairy
Zinc

• Important role in normal growth and development of immune cells

• Beware: too much zinc can impair immune function, too.

• Food sources:
  • Oysters, red meat, poultry, beans, nuts, seafood, whole grains, dairy products, peas
In Conclusion:

1. Remember, you are what you eat!
2. Test yourself to determine if you have food sensitivities and remove those foods.
3. Avoid foods high in sugar, saturated animal and trans fats. These foods create inflammation.
4. Choose foods rich in probiotic cultures, Vitamins A, D, zinc, sulfur, antioxidants. Supplement when necessary.
5. Make sure your gut is healthy. If you have any digestive symptoms or if you learn you have many food sensitivities, consider doing a stool analysis from an integrative/functional medicine practitioner.