Nutritional Influences on Inflammation of GI Disorders

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SoCal DIFM
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Key Message

A Healthy Gut is critical to being able to achieve wellness
Energy Transfer

The World is Fat
Barry Popkin
W. R. Kenan, Jr. Distinguished University Professor
Department of Nutrition

Australian Youth Gain Fat, Shift Body Shape
Obesity

Altered Metabolism
Increase Chronic Disease
Affects future generations
Genetic Miss Match

Systems Biology

Cells
Tissues
Organs
Systems
Body

300 Billion New Cells Every Day
200 Million Per Minute

Advanced Integrative Functional Nutrition Practitioner
Nutritional Consultation
GET:
Does this person have an unmet individual need?

RID:

GET:
Does this person need to be rid of something toxic, allergic, or infectious?

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Learning Objectives:

1. Review the nutritional-related mechanisms of chronic inflammation

2. Identify the two primary nutrient groups that modulate acute and chronic inflammation

3. Name three components of healthy gut ecology that influence nutritional status

4. Consider adding this information for modulating GI inflammation to your nutrition tool box
Inflammation Mechanism #1-a

• **Fatty Acid Metabolism**
  • **Gut Barrier Structure**
    • Eicosanoids
    • Prostaglandins
Leaky Gut & Chronic Inflammation
Inflammation Mechanism # 1-b

- **Fatty Acid Metabolism**
- **Gut Barrier Structure**
  - **Eicosanoids**
  - **Prostaglandins**

Primary physiological mechanisms of inflammation and immune dysregulation with nutritional influences:

EICOSANOIDs
EICOSANOID METABOLITES

SPECIALIZED PRORESOLVING MEDIATORS (S.P.M.)
Inflammation Mechanism #2

• Gut Microbiome

“GUT PARTNERS”

- Microbiota
- ~3 pounds adult
- Produce Vitamins
- Stimulate Immune
- Anti-inflammatory

Human microbiome

Human genome

23,000 genes

1,000,000+ genes
The gut microbiota as an environmental factor that regulates fat storage

GUT Microbiota Regulates Fat Storage

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HEAD-ORAL CAVITY

ORAL CAVITY

• MOUTH / LIPS
• TEETH
• PERIODONTAL TISSUE / GUMS
• DIGESTIVE GLANDS: Tonsils, Adenoids, Parotid

Oral Diseases
Background: The potential impact of periodontal disease on diabetes has been the subject of intense investigation and debate for several decades. Despite conflicting reports in the literature, recent findings suggest that periodontal disease is more common in patients with diabetes mellitus than in those without the condition. The purpose of this review is to provide the reader with the best available evidence pertaining to this association.

Methods: Over 200 articles have been published over the past two decades examining the relationship between diabetes mellitus and periodontal diseases. This review summarizes the results of these studies, drawing upon those that met the inclusion criteria for prevalence, extent, and severity of periodontal disease. The studies were selected on the basis of methodological rigor, appropriate level of care, and consistency of results. A systematic, critical review of the literature was conducted, and the conclusions of the authors were discussed.

Results: The results of this review indicate that diabetes increases the risk of periodontal disease and that periodontal disease also affects glycemic control in diabetic patients. The mechanisms through which periodontal disease and diabetes interact are not fully understood, but the results of this review suggest that periodontal disease may be a contributing factor in the development and progression of diabetes mellitus.

Conclusions: This review provides the best available evidence for the relationship between diabetes mellitus and periodontal disease. Further research is needed to clarify the mechanisms through which these conditions interact and to develop effective strategies for their management.

The Burden of Oral Disease

Oral health is often taken for granted, but it is an essential part of our overall health and well-being. Oral health encompasses the structures and functions of the mouth, including the teeth, gums, tongue, and related muscles and nerves. Oral health is a complex and dynamic system that is influenced by a variety of factors, including genetics, environment, lifestyle, and behavioral choices. Oral health is often assessed through the examination of the teeth, gums, and mouth, and it is an important indicator of overall health, as oral health is linked to the development of systemic diseases, such as heart disease, stroke, diabetes mellitus, and respiratory infections. Oral health is also a key factor in the quality of life, as oral health can affect the ability to function, communicate, and interact with others.

Diabetes Mellitus and Periodontal Diseases

Diabetes mellitus is a chronic metabolic disorder that affects the body's ability to regulate blood sugar levels. Diabetes is characterized by high blood sugar levels, which can occur due to a number of factors, including insulin resistance, an increase in the production of glucose, and a decrease in the ability of the body to use glucose as an energy source. Diabetes is divided into two main types: type 1 diabetes and type 2 diabetes. Type 1 diabetes is an autoimmune disorder, in which the immune system attacks and destroys the insulin-producing cells in the pancreas, leading to high blood sugar levels. Type 2 diabetes is a metabolic disorder, in which the body becomes resistant to the effects of insulin, leading to high blood sugar levels. Diabetic patients are at risk of developing periodontal disease, which is an infection of the gums and other structures around the teeth.

Periodontal disease is an infection caused by bacteria that colonize the mouth and interact with the immune system to cause inflammation and tissue destruction. Periodontal disease is characterized by the presence of bacteria, which are present in the mouth and can cause an inflammatory response. Periodontal disease is often associated with a decrease in the number of blood cells, which can result in anemia, a reduction in the size of the bone, and an increase in the risk of infection. Periodontal disease is often characterized by pain, swelling, and bleeding, and it can lead to the loss of teeth and the destruction of the jawbone.

The relationship between diabetes mellitus and periodontal disease is complex, and the mechanisms through which these conditions interact are not fully understood. However, the results of this review suggest that periodontal disease may be a contributing factor in the development and progression of diabetes mellitus.

The Authors describe the current pathogenic concepts like the "immuno-" and "autoimmune" hypothesis and the "microbial hypothesis". A focal infection is a localized or generalized infection caused by the dissemination of culturable microorganisms or in cases where are present but in extremely small abundance. Less clear is the impact of periodontal diseases on glycemic control of diabetes and the mechanisms through which this occurs. Real clinical outcomes are presented with different clinical criteria applied to prevalence, extent, and severity of periodontal disease. The authors have supported their evidence with references to certain "classic" articles published in English over the past 20 years, with particular focus on primary research and review articles, after historical signs, symptoms, and symptoms of diabetes and periodontal disease.

Conclusions: This review provides the best available evidence for the relationship between diabetes mellitus and periodontal disease. Further research is needed to clarify the mechanisms through which these conditions interact and to develop effective strategies for their management.

Oral Health: Managing Cavities, Lost Teeth, Tooth Loss, and other problems at a glance 2017

Most Oral Diseases are Preventable

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The Burden of Oral Disease
Stomach Acid requires nutrients: Sodium, Potassium, Zinc, and/or Iodine.

- BILE ~1 quart/day
- Emulsifies Fat and Fat-Soluble Vitamins
- Toxin-Carrier
- Helps reduce “Bad” LDL Cholesterol
SMALL INTESTINE
ILEUM

COLON
- 8 feet long
- 2-3 BM’s / day
- Most Microbiota
- Fluid Absorption
- Nutrients

ASCENDING COLON
2. Two primary nutrient groups that modulate acute and chronic inflammation

- **Fats and Oils**
  - Beneficial Saturated Fats (Short Chain & Medium Chain)
  - Cholesterol
  - MUFAs
  - Omega 6
  - Omega 3

- **Phytonutrients**
Inflammation

Lipid Status

Fats & Oils & Phospholipids
Cholesterol

Omega 6: LA

Evening Primrose Oil
Black Currant Oil
Hemp Oil
(Borage Oil)

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Omega 3: EPA / DHA

Omega 3 EPA/DHA Fatty Acids
(DHA for brain, eyes, nerves)

Omega 9: Oleic Acid

Beneficial Saturated Fat

Grass Fed Butter
Organic Eggs
Coconut Oil
Inflammation Control Mechanisms

**Phytochemicals:** Phytochemicals are a large group of plant-derived compounds hypothesized to be responsible for much of the disease protection conferred from diets high in fruits, vegetables, beans, cereals, and plant-based beverages such as tea and wine.

3. Three components of healthy gut ecology that influence nutritional status

1. Pre-biotics: soluble fiber
2. Probiotics: fermented foods-supplements
3. Short Chain Fatty Acids (Butyrate)

Fiber & Cultured Foods

**Fiber:** Veggies, Beans, Fruit, Nuts, Seeds

**Cultured Foods:**
- Yogurt, Kefir
- Miso, Tempeh, Natto
- Sauerkraut, Kim Chee, Pickles
Pre-biotics

Common soluble fibers that promote beneficial bacteria:
- Glucomannan
- Oat Fiber/Bran
- Rice Bran
- Gum Acacia
- Chicory inulin, a highly effective prebiotic with the immunomodulating actions
- Larch arabinogalactan
- Purified yeast beta-glucan
- Modified Citrus Pectin
- Apple Pectin
- Galactooligosaccharides

Probiotics

Lactobacillus acidophilus
- Highly resistant to gastric acid, bile, pepsin, and pancreatin. Possesses more than 20 known peptidases and breaks down casein and gluten. Ferments lactose and monohexose a variety of other sugars and polysaccharides. Antagonizes a wide range of pathogenic bacteria. Reduces immunostimulatory concentrations of carcinogenic enzymes.
- Lactobacillus rhamnosus
  - Lactobacillus casei
    - Lactobacillus salivarius
    - Lactobacillus plantarum
    - Lactobacillus paracasei
      - Excellent acid-tolerance. Highly resistant to pancreatin. Ferments inulin and rhizome and produces high levels of lactic acid. Antagonizes C. difficile and Staphylococcus aureus as well as other pathogens. Contributes to a healthy vaginal microflora. Has supportive benefit in conditions ranging from allergic rhinitis to nonrotavirus diarrhea in children.
### Probiotics

**Lactobacillus brevis**
- A colonizing species producing lactate, carbon dioxide, ethanol, and acetate. Resistant to gastric acid, bile acids, and digestive enzymes. Excellent adherent properties. Increases production of interferon. Metabolically unique in the production of arginine deiminase to break down arginine and reduce polyamine production, compounds associated with vaginal dysbiosis and intestinal carcinogenesis.

**Bifidobacterium bifidum**
- Present in large numbers in a healthy colon. Populations are reduced in allergic infants and decline significantly with age. Enhances IgG and IgE responses to select antigens. Activates B cell IgA secretion. Enhances IgA response to C. difficile toxins A. Along with L. acidophilus, supports gut microflora during antibiotic therapy and reduces positive testing for C. difficile toxins.

**Bifidobacterium infantis**
- Frequently found in infants’ intestinal tracts, but rarely in older adults. Strong suppressive effect on Bacteroides vulgatus, a commensal bacteria thought to have a role in inflammatory bowel disease. Reduces proinflammatory cytokine production. Supports normal microflora and inflammatory cytokine ratios in patients with irritable bowel syndrome. Together with L. acidophilus, supports the gut microflora in very low birth weight infants decreasing the risk of necrotizing enterocolitis and promotes normal microflora in children with diarrhea.

**Bifidobacterium longum**
- Often the dominant Bifidobacterium species in humans. Ferments a broad spectrum of oligosaccharides. Resistant to high bile salt concentrations. Inhibits human neutrophil elastase which may be important to innate immunity and attenuate harmful intestinal inflammation. Inhibits enterotoxinogen E. coli receptor binding and translocation. Augments intestinal IgA secretory response to dietary proteins. Favorably modulates inflammatory cytokine response to respiratory antigens. Improves immunological and alleviates colitis.

**Bifidobacterium breve**
- Secretes compounds, such as lactosidase, that favorably modify intestinal microflora by reducing Bacteroides and Clostridium concentrations and degrading mucus. Stimulates Peyer's patch B cell proliferation and antibody production. Eliminates most Campylobacter jejuni in campylobacter enteritis restoring normal intestinal microflora. Antagonizes rotavirus and decreases rotavirus shedding in infants with rotavirus diarrhea.

**Streptococcus thermophilus**
- A transient species with a long history of use as a starter culture for yogurt and cheese. Highly adapted to lactose metabolism. Many fermentation end-products including formate, acetoin, acetylaldehyde, diacetyl, and acetate that inhibit pathogenic bacterial proliferation. Reduces DNA damage and premalignant lesion formation by protecting against carcinogens. Along with other probiotics supports normal microflora and gastrointestinal function in conditions ranging from rotavirus diarrhea in infants to remission in ulcerative colitis.

**Lactobacillus bulgaricus**
- A highly adapted, transient species closely related to L. acidophilus. Along with S. thermophilus, it has long been used in the production of yogurt and cheese. Supports normal cholesterol levels and reduces low density lipoprotein cholesterol oxidation. Suppresses proinflammatory cytokine production.

**Lactobacillus gasseri**
- Native to the human gut and vaginal tracts of healthy women and normally present in human breast milk. Produces hydrogen peroxide and bacteriocins inhibitory to Clostridium, Listeria, and Enterococcus. Protects against intestinal mitogens and carcinogens.

**Bifidobacterium lactis**
- Produces large amounts of anti-microbial formate. Enhances leukocyte tumor cell-killing properties and phagocytic activities. Increases numbers of total, helper, and activated T cells. B. lactis significantly increases serum and mucosal IgA responses to cholera toxin and tetanus toxoid.
Probiotics

Saccharomyces boulardii
Hardy, nonpathogenic yeast. Broad antimicrobial activities against C. difficile, toxigenic E. coli, Candida, and other gastrointestinal pathogens. Augments colon bifidobacteria populations and increases butyrate concentrations. Enhances brush border enzyme activities and improves gut barrier function. "Anti-Yeast"

INDEPENDENT TESTING OF COMPETITIVE PRODUCTS

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<th>Brand</th>
<th>Potency Claim per Capsule</th>
<th>Test Result</th>
<th>Percent of Label Claim</th>
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LIFESTYLE

Toxins
- FOOD
- AIR
- WATER
- ENDOGENOUS TOXINS
Obesity

Altered Metabolism

Increase Chronic Disease

Affects future generations

Genetic Match
Immune Compromised

- 11 year old girl
- Blood tests low immune factors x 2 years
  - WBC 2.5-3.2
  - IgA blood Low
  - Skin rashes
  - Sick frequently – abdominal cramping, bloating
- School Absent Sick Days > School Days
- Quarantined frequently – easy infection

Immune Compromised

- 11 year old girl
- Regular SAD Diet (hi milk, gluten)
- History of 2-3 Antibiotic Tx per year since 3 yo
- Fatigued
- Weak muscle
Immune Compromised

- 11 year old girl
- Integrative Medicine Approach
  - Previous Medical Blood Tests & Diagnosis
  - Stool Test
  - Elimination Diet: 3 months
    - Avoid dairy/casein
    - Avoid gluten
    - Avoid oranges
    - Avoid Soy
Initial Nutrition Intervention

• Eliminate gluten/dairy-casein/oranges

• 450 bil Probiotics (VSL#3) daily (in coconut yogurt)

*Probiotics*

• AMOUNT/QUANTITY MATTERS!
50 bil minimum/day

• Species type matters!

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Initial Nutrition Intervention

• Two months Elimination Diet (no gluten/dairy)

• 450 bil Probiotics (VSL#3) daily (in coconut yogurt)

• Evening Primrose Oil/EPA-DHA 2 cap (GLA 90 mg and 36 mg EPA/DHA)

• Vitamin D3 emulsified 2000 IU/day

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Immune Compromised

Healthy Happy
11 year old
Normal WBC

• Two months Elimination Diet (no gluten/dairy)

• 450 bil Probiotics (VSL#3) daily (in coconut yogurt)

• Evening Primrose Oil/EPA-DHA 2 cap (GLA 90 mg and 36 mg EPA/DHA)
• After reviewing all the initial nutrition data, what pattern do you see start to emerge?

• Also, be looking for inflammation evidence from Nutrition Physical Exam, labs, lifestyle, medical history.

• When you find the inflammation start looking for the etiology, or reason for the chronic non-resolving type of inflammation... diet, lack of sleep, toxins, nutrient insufficiencies (especially fatty acids and phytonutrients) and excess of poor nutrient foods, genetics,

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Essential nutrients

Conditionally essential nutrients

Omega 3
- ALA
- EPA
- DHA

Omega 6
- LA
- CLA
- GLA
- DGLA

Omega 9
- Oleic Acid
- "Bene Sats"

SCFA/Butyric
MCT

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A New Roadmap: IFMNT Radial

Biological Systems

Sign & Symptoms

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[Diagram of A New Roadmap: IFMNT Radial with various pathways and systems]

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Obesity to Lean Fit

- Altered Metabolism
- Increase Chronic Disease
- Affects future generations

Thank you!

A Healthy Gut is critical to being able to achieve wellness

“Nutritional Influences on Inflammation of GI Disorders”
diana@diananoland.com
Extra Clinical Pearls

Vagus Nerve

20% of vagus nerve fibers send information from the brain to the stomach

These signals control:
- Digestive enzymes secreted
- Gastric capacity
- Blood glucose

80% of vagus nerve fibers send information from the stomach to the brain

These signals control:
- Satiation (hunger)
- Regulation of metabolic function

WHAT THE VAGUS NERVE DOES

- Contact to the brain and is responsible for your "feel good" feeling
- Conveys information from the organs to the brain
- Regulates blood pressure and heart rate
- Affects the immune system
- Influences the endocrine system
- Supports the respiratory system
- Influences the digestive system
- Supports the cardiovascular system
- Influences the nervous system

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GI Disordered patients should always be screened for history for head/neck/back injuries, or birth trauma (like use of forceps), may be vagus nerve damage.