Back To Chiropractic CE Seminars

Nutrition: GI Disorders 101 ~ 6 Hours

Welcome to Back To Chiropractic Online CE exams:

This course counts toward your California Board of Chiropractic Examiners CE.
(also accepted in other states, check our website or with your Chiropractic State Board)

The California Board requires that you complete all of your CE hours BEFORE the end of your Birthday month. We recommend that you send your chiropractic license renewal form and fee in early to avoid any issues.

Exam Process: Read all instructions before starting!

1. You must register/pay first. If you haven't, please return to: backtochiropractic.net

2. Open a new window or a new internet tab & drag it so it's side-by-side next to this page.

3. On the new window or new tab you just opened, go to: backtochiropractic.net website.

4. Go directly to the Online section. DON'T register again.

5. Click on the Exam for the course you want to take. No passwords needed.

6. Follow the Exam instructions.

7. Upon passing exam, you'll be able to immediately download your certificate, and it'll also be emailed to you. If you don't pass, you must repeat the exam.

Please retain the certificate for 5 years. DON'T send it to the state board. If you get audited and lose your records, I'll have a copy.

I'm always a phone call away... 707.972.0047 or email: marcusstrutzdc@gmail.com

Marcus Strutz, DC
Back To Chiropractic CE Seminars

Douglas L. Weed, D.C.

Evaluating Gastrointestinal Disorders

A Functional Approach

Part 1

Disclaimer

The information contained in this lecture is intended for educational purposes only, and is derived from the published literature. It is intended only for the purposes of disseminating said information. It is not intended to diagnose or treat any disease or condition and does not replace a patient-physician relationship in evaluating, assessing, diagnosing, or treating any condition. By continuing with this presentation the reader acknowledges the understanding and intent of this discussion.

Course Objectives

Nutrition: Gastro-Intestinal Disorders ~ 6 HOURS Back to Chiropractic CE Seminars

Introduce doctors to the philosophy and methodology of a natural approach to non-pathological GI complaints

Educate physicians on a top-down methodology to assessing common GI problems

Provide doctors with an integrated approach to evaluating common GI disorders

Discuss currently available advanced testing currently available for common GI complaints
Integrate assessment with recommendations that doctors may easily implement

Presented by: Douglas L. Weed, D.C.

I.
Seminar Outline

- Introduction
- Integrated Philosophy and Assessment
- Digestion Physiology
- Digestion Assessment Questionnaire
- In-Office labs
- General Labs & GI Function
- Stomach Acid, Hypo and Hyperchlorhydria

INTRODUCTION

I got the bill for my surgery. Now I know why those doctors were wearing masks.

James H. Boren

Doctors are just the same as lawyers; the only difference is that whereas lawyers rob you, doctors rob you and kill you, too.

Ant*on Chekov*

Who Do I Trust?

"If excessive smoking actually plays a role in the production of lung cancer, it appears to be a minor one."

Dr. W.C. Heuper, NCI (New York Times, 4/14/1954)

"For the majority of people smoking has a beneficial effect."

Dr Ian MacDonald (Newsweek, November 1963)

There is growing evidence that smoking has pharmacological effects that are of real value to smokers.

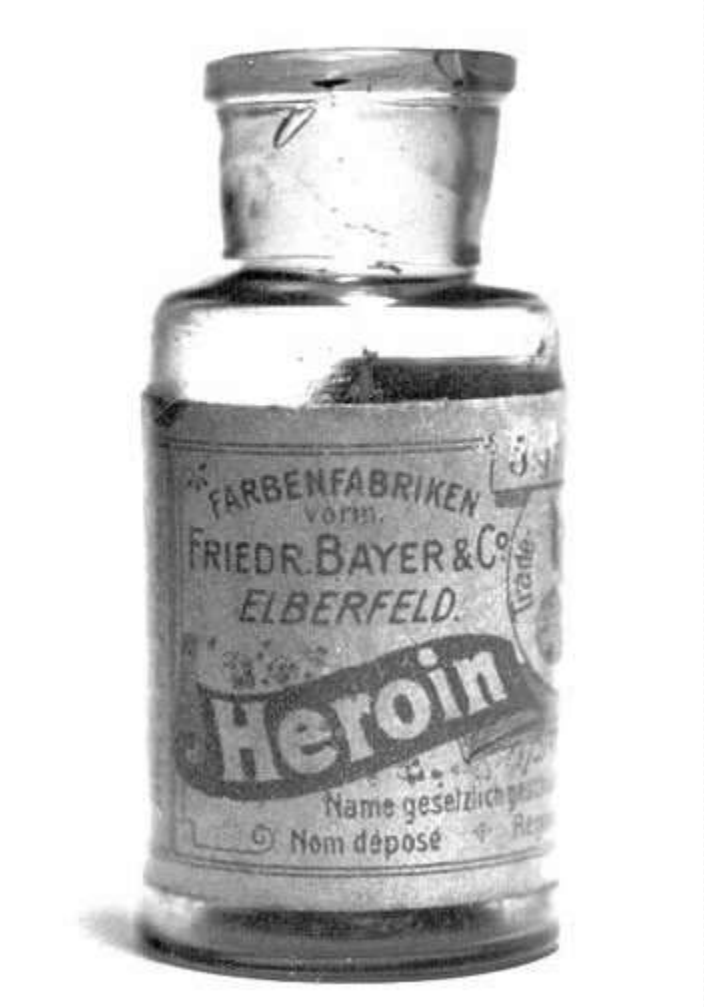
President of Philip Morris, Inc., 1962

"My doctor gave me six months to live, but when I couldn't pay the bill he gave me six months more."
Walter Matthau

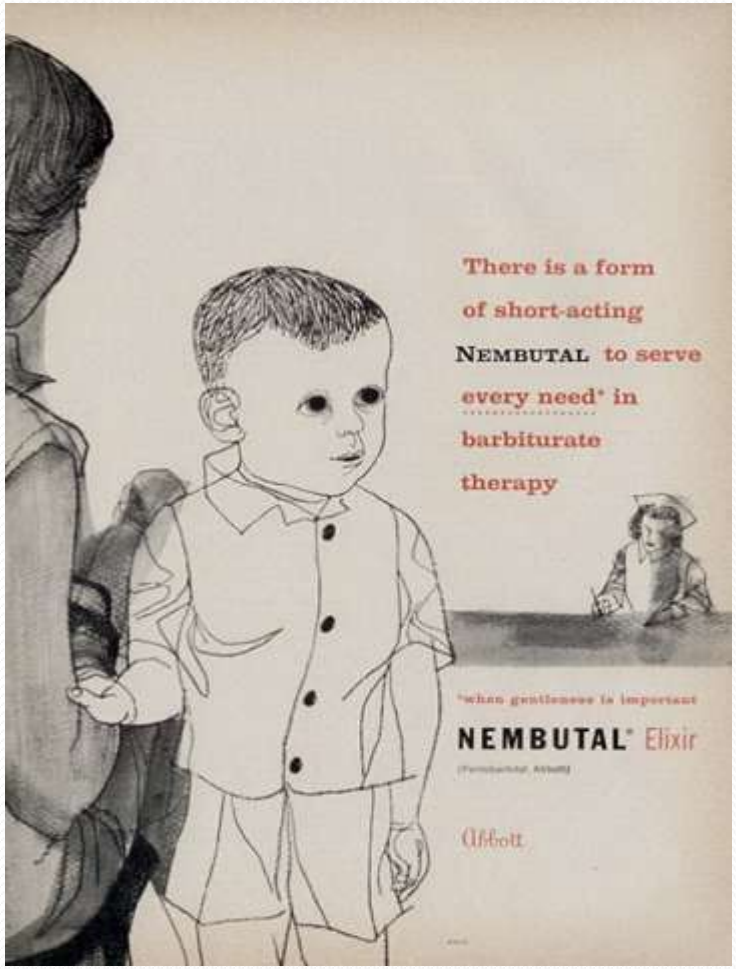

Gee, Dad, you always get the best of everything ...even
Marlboro!
Yes, you need never feel over-smoked ....that's the Miracle of Marlboro!
MARLBORO
CIGARETTES
YOUR CHOICE OF IVORY TIPS
PLAIN ENDS
BEAUTY TIPS (RED)


Viceroys
FILTER
the Smoke!
As your Dentist, I would recommend VICEROYS
VICEROY
Filter Tip
CIGARETTES
TWENTY


For a better start in life
start COLA earlier!
- Promotes Active Lifestyle!
- Boosts Personality!
- Gives body essential sugars!
How soon is too soon?
Not soon enough. Laboratory tests over the last few years have proven that babies who start drinking soda during that early formative period have a much higher chance of gaining acceptance and "fitting in" during those awkward pre-teen and teen years. So, do yourself a favor. Do your child a favor. Start them on a strict regimen of sodas and other sugary carbonated beverages right now, for a lifetime of guaranteed happiness.
The Soda Pop Board of America
1515 W. Hart Ave. - Chicago , ILL.


FARBENFABRIKEN
vorm.
FRIEDR. BAYER & Co.
ELBERFELD.
Heroin
Nom déposé

Barbiturates for Babies



Prescription Drugs – A Leading Killer in USA

- More than two million American hospitalized patients suffered a serious adverse drug reaction/year
- Over 100,000 died
- Reactions were due to the inherent toxicity of the drugs rather than allergic reactions.
- Did not include fatal reactions caused by accidental overdoses or errors in administration
- Including them adds another 100,000 deaths/year.
- Concluded that ADRs are the fourth leading cause of death
  - *Incidence of Adverse Drug Reactions in Hospitalized Patients, Journal of the American Medical Association (JAMA), Vol. 279. April 15, 1998, pp. 1200-05..*

NSAID Deaths

- 1/3 of deaths come from low-dose aspirin
- 16,500 deaths from people with RA alone
- Causes both upper and lower GI mortality
  - *NSAID-Associated Deaths-Byron Cryer MD*

American Journal of Gastroenterology, April 2005

VIOXX

- 55,000 deaths
- Increases B.P.
- Accelerates Atherosclerosis
- 80% Increase in Heart failure
- 500% increase in heart attacks
- Est. 160,000 M.I. and Stroke
  - *Article by Public Citizen's Health Research Group at worstpill.org*

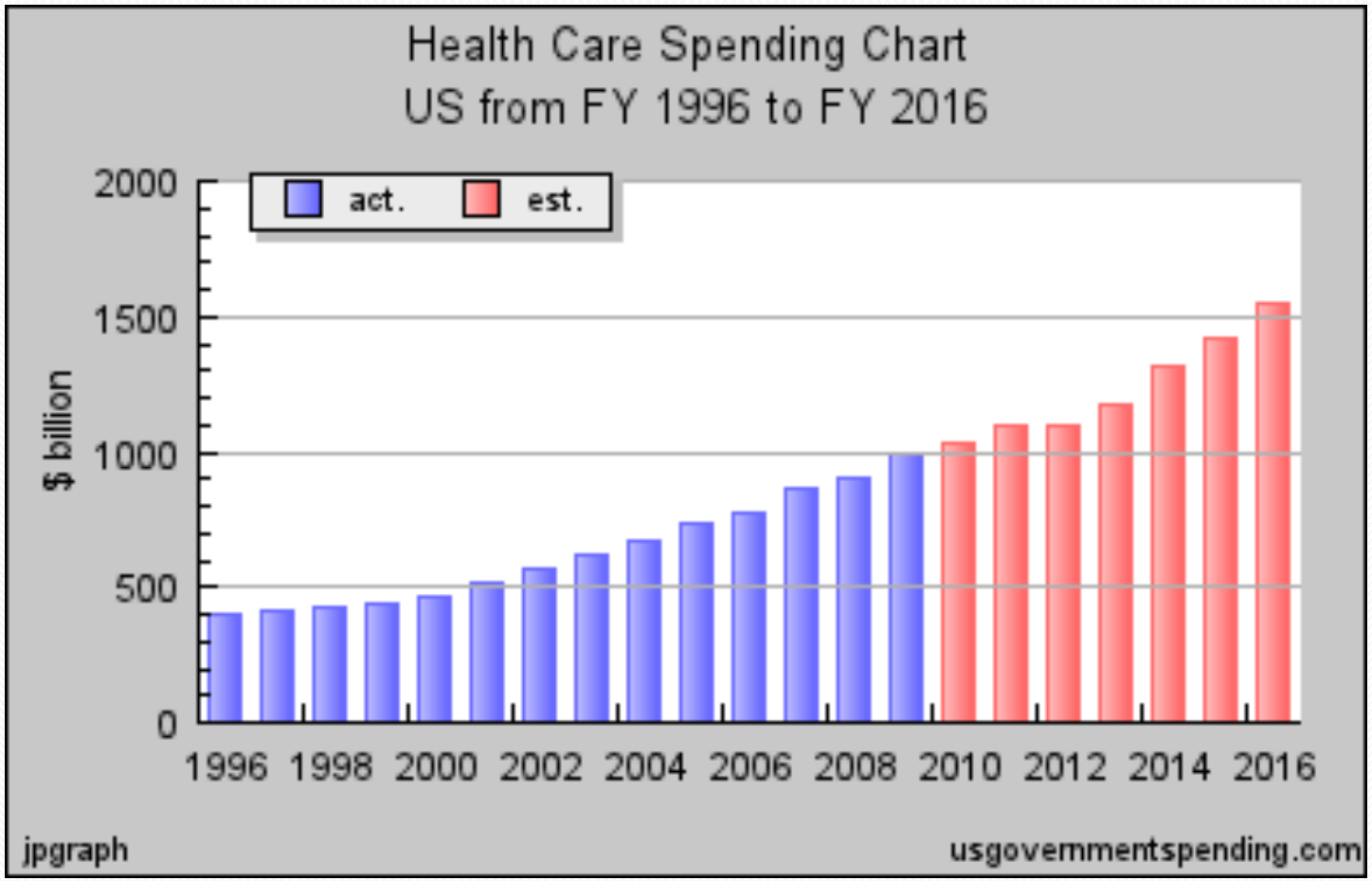
AVANDIA

- 43% higher risk of heart attack
- 500 M.I./month
- 300 Strokes per month
  - *New England Journal of Medicine, 2007*

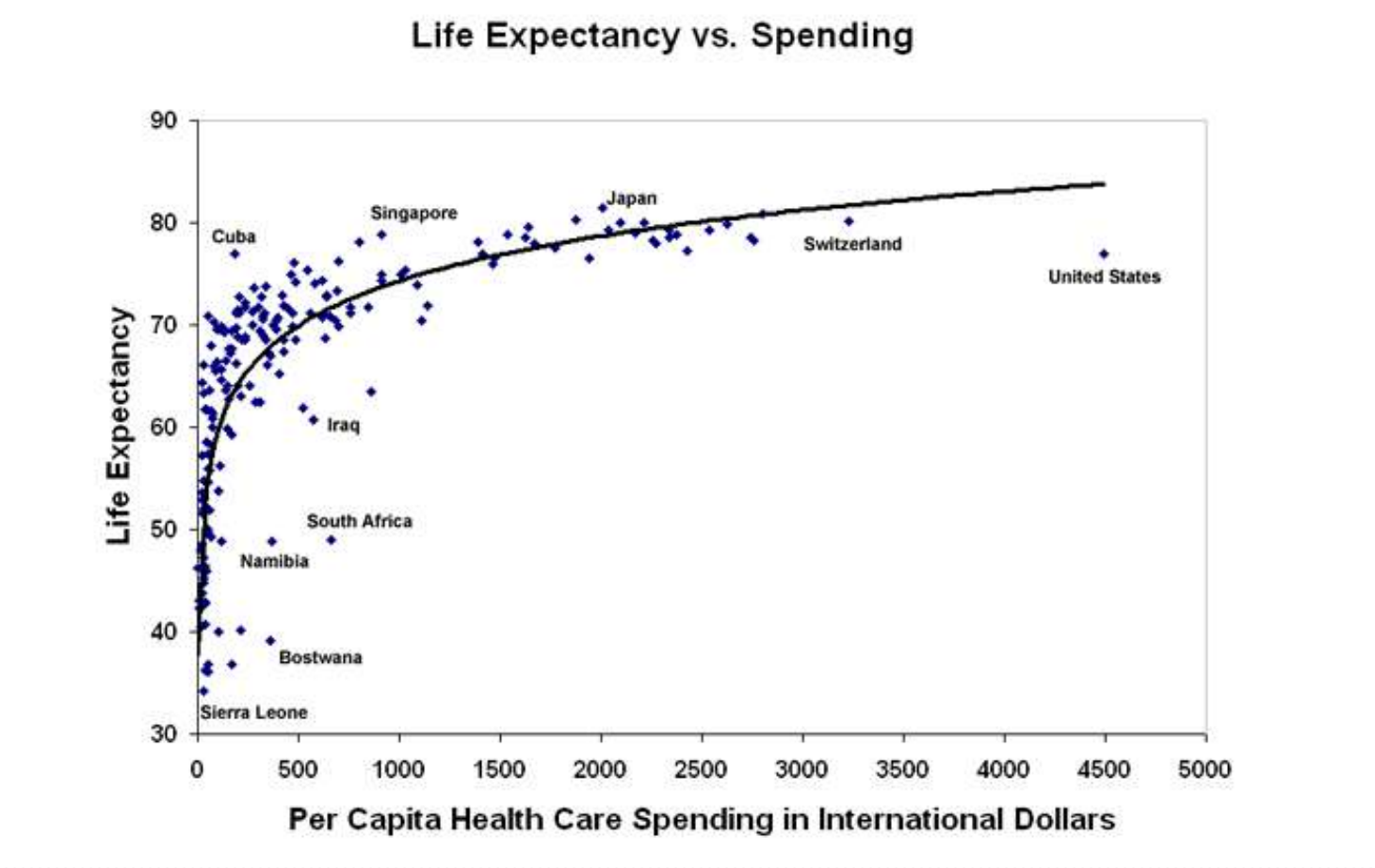
America has the best doctors, the best nurses, hospitals, medical technology, and breakthrough medicines in the world. There is absolutely no reason we should not have in this country the best health care in the world.

Bill Frist, MD

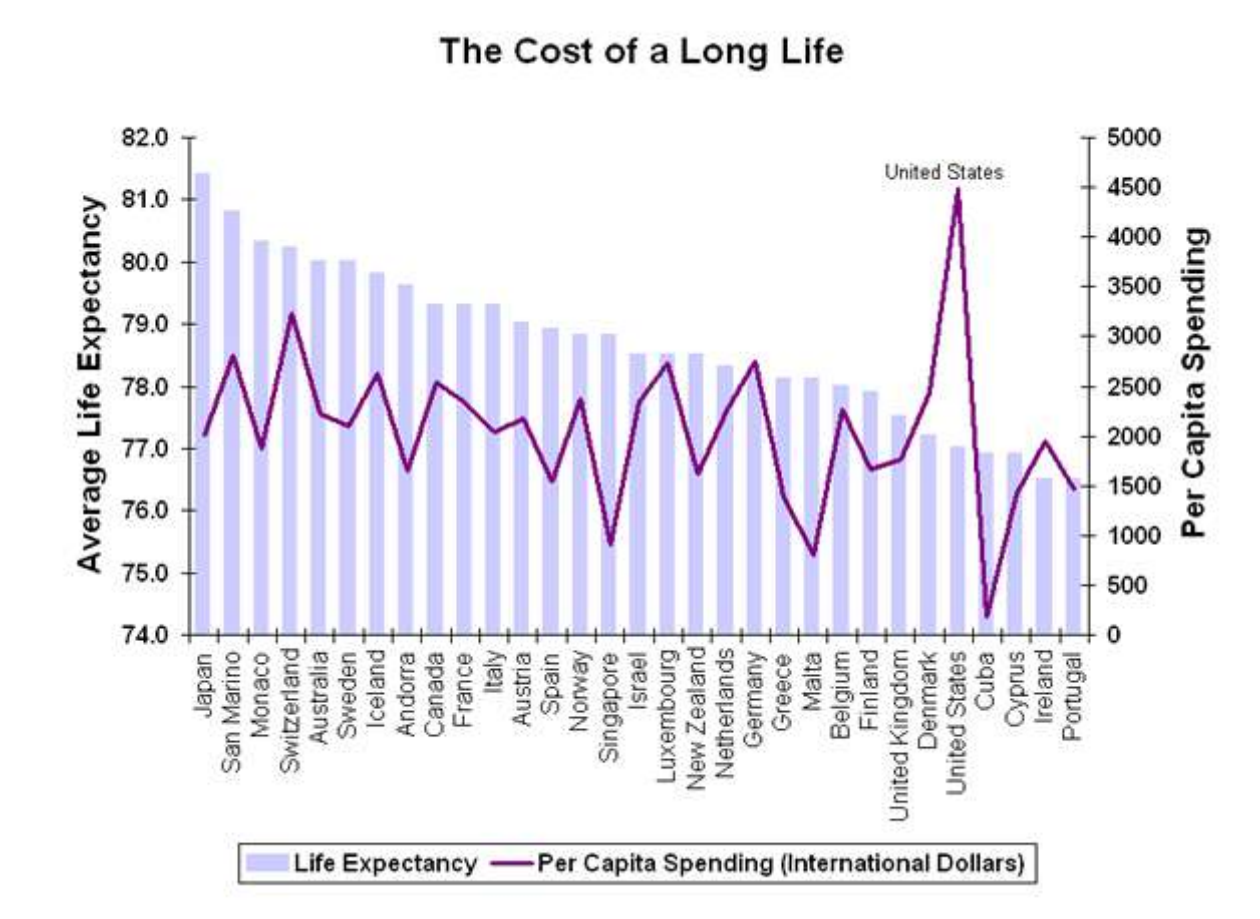
U.S. Healthcare Costs Rise Exponentially



Health Care Spending

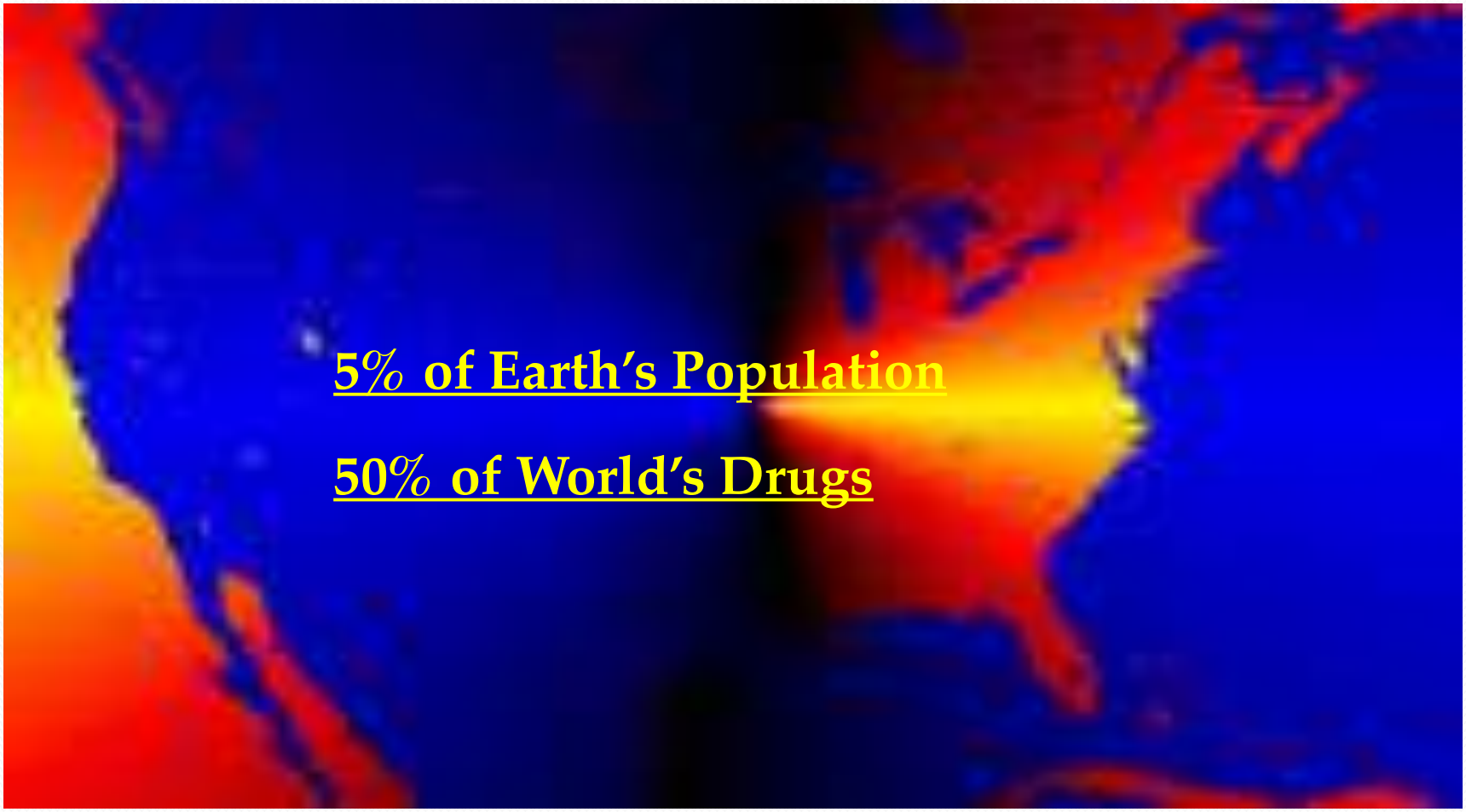


Cost / Longevity



How Healthy Are We?

- World Leaders In:
  - Heart Disease Deaths
  - Diabetes Deaths
  - Obesity
- 29th in Infant Mortality
- 121 countries have a lower mortality rate
- Highest health care costs in the world


5% of Earth's Population
50% of World's Drugs

Insanity…

To keep doing the same thing over and over and expecting different results

Albert Einstein

II.
INTEGRATED PHILOSOPHY & ASSESSMENT

It is more important to know what sort of person has a disease than to know what sort of disease a person has."

Hippocrates

Allopathic vs. Functional Philosophy

Allopathic

- Reductionist
- Simplify multiple problems
- Diagnosis Critical
- Drug to match diagnosis
- Must have disease
- Treats disease end-point
- Cookbook approach
- Organ Specific-Specialization
- Applies to "Green Medicine"

Functional

- "Wholism
- Web of Interactions
- Diagnosis not Holy Grail
- Biochemistry & Homeostasis
- Function vs. disease
- Treats root causes
- Highly individualized treatment
- Treats whole individual
- Evidence based

I'm 86 and my doctor used to tell me to slow down - at least he did until he dropped dead.

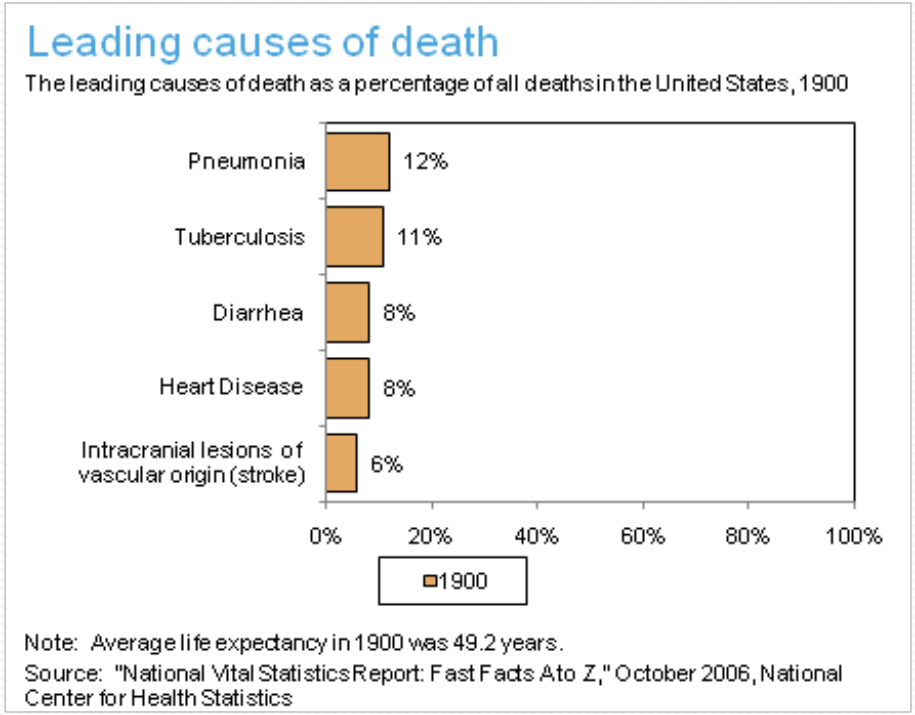
Cesar Romero

Why a Functional Philosophy?

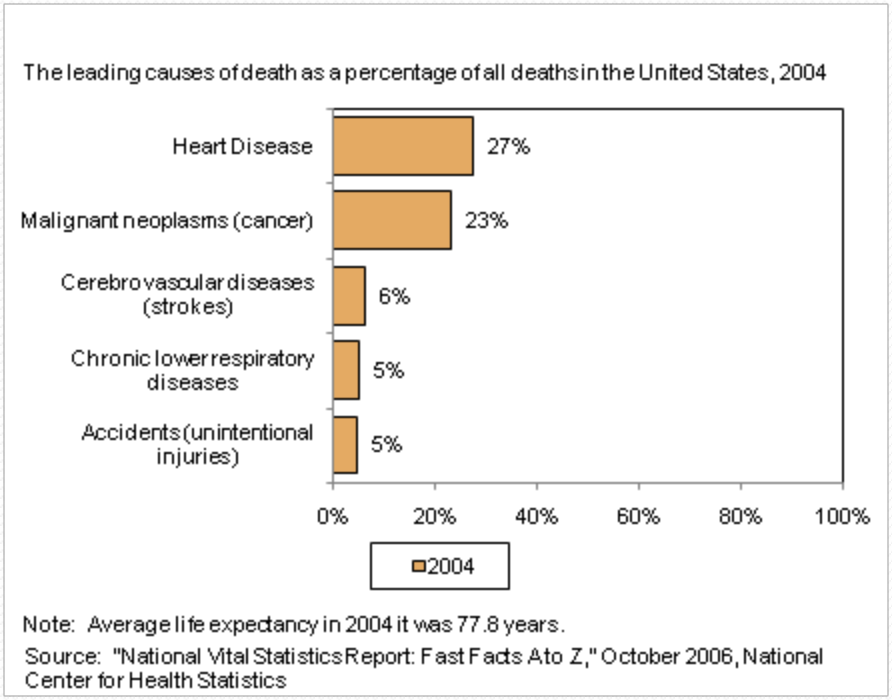
- Current direct and indirect cost of chronic disease:
  - $1.3 Trillion
- Estimated cost in 15 years:
  - $4.2 Trillion
- Current number of people with at least one chronic disease:
  - 100 million

Institute for Functional Medicine Executive Summary (2010)

Top 5 Causes of Death 1900



Top 5 Causes of Death 2005



2006 Leading Causes of Death

- **Number of deaths for leading causes of death**
- Heart disease: 631,636
- Cancer: 559,888
- Stroke (cerebrovascular diseases): 137,119
- Chronic lower respiratory diseases: 124,583
- Accidents (unintentional injuries): 121,599
- Diabetes: 72,449
- Alzheimer's disease: 72,432
- Influenza and Pneumonia: 56,326
- Nephritis, nephrotic syndrome, and nephrosis: 45,344
- Septicemia: 34,234

"Listen to your patient, he is telling you the diagnosis,"

William Osler, MD (The Father of Modern Medicine)

41% of GI Disorders are Never Diagnosed

American College of Gastroenterology

Case Study

- 19 year-old female
- Unresponsive anemia 4 years
- Osteopenia
- Depression after Menarche onset
- Treatment: Iron 325mg. FeSO4 B.I.D. & Prozac
- PMH: Anorexia age 14, eczema
- Other S/S: yellow extremities, White spots on nails, athlete's foot, dark circles under eyes, cheilosis
- Hospitalized for anorexia

Initial Assessment

- Requested Medical records
- In-Office U.A.
- Adrenal Stress Index
- MetaMetrix ION Panel
- Food Sensitivity Testing
- Heidelberg Gastric pH analysis
- MetaMetrix GI Effects

Prior Labs

WBC	3.6	Low
RBC	3.81	Low
HGB	12.0	Low
Ferritin	6	Low
EOS	7.0	High
Alk Phos	53	Low
Glucose	48	Low
Phosphorus	4.6	High
CO2	33	High

Assessment: Anemia secondary to iron and zinc deficiency, probably hypochlorhydria with metabolic alkalosis, severe hypoglycemia. Elevated Eosinophils may indicate helminthic parasite

U.A. Results

Specific Gravity	1.00	Low
pH	7.2	High
Vit. C		Low
Urine Calcium		Low
Urine Sediments		Low

Assessment: Possible malabsorption

Adrenal Stress Profile

- Morning Cortisol — Low
- Noon Cortisol — Normal
- Afternoon Cortisol — Low Normal
- Night Cortisol — Normal
- DHEA — Normal

Assessment: Cortisol dysrhythm, with low morning levels

MetaMetrix ION Test

Energy Production

- CoQ10 Low
- Lactate High
- Succinate High
- Malate High

Assessment: poor energy production from mitochondropathy secondary to multiple vitamin and mineral deficiencies

Microbial Origin Compounds

- Benzoate
- Hippurate
- P-Hydroxybenzoate
- Tricarballate
- D-Lactate
- D-Arabinitol

Assessment: Multiple positive markers for bacterial/yeast overgrowth

MetaMetrix IgG4 Food Antibody

- Casein +2
- Egg +3
- Milk +4
- Navy Bean +5
- Peanut +2
- Almond +3
- Cashew +3
- Pistachio +2

Assessment: Multiple mild to severe sensitivities the above foods

Heidelberg Results

Hidden Hypochlorhydria- complete interpretation to follow

MetaMetrix GI Effects Test Results

- General Overgrowth of Obligate Anaerobes
- Elevated Lactobacillus
- H. Pylori Infection
- Yeast Positive
- E. Nana Positive
- Strongyloides Positive

Assessment: H. pylori infection, dysbiosis secondary to bacterial and yeast overgrowth, gluten sensitivity, GI bleeding due to helminthic parasite infection with Strongyloides

- Total SCFA Low
- Anti-Gliadin Ab High
- Occult Blood Positive

Metametrix
Clinical Laboratory
3425 Corporate Way
Duluth, GA 30096
770.446.5483 Fax 770.441.2237

Accession Number:
Reference Number:
Patient
Age: 19 Sex: Female
Date of Birth:
Date Collected:
Date Received:
Report Date:
Telephone: 707-337-0769
Fax (866) 649-1867
Requested:
Comment:

Douglas Weed D.C.
837 Marina Dr.
Napa, CA 94559

2100 Gastrointestinal Function Profile

Methodology: DNA Analysis, GC/MS, Microscopic, Colorimetric, Automated Chemistry, ELISA

Pathogenic Bacteria		95% Reference Range
Helicobacter pylori	6.8E+005 H	<=1.0E+005
Campylobacter sp.	<0.01	<=1.0E+005
Clostridium difficile	<0.01	<=1.0E+005
E.H.E. coli	<0.01	<=1.0E+005

Yeast/Fungi		
Yeast/Fungi; taxonomy unavailable.	+3 => 10000 pg DNA/g specimen	Neg

A taxonomy unavailable finding may indicate ingested mold. The higher the number, the greater the indication for treatment, particularly when accompanied by clinical symptoms.

Yeast/Fungi
Yeast overgrowth has been linked to many chronic conditions, in part because of antigenic responses in some patients to even low rates of yeast growth. Potential symptoms include diarrhea, headache, bloating, atopic dermatitis and fatigue. Positives are reported as +1, +2, +3 or +4 indicating >100, >1000, >10000 or >100000 pg DNA/g

Parasites		
Endolimax nana	Positive	Neg
Strongyloides sp.	Positive	Neg
Parasite present; taxonomy unavailable.	Positive	Neg

A taxonomy unavailable finding likely indicates an ingested protozoan and not a human parasite. It does not indicate treatment unless patient symptoms and other inflammatory markers are consistent with parasite infection.

Parasites
Parasite infections are a major cause of non-viral diarrhea. Symptoms may include constipation, gas, bloating, increased allergy response, colitis, nausea and distention.

Adiposity Index		
Firmicutes	73	<= 80
Bacteroidetes	27	>= 20

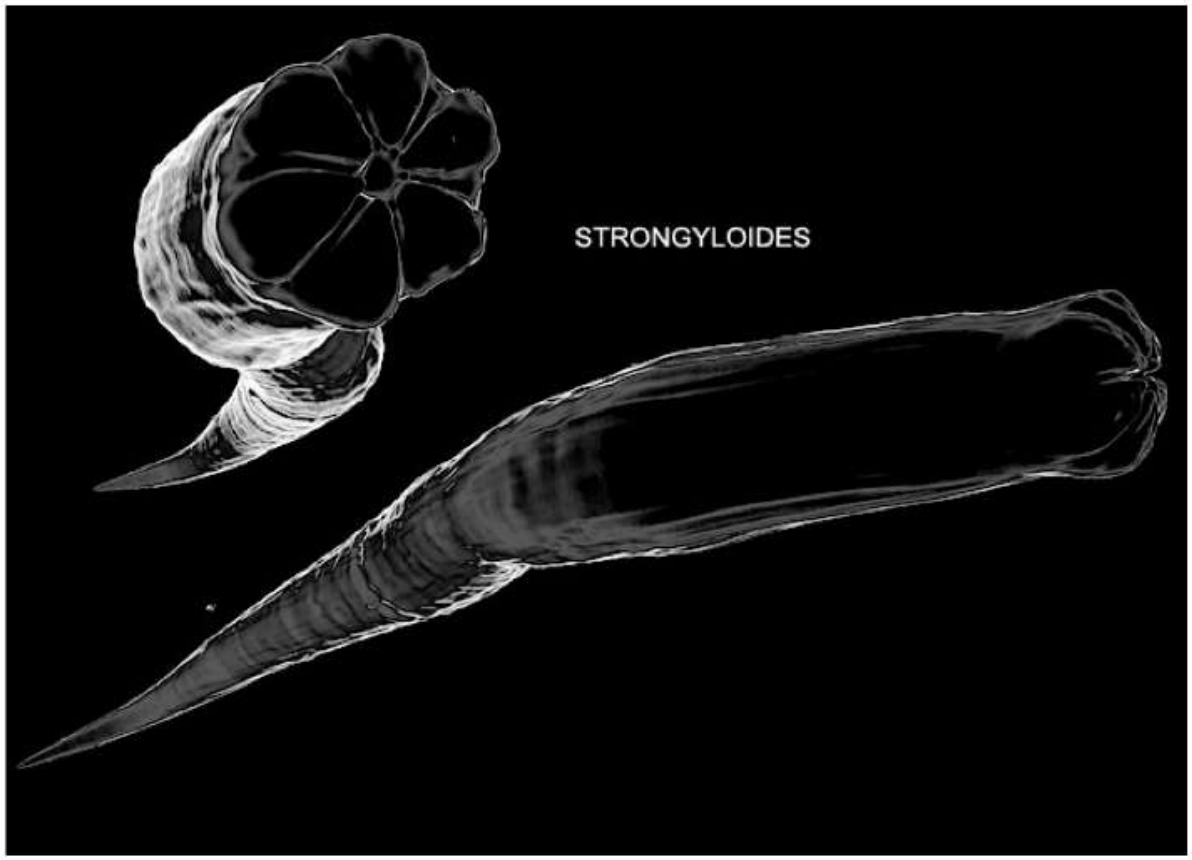
The **Adiposity Index** is derived by using DNA probes that detect multiple genera of the phyla Firmicutes and Bacteroidetes. Abnormalities of these phyla may be associated with increased caloric extraction from food.

Drug Resistance Genes			
aacA, aphD	Neg	gyrB, ParE	Neg
mecA	Pos	PBP1a, 2B	Neg
vanA, B, and C	Neg		

Case Summary

- Anemia, Osteopenia, yellow extremities, White spots on nails, athlete's foot, dark circles under eyes, cheilosis all indicate severe malabsorption.
- H. pylori infection, low vitamin c, low iron, low zinc indicate hypochlorhydria (low stomach acid)
- Heidelberg tests confirms hypochlorhydria
- Multiple food sensitivities including gluten
- Low Alk. Phos. and white fingernails confirm zinc deficiency
- Athletes foot implies systemic fungal/yeast overgrowth
- Cheilosis diagnostic for Vitamin B deficiencies
- Yellow extremities indicates liver dysfunction
- Bacterial and yeast overgrowth, and E. nana infers dysbiosis
- The root cause of all the above is an infection with the worm parasite Strongyloides. The elevated eosinophil count in the absence of environmental allergies or asthma strengthens this impression.

The Monsters Within Us



Strongyloides Stercoralis

- AKA- Threadworm
- 0.9-2.5cm.
- Lives in tunnels in small intestine
- Rare in developed countries– mainly in rural areas
- Auto-infection by parthenogenesis
- Infection documented to 65 years
- Auto-infective Larvae carry gut bacteria back into body

Treatment

- Referred for course of Albendazole to treat Strongyloides
- Betaine HCL for hypochlorhydria
- Essential nutrients to correct nutritional deficiencies
- Comprehensive GI repair program to repair intestinal epithelium
- H. pylori treated with combination of nutraceuticals

Outcome

- Within 6 months patient had total resolution of all symptoms, including chronic depression. Patient was medication-free. She gained 10 pounds.
- Anemia was resolved
- Athletes foot was resolved
- Repeat stool testing negative for H. pylori, Strongyloides, bacterial dysbiosis, and yeast overgrowth

- Root Cause: chronic GI infection with Helminthic parasite Strongyloides, leading to GI bleeding, malabsorption, and dysbiosis

I was going to have cosmetic surgery until I noticed that the doctor's office was full of portraits by Picasso.

Rita Rudner

III.
DIGESTION PHYSIOLOGY

GI Facts

- Extends from brain to rectum
- Has its own brain Enteric Nervous System
- Surface area greater than a tennis court
- Where outside world meets the inside of the body
- Lies entirely outside the body (think about it-tube through the body)
- Battle between our body and organisms that want to feed on it
- 10x more bacteria in GI tract than cells in our body
- Contains majority of immune cells in the body
- Interacts with food, living organisms and toxins
- Major system where things go wrong
- Barrier system only one cell thick

Components of Digestion

- Neurological
- Chemical
- Mechanical
- Hormonal

GI Zones

- Brain
- Mouth
- Esophagus
- Stomach
- Small Intestine
- Liver, Gall Bladder and Pancreas
- Colon

Phases of Digestion

- Cephalic
- Gastric
- Intestinal
- Basal

I.Cephalic Phase of Digestion

- Cephalic phase of digestion
  - Stomach responds to sight, smell, taste, or thought of food
  - 20% of total acid secretion occurs before food enters the stomach.
  - sensory and mental inputs converge on the hypothalamus to induce responses needed for preparing the gastrointestinal tract for food processing
  - Vagus nerve fibers from the medulla dorsal motor nucleus stimulate the parasympathetic nervous system of the stomach causing:
    - gastric secretion (via parietal and G cells activation by post-ganglionic release of acetylcholine, as well as histamine release) of stomach acid (H+)
    - gastrin release by G cells ⇨ additional H+ via histamine
    - inhibits D cells ⇨ decreased somatostatin production, thereby reducing gastrin inhibition
  - secretory activity brought on by thought or sight of food is a conditioned reflex, only occurs when food is desired. When appetite is depressed this part of the cephalic reflex is inhibited.

Mouth

- Mastication
  - Increases surface area
- Salivary Enzymes
  - Amylase
- Lubricants
- Anti-microbials
  - Lactoferrin
  - Lysozyme
  - Hypothiocyanate
  - Sig A
  - Normal pH: 7.0-7.4

Esophagus

- Food transportation: moves food from mouth to stomach
- Peristalsis: radially symmetrical contraction and relaxation of smooth muscles, propagating a wave down a tube, in an forward direction; a progression of coordinated contraction of involuntary circular muscles, preceded by simultaneous contraction of longitudinal muscle and relaxation of circular muscle in the lining of the gut
- Protected by mucus secretions
- Esophageal sphincter prevents reflux
- Major problem: Erosion

II. Gastric Phase of Digestion

Stomach

- HCL production:
  - Sterilizes microbes
  - Pepsinogen production
  - Gastrin production
  - Initiates protein digestion
  - Enhances nutrient absorption
  - Initiates intestinal peristalsis
- Stores food

Stomach

Gastric Phase of Digestion

- 50-60% of total gastric acid secretion occurs during this phase
- Swallowed food and semi-digested protein activate gastric activity
- Ingested food stimulates gastric activity by:
  - gastric contents stimulating receptors in the stomach
  - stretching the stomach ⇨ a short reflex mediated through the myenteric nerve plexus, and a long reflex mediated through the vagus nerves and brainstem

Stomach

- Stretching or distention of the stomach leads to:
  - activation of an afferent pathway which then stimulates an efferent response from the dorsal nucleus of the vagus nerve ⇨ Stimulation of acid secretion as in the cephalic phase.
  - Local Enteric Nervous System (ENS) Pathway: Activated ENS releases acetylcholine stimulating parietal cells to secrete acid

Stomach

- Chemical Pathway
  - Digested protein breaks down into peptides and amino acids, which directly stimulate the G cells to secrete even more gastrin – a positive feedback loop accelerating protein digestion.
  - Gastrin stimulates parietal cells and ECL cells to produce histamine (histamine stimulates parietal cells to produce acid).
  - Small peptides buffer stomach acid so pH does not fall excessively low.
  - Activated ENS releases acetylcholine stimulating parietal cells to secrete acid
  - Acid secretion stimulated by: acetylcholine, gastrin, and histamine, all via parasympathetics
  - Acetylcholine also stimulates secretion of protective mucus
  - Gastrin also stimulates the Chief cells to produce pepsinogen
  - Low stomach pH causes feedback inhibition of acid production by D cells producing somatostatin, which reduces gastrin secretion

Gastrin

- Peptide Hormone
- Released by:
  - Vagal Stimulation (Cephalic phase)
  - Stomach Distention
  - Protein peptides
  - Hypercalcemia (Gastric phase)
- Stimulates histamine release
- Causes production & release of HCL & Pepsinogen
- Relaxes ileocecal valve & pyloric sphincter
- Relaxes esophageal sphincter
- Increases stomach motility
- Induces pancreatic and gall bladder emptying
- Inhibited by:
  - HCL- pH below 3 (negative feedback)
  - Secretin
  - Enterogastric reflex

Wikipedia (Gastrin)

Enterogastric Reflex

- Induction of peristalsis caused by food in the stomach
- Stimulating Factors:
  - Parasympathetic stimulation
  - Stomach distention
- Inhibitory Factors:
  - Stomach pH below 1.5
  - Presence of acid in duodenum (pH 3-4)
- Shuts off Gastrin release

Wikipedia (Enterogastric Reflex)

Elevated Gastrin Levels

- Caused by:
  - Chronic gastritis
  - H. pylori infections
  - Use of antacids / acid blockers / PPI
  - Chronic kidney disease
- Causes peptic ulcer disease

http://www.nlm.nih.gov/medlineplus/ency/article/003697.htm

Pancreas (Exocrine)

The pancreas produces in addition to insulin:

- Sodium / potassium bicarbonate- neutralizes stomach acid to create an alkaline environment in the duodenum
- Carbohydrate, protein and fat digestive enzymes (amylases, lipase, and proteases)

III. Intestinal Phase

- Only 5-10% of acid secretion occurs during this phase
- Food (chyme) in duodenum initially stimulates acid secretion by:
  - peptides stimulate duodenal G cells to produce gastrin and enterooxytocin
  - duodenal amino acids stimulate acid by unknown mechanism
  - increased osmolarity in duodenum stimulates acid secretion
  -

III. Intestinal Phase

- Duodenal inhibition of gastric acid secretion
  - After initial stimulation enterogastric reflex sends efferent signals through ENS causing inhibition of vagal nuclei reducing parasympathetic signals
  - Duodenal release of secretin and cholecystokinin also reduce acid secretion, gastric motility and gastrin production
  - Decreased gastrin causes closure of pyloric sphincter, limiting food entry into the duodenum, giving time to further digest food

III.Intestinal Phase

- Secretin
- Cholecystokinin
- Protein digestive enzymes
  - Trypsin
  - Chymotrypsin
- Amylase (Carbohydrate)
- Fat Digestive Enzymes
  - Lipase
  - CHO Esterase
  - Phospholipase

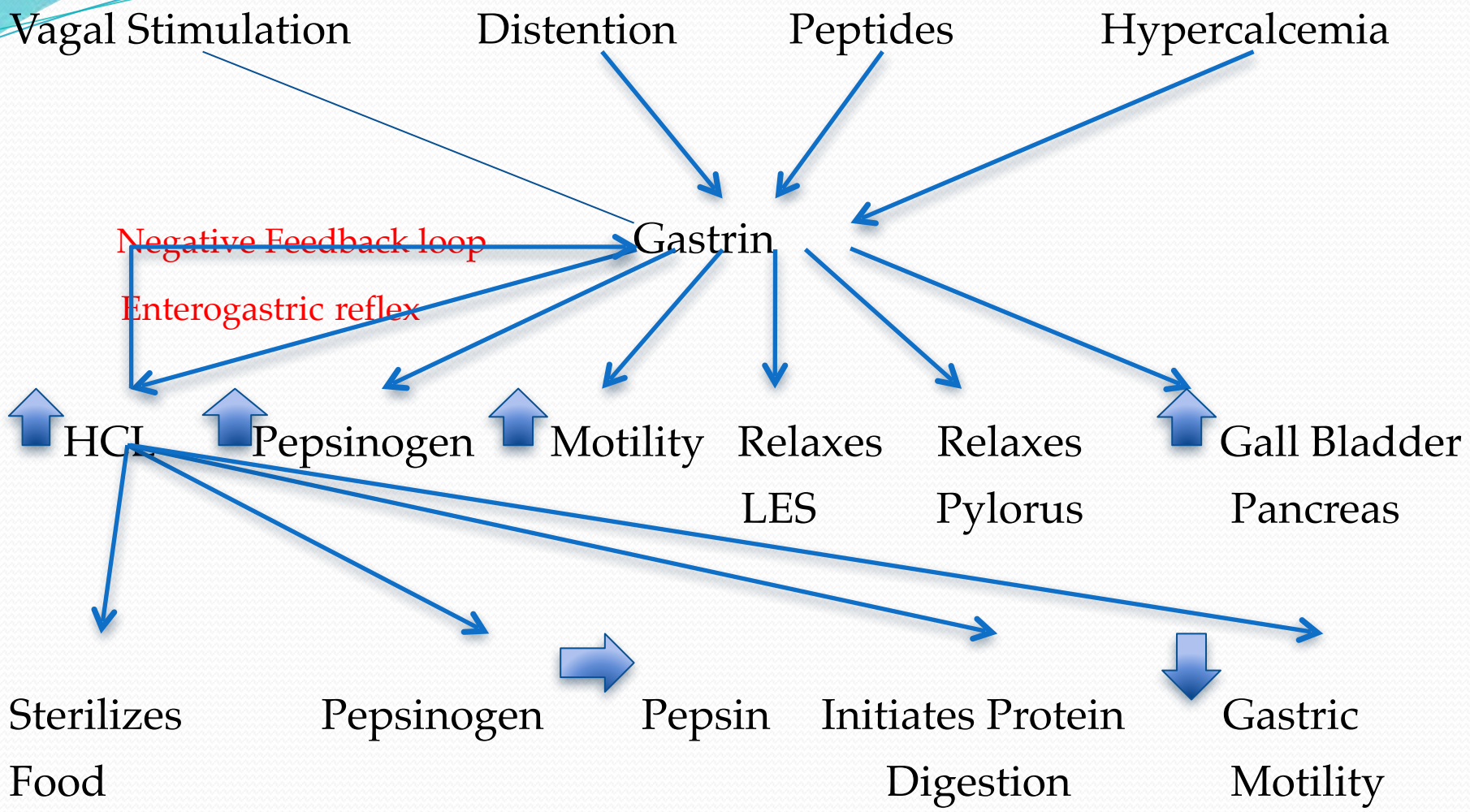
Secretin

- Stimulated by:
  - Duodenal pH 2-4.5
  - Peptides
- HCL converts inactive Prosecretin to Secretin
- Regulates duodenum pH
  - Stimulates pancreatic bicarbonate secretion
    - Neutralizes stomach acid
      - Activates pancreatic digestive enzymes
- Stimulates production of digestive enzymes
- Enhances CCK effects
- Stimulates insulin release in response to glucose
- Stimulates Pepsinogen release
- Plays roll in osmoregulation

Wikipedia (Secretin)

Cholecystokinin (CCK)

- Secreted in duodenum
- Stimulates pancreatic fat & protein digestive enzymes
- Stimulates bile release from gallbladder
- Neuropeptide that helps mediate satiety
- Inhibits gastric motility
- Inhibits HCL secretion
- Receptors throughout nervous system


Vagal Stimulation
Distention
Peptides
Hypercalcemia
Negative Feedback loop
Enterogastric reflex
Gastrin
HCL
Pepsinogen
Motility
Relaxes LES
Relaxes Pylorus
Gall Bladder Pancreas
Sterilizes Food
Pepsinogen
Pepsin
Initiates Protein Digestion
Gastric Motility

IV. Basal State

At rest, between meals and overnight there is small continuous basal secretion of gastric acid of usually less than 10 mEq/hour (1/100 of a mole of H+)

Migrating Motor Complex

Migrating motor complex (MMC) is a regular pattern of electrical activity during fasting

This activity stops upon eating a meal, and induces motor activity that acts as a "interdigestive housekeeper" in the small intestine

MMC triggers peristaltic waves, which facilitate transportation of substances from the stomach, through the small intestine, and into the colon

MMC occurs every 90–230 minutes during the interdigestive phase (i.e., between meals) and is responsible for the rumbling experienced when hungry (borborygmus)

MMC serves to transport bacteria from the small intestine to the large intestine and to inhibit the migration of colonic bacteria into the terminal ileum

Impairment to the MMC typically results in small intestinal bacterial overgrowth (SIBO)

MMC controlled by the central and enteric nervous systems, intestinal muscles, numerous peptides and hormones and does not directly depend on extrinsic nerves. Insulin, gastrin, cholecystokinin, glucagon, and secretin may disrupt the MMC.

Eating interrupts the MMC. The number of calories and nature of food determine the length of the disruption. Fats > carbohydrates > protein

Most MMC waves happen during sleep. It may be beneficial to space out food intake to allow for cleaning waves to occur between meals throughout the day

Food poisoning by a pathogen producing Cytolethal Distending Toxin B (CDTB), leading to autoimmunity; such as *C. jejuni*, may be the leading cause of MMC impairment. Narcotics also to impair MMC Stress reduces MMC activity

Patients with SIBO and IBS have on average a third as many MMC phase III events with those events being roughly 30% shorter on average

Decreased MMC > constipation

IV.

Digestion Assessment Questionnaire

This questionnaire assists the doctor in locating the specific area(s) of the digestive tract from which symptom(s) may be arising, thereby focusing attention on the root cause(s) of the patient's condition

To request a .pdf of the complete questionnaire, please email me at:

drdouglasweed@gmail.com

Key

Circle the answer that best describes you for each question.

0=No, symptom does not occur

1=Yes, minor or mild symptom, rarely occurs (monthly)

2=Moderate symptom, occurs occasionally (weekly)

3=Severe symptom, occurs frequently (daily)

Please total the points in each section.

Stomach

- Fingernails chip or break easily
- Distaste for meat
- Epigastric burning or gastric reflux
- Excessive fullness after meals
- Burp, belch, or have gas or pain within 30 minutes of eating
- Small white spots on fingernails
- History of ulcers
- History of:
  - Diabetes
  - Asthma
  - Eczema
  - Rosacea
  - Psoriasis
  - Osteoporosis
  - Hypothyroid?

Gall Bladder & Liver

- Past history of “gall bladder attacks”
- Pain between shoulder blades
- Bitter taste in mouth, especially after meals
- Greasy foods upset stomach
- Stools greasy or shiny, unusually odiferous, light or clay colored
- Stools frequently float on the surface of the toilet water
- Dry Skin
- Tiny red bumps on arms

Pancreas

- Belch or have gas after about one hour of eating
- Constipation
- Bloated after eating
- Frequent flatulence
- Undigested food in stool
- Diabetes

Small Intestine

- Specific foods cause bloating or fatigue after eating
- Known food allergies
- Constipation/diarrhea
- History of asthma, sinus infections, or a stuffy nose
- Frequent or prolonged courses of antibiotics
- Sick after drinking unsafe water
- Traveled to a Third World country
- History of food poisoning
- Ears itch

Colon

- Stools hard or difficult to pass
- Stools small, poorly formed or ribbon-like
- Constipation
- Coated tongue
- History of colitis, IBS, Celiac or Crohn's disease
- Frequent or prolonged courses of antibiotics
- History of fungus or yeast infections

V.

In-Office Labs

These labs may be done in-office, and require only a centrifuge, test tubes, reagents and a complete UA test strip. Some may be purchased on Amazon, Apex Energetics and Rocky Mountain Reagents

In-Office Lab Gateway Testing

Use first morning urine specimen for all tests

- Indican
- Sediments
- Specific Gravity
- Calcium
- Vitamin C
- Chloride (Koenisburg)

Urinary Indican Test

Measures Common Causes of Dysbiosis

- Antibiotics
- Hypochlorhydria
- Intestinal pathogens (bacterial & parasitic)
- Low SigA
- Pancreatic insufficiency
- Slow bowel transit time
- Xenobiotics
- Nutrient & fiber deficiencies
- High GI pH

Dysbiosis-Related Conditions

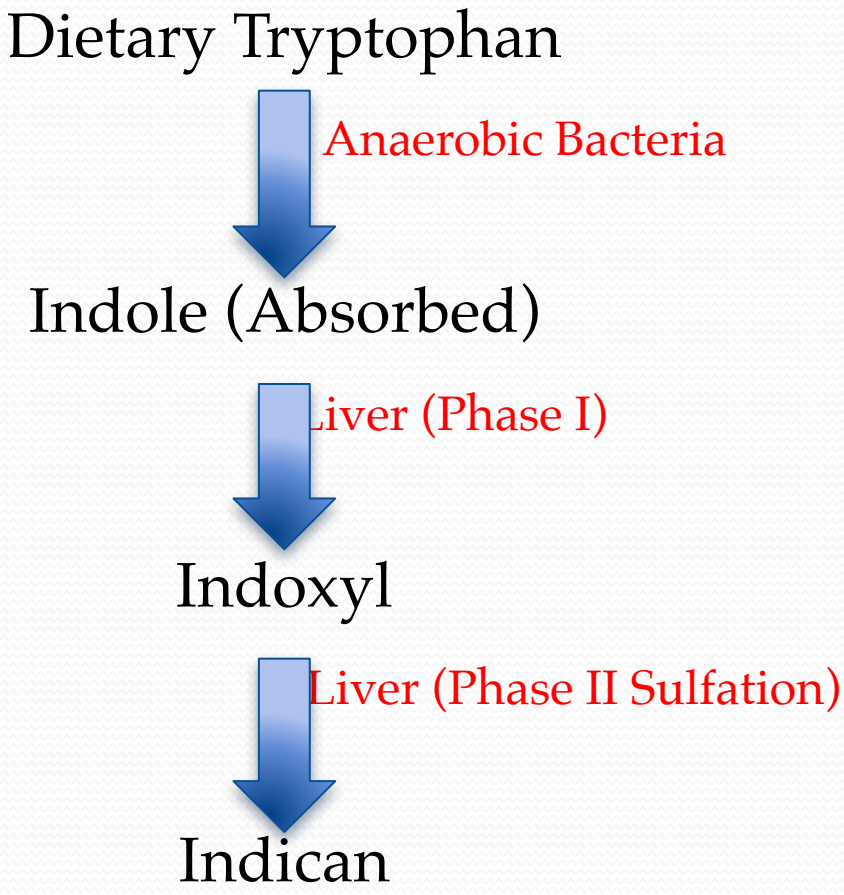
- Autoimmune disorders
- Chronic Fatigue
- Colon Cancer
- Dermatological conditions
- Food Sensitivities
- GI Permeability (Leaky gut)
- Headaches
- IBS / IBD

Dysbiosis Toxins

The following are a few of the toxins produced by dysbiosis that may cause a variety of clinical conditions:

- Ammonia- CNS
- D-Lactate- CNS
- Histamine- arrhythmia, depression, headache, low BP, nausea, skin irritation
- Indole (Indican)- bladder cancer
- Phenol- CNS, liver, kidney
- Putricene / Cadaverine- Low BP
- Skatole- RBC damage, CNS depressant

Urinary Indican (Obermeyer) Test



Interpretation of Indican Test

- Excess Dietary Tryptophan
- Pancreatic Insufficiency (Lack of Digestive Enzymes)
- Excess free oil consumption or steatorrhea
- Overgrowth of anaerobic bacteria in upper small intestine
- Hypochlorhydria

Indican Test Required Items

- Obermeyer's Reagent
- Chloroform
- Potassium Chlorate (10%)

15ml glass or plastic test tube/bottle with top

Warning: Wear Safety Goggles! Chemicals are very caustic!

Indican Test Directions

- Pipette 5ml. urine into 15ml test tube
- Pipette in 5ml. Obermeyer's Reagent
- Invert several times to mix
- Wait 5 minutes
- Pipette in 2 ml. chloroform
- Invert several times to mix
- Wait 2 minutes for chloroform to settle to bottom
- Note color in chloroform layer on bottom
- Light blue to black is positive test (Scored +1 to +4)

Interpretation of Indican Test

Quantitative Indican Directions

To positive test add potassium chlorate, 1 drop at a time. Note the number of drops required to change color back to clear.

Interpretation

- 1-3 Drops- Mild Dysbiosis
- 4-7 Drops Moderate Dysbiosis
- > 7 Drops, Severe Dysbiosis

Urine Sediment Test

- Sediment increases specific gravity
- Undigested carbohydrate, fat & protein increases sediment
- Carbohydrate >>> calcium phosphate
- Protein >>> uric acid
- Fat >>> oxalate *

* *Effects of dietary Intake on Urinary Oxalate Excretion (Eur Urol 2003;37:140-4)*

Urine Sediment Required Items

- Centrifuge
- 50% Ferric Nitrate

Warning: Ferric Nitrate will stain. Avoid contact with skin, eyes, clothes

Urine Sediment Test Directions

- Pipette 10 ml. urine into 15ml. Graduated (marked) centrifuge tube with top
- Add 4 drops ferric nitrate (do not mix)
- Centrifuge 1 minute
- Gently pour off fluid
- Tap tube on countertop to level sediment.
- Measure volume of sediment
- Normal test = o.5ml sediment

Urine Sediments Interpretation

- Low Sediment (< 0.5ml)
  - Malabsorption
  - Glucose dysregulation
  - Consumption of large quantities of water (Specific gravity will be low)
  - Look for low total protein on blood test

Urine Sediments Interpretation

- High Sediment (>0.5ml.)
- Maldigestion
  - Deficient pancreatic enzymes
  - Excess refined carbohydrates/sugar
  - Excess fat
  - Excess protein
  - Poor carbohydrate, fat or protein metabolism

Urine Specific Gravity

- Measure of total solutes in urine
  - Electrolytes
  - Urea
  - Uric acid
  - Creatinine
  - Metabolites from incomplete digestion
  - Organic Acids
- Measure of kidney function
- Highest in morning
- Affected by quantity of water consumed

Urine Sp. Gravity Required Items

Hydrometer

&

Urometer

or

Urine Dipstick

Urine Sp. Gravity Directions

- Fill Urometer with first morning urine
- Place hydrometer in Urometer
- Read results

Or (easier method)

- Place dipstick in urine
- Read results

Urine Sp. Gravity Interpretation

Normal range: 1.005-1.03

Functional Range: 1.01-1.02

High (Concentrated Urine)

- Elevated Solutes-Check dipstick for protein, glucose, ketones
- Adrenal Insufficiency- ↓ aldosterone, check Koenisberg test
- Increased Minerals- Check urinary Calcium
- Diabetes- Check glucose & ketones
- Maldigestion- ↑ Urine Sediments
- Dehydration
- Protein Malnutrition

Urine Sp. Gravity Interpretation

Normal range: 1.005-1.03

Functional Range: 1.01-1.02

Low (Dilute Urine)

- Kidney Dysfunction
- High Fluid Intake
- Malabsorption

Urine Calcium (Sulkowitch) Test

- Urinary calcium reflects levels of blood calcium
- Test measures excreted calcium
- Kidneys have threshold level, below which little excreted
- Threshold level 7.5-9.0 mg/dl
- Optimal Serum calcium 9.2-10.0 mg/dl
- Below 7.5 mg/dl- no excretion
- Above 10.0 increased excretion
- Affected by stomach acidity, diet/supplement intake, magnesium and phosphorus

Urine Calcium Required Items

- Test tube
- 1 ml. pipette
- Sulkowitch Reagent

Urine Calcium Test Directions

- Place 1 ml. of urine in test tube
- Add 1 ml. of Sulkowitch Reagent
- Mix
- Wait 1 minute and observe turbidity

Urine Calcium Test Interpretation

Place white page with black type behind test tube

- Clear- little / no precipitate- type easily read
- Light- slight precipitate, type can be read
- Moderate- more turbidity- type seen but not read
- Heavy- can not see type
- Severe/Milky- Looks like skim milk

Urine Calcium Test Interpretation

High Urine Calcium- Heavy / Milky

- Excess Calcium intake
- Increased dietary phosphorus / calcium ratio
- Metabolic acidosis
- Bone resorption
- Hypothyroidism
- Hyperparathyroidism
- Excess Cortisol

Urine Calcium Test Interpretation

Low Urine Calcium- Clear

- Low dietary calcium intake
- Hypochlorhydria
- Malabsorption
- Hypoparathyroidism
- Vit. D deficiency

Urine Vitamin C Test

- Excretion Threshold- 1.2mg/dl
- Optimal Levels > Urinary excretion of Vit C
- Low Levels > No detectable urinary Vit. C
- Indirect measure of anti-oxidant capacity

Urine Vitamin C Test Required Items

- Test Tube
- Reagent

Urine Vit. C Test Directions

- Place 1 drop of reagent in test tube
- Add urine 1 drop at a time
- Count drops until color changes from blue to clear

Urine Vitamin C Interpretation

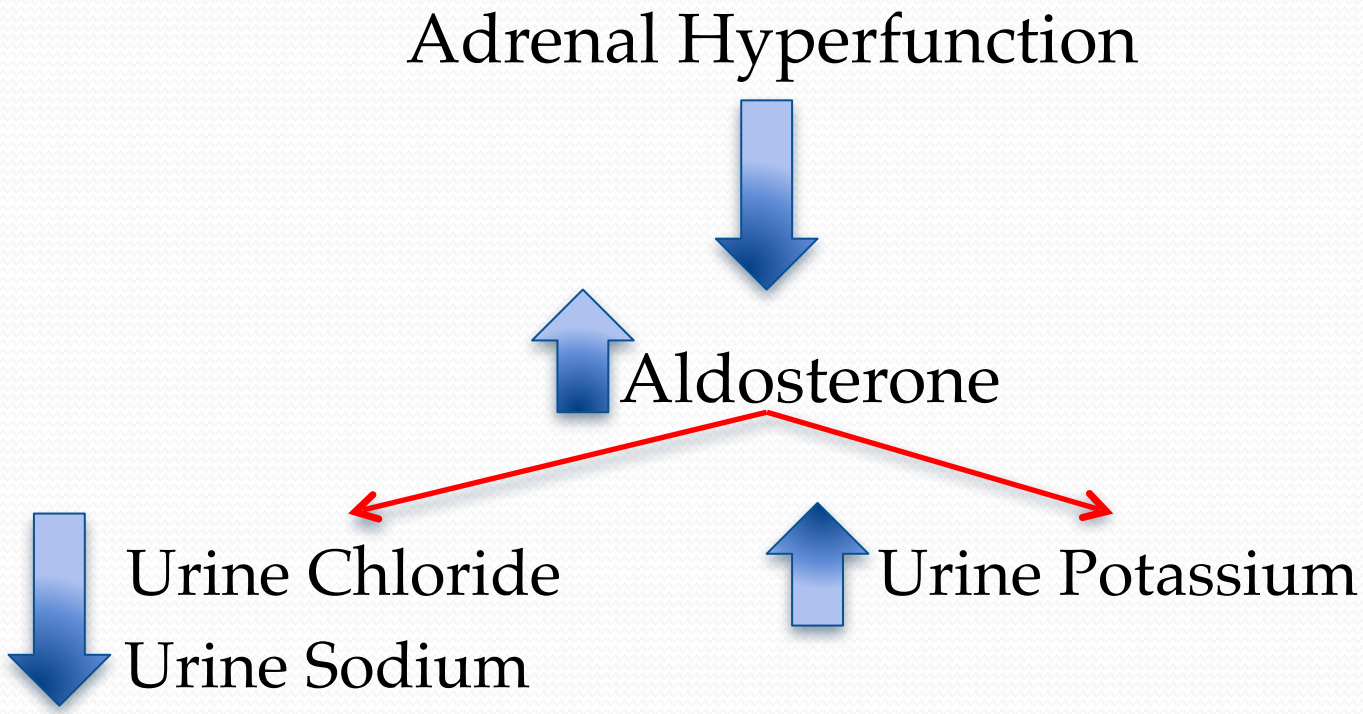
- Adequate Vitamin C – 5 drops or less to neutralize color
- Inadequate Vitamin C > 5 drops to neutralize color

Urine Adrenal Function Test

Koenisburg Test

- Adrenal Cortex Hormones
  - Cortisol
  - DHEA
  - Aldosterone
- Measures Urinary Chloride
- Indirect Measure of Adrenal Stress
- Low Cortisol > Decreased Sig A

Urine Adrenal Function Test



Koenisburg Test Required Items

- 10 ml test tube
- 1 ml pipette
- 20% Potassium Chromate
- 2.9% Silver Nitrate

Koenisburg Test Directions

- Pipette 10 drops of urine into test tube
- Add 1 drop of Potassium Chromate
- Add 1 drop at a time of silver nitrate and mix
- Record number of drops required to turn color permanently from yellow to brick red

Koenisburg Test Interpretation

Low Urine Chloride (<14 drops)

- Adrenal Hyperfunction
  - Increased Aldosterone
  - Increased Cortisol
  - Increased potassium loss

Koenisburg Test Interpretation

High Urine Chloride (>25 drops)

- Adrenal Hypofunction
- Kidney Stress
- Alkaline Mineral (Mg/K) Deficiency
- Hypochlorhydria- due to lack of CL^- to produce HCL
- Increased Inflammation > oxidative stress

Chemstrip 10 testing

- Specific gravity: discussed above
- pH: diet and various medical conditions
- Bacterial Infections
  - Leukocytes
  - Nitrate
  - Blood
- Protein: kidney dysfunction
- Glucose: diabetes
- Ketones: ketogenic diet, keto acidosis, diabetes
- Urobilinogen: ⇧ liver disease, hemolytic anemia, ⇩ bile duct blockage
- Bilirubin: normally not present. ⇧ indicates liver disease

Case History In-Office Labs

Urinalysis

- Specific Gravity 1.00 Low
- pH 7.2 High
- Vit. C Low
- Chloride Low
- Urine Calcium Low
- Urine Sediments Low

Adrenal Stress Profile

- Morning Cortisol Low
- Noon Cortisol Normal
- Afternoon Cortisol Low Normal
- Night Cortisol Normal
- DHEA Normal

Case History In-Office Labs Interpretation

- Low Sp. Gravity: high fluid intake, r/o kidney dysfunction and malabsorption
- High pH: possible metabolic alkalosis or hypochlorhydria
- Low Vit C: Sub-optimal Vit C levels or overt deficiency
- Low Koenisburg: Increased cortisol, aldosterone, retained Na+, low K+
- Low Calcium: Low calcium intake, hypochlorhydria, malabsorption, Vit. D deficiency, hypoparathyroidism

Low Urine sediments: Malabsorption, blood sugar dysregulation, high fluid intake, low total protein

Urine tests are screening tests only and must be confirmed by additional testing. In the above tests, the low Koenisburg, was not confirmed by the salivary Adrenal Stress Index, which showed low cortisol levels

VI.

Routine Labs

&

Gastrointestinal Function

General Labs

- CBC
- WBC
- Eosinophils
- Total Protein / Globulins
- BUN
- Thyroid
- Phosphorus
- Cortisol & SigA
- C-RP & ESR
- ANA
- Amylase / Lipase

Functional vs. Pathological Ranges

- Pathological range purpose is to detect disease
  - Lab ranges established by each laboratory
  - Normal range considered to be 2 S.D. from the mean
  - By definition 95% of all people are "Normal"
  - Only 2.5% of values above & below range are abnormal
  - Illogical as more than 2.5 % of population has many of the diseases the labs are intended to diagnose.
- Functional range purpose is to determine health
  - Uses tighter ranges based upon healthy individuals
  - Guidelines from Am Assoc Clin Chemists
  - Generally uses the 80th percentile range

Complete Blood Count (CBC) & Iron Study

- ⇩ RBC, HB & HCT = ⇩ RBC production
- ⇩ MCV, MCHC & MCH = microcytic anemia
- ⇧ TIBC & Transferrin; ⇩ iron & ferritin = iron deficiency
- ⇩ TIBC & Transferrin; ⇧ iron & ferritin = iron overload, hemochromatosis, hemosiderosis, and oxidative damage

WBC Count

- Low WBC
  - Chronic infection
  - Immune compromise
- ⬆ PMN / Lymphocyte = bacterial infection
- ⬇ PMN/ Lymphocyte = viral infection
- ⬆ Monocyte = recent immune challenge, or chronic inflammation
- ⬆ Basophils = inflammation (release histamines)

Eosinophils

- Involved in later stages of inflammation
- Are phagocytic
- Contain histamine granules
- Increased in
  - Allergy & Asthma
  - Parasitic Infections (Helminthic)
- Normal Lab values: 0-5%
- Functional lab values 0-3%

Total Protein

- Albumin
  - Carrier Protein
  - Maintains osmotic pressure
- Globulins
  - Alpha
  - Beta
  - Gamma: ⇧ in infection

Blood Urea Nitrogen

- Formed from protein catabolism by liver
- BUN is nitrogen portion of urea
- Increased
  - Fever
  - Diabetes
  - ⇧ Dietary protein intake
  - Liver/Kidney dysfunction, Adrenal stress
- Decreased
  - Low protein intake
  - Protein Malabsorption
  - Hypochlorhydria
  - Poor liver function

Phosphorus

- Normal Range 2.8-4.5
- 85% in hydroxyapatite crystals in bone
- 15% intracellular
- Increased in metabolic acidosis
- Involved with energy storage & transfer (ATP)
- Phospholipids
- Increased with:
  - ⇧ Bone turnover
  - ⇩ Calcium
- Decreased:
  - Hypochlorhydria- levels below 3.0 indicator for low stomach acid

Sig A

- Sig A (Secretory Immunoglobulin A)
  - Predominant Antibody- produced in GI tract
  - Produced at mucosal surfaces
  - Non-Specific Immunity (bacteria, viruses, toxins, amoebic parasites, large food particles)
  - Protects mucosal barrier
  - Does not elicit a systemic (immune) response
  - Agglutinates, binds & opsonifies particles preventing adherence
  - Prevents intestinal inflammation
  - Downregulates IL-1
  - Inhibits IgE mediated hypersensitivity
  - Inhibits penetration of intestinal epithelium
  - Promotes immune tolerance

Sig A

- Acute stress upregulates
- Chronic stress downregulates
- Prolonged elevation of cortisol reduces levels
- Relaxation increases
- Decreases with age
- Promotes eosinophil degranulation

Figure 6. **Effect of Emotion on SIgA Release**

Autonomic System Imbalance Caused by Emotional Stress Inhibits SIgA Release

HeartMath Institute

C-Reactive Protein (C-RP)

- Blood protein increases with inflammation
- Acute phase response- increases rapidly
- Short half-life (18 hours)
- Acute infections/trauma levels 200 or higher
- Chronic inflammation levels 3-20
- Ongoing levels above 3 indicate chronic inflammation
- Produced in response to IL-6 from macrophages and adipocytes
- Binds to Phosphatidyl Choline on dead or dying cell surface
- Activates Complement system
- Independent risk factor for cardiovascular disease above 3
- Make sure to use only high sensitivity C-RP, called C-RP(hs)
- May be best single lab test as measures underlying cause of chronic conditions (inflammation)

Anti-Nuclear Antibody (ANA)

- Antibodies directed against cell nucleus
- Present in most autoimmune diseases
- Positive result indicates need for additional testing
- Presence may predate onset of autoimmunity by up to 5 years

Arthritis Research & Therapy 2011, 13:R30doi:10.1186/ar3258

Arthritis Research & Therapy 2011, 13:109 doi:10.1186/ar3282

Serum Amylase / Lipase

- Amylase
  - Catalyses conversion of starches into sugars
  - Produced in saliva and by pancreas
  - ⇧ in pancreatitis
  - ⇩ in pancreatic insufficiency
- Lipase
  - Catalyses hydrolysis of fats
  - Produced by pancreas
  - ⇧ in pancreatitis
  - ⇩ in pancreatic insufficiency

Hypochlorhydria Pattern

- Increased Globulin > 2.8 g/L
- BUN > 16 with Albumin < 4 g/L
- Low BUN , 6 or less
- Total protein < 6.9 g/L
- Phosphorus < 3.0
- Gastrin < 50 or >100
- MCV > 95 & MCH < 31.9
- Calcium < 9.0 or > 10.4
- Low Fe, Cl, Mg, Zn, Cu
- Alk Phos < 70 (marker for zinc deficiency)

VII.
Importance of Stomach Acid

Why Test For Stomach Acid?

Functional Stomach Disorders

- GERD
- Hypochlorhydria
- Hyperchlorhydria (rare)
- Gastritis
- H. pylori infections
- Ulcers

Gastro-Esophageal Reflux Disease (GERD)

- Increased frequency with age
- 20% of adult Americans have heartburn at least twice/week
- 5-8 % of adolescents have heartburn
- Everyone has gastro-esophageal reflux

American Society for Gastrointestinal Endoscopy

Am. Acad. Of Otolaryngology

GERD

- "Reflux-esophagitis was found in patients with hyperacidity as well as in patients with normal and hypoacidity."
- The conclusion is made that the gastric acidity is not a decisive factor in the development of the dyspeptic syndrome in peptic ulcer.

Vutr Boles 1988;27 (6): 14-20

Proton Pump Inhibitors (PPI)

- Use of GA (Gastric Acid) inhibitors was associated with an increased risk of acute gastroenteritis and … pneumonia in otherwise healthy GERD-affected children.
- GA suppression is a major risk factor for infections.
- Sustained even after the end of therapy.
- Direct inhibitory effect of GA inhibitors on leukocyte functions and qualitative and quantitative gastrointestinal microflora modification.

Pediatrics May 2006; 117 (5) 817-20

Intestinal Infections and PPI

- Patients with achlorhydria and resected stomachs have excessive growth of bacteria in the digestive tract
- Much higher incidence of gastrointestinal infections.
- Treatment of reflux esophagitis with PPI creates a similar low acid state.
- Suppression of gastric acid secretion causes a dose dependent increased risk of intestinal infections especially for people over 65.

Ned Tijdschr Geneeskd. 1998 Dec 11;143 (50):2511-4

PPI Relaxes Lower Esophageal Sphincter (LES)

"PPI relaxed the LES in a concentration-dependent manner and suppressed… contractions. Furthermore, PPI attenuated spontaneous contractile activity of the tissue." (i.e. decreased MMC)

J Pharm Pharmacol. 2011 Oct;63(10):1295-300.

Effect of Gastrin on LES Function

- Infusion of gastrin resulted in plasma levels comparable to those reached after a meal.
- Lower esophageal sphincter pressure decreased significantly
- Gastroesophageal reflux and the number of transient LES relaxations associated with reflux were significantly increased.

Dig Dis Sci. 1997 Dec;42 (12) 2547-51

PPI and Gastrin Secretion

- Therapeutic gastric acid suppression was present in (86%) patients with an elevated fasting serum gastrin with patients on PPI.
- Significant inverse correlation between fasting serum gastrin and gastric acid in patients with GERD on PPI
- An elevated fasting serum gastrin concentration while on PPI therapy suggests that gastric acid secretion is adequately suppressed.

Dig Dis Sci. 2000 Jan;45 (1) 34-9

PPI and Cellular Proliferation

- Patients with Barrett's esophagus (BE) are commonly treated with PPIs.
- Use of PPI can lead to significant elevation of gastrin.
- Known tumorigenic and proliferative effects of gastrin.
- In nondysplastic BE patients on PPI therapy, serum gastrin levels were significantly correlated with cellular proliferation.
- Potential causal effect of gastrin on neoplastic progression in BE.

Therap Adv Gastroenterol. 2011 March; 4(2): 89-94

HCL and LES Pressure

- Autonomic nervous system activates LES in response to increased stomach acidity.
- LES neuro-receptors “measure” acidity
- Increased HCL increases LES pressure (contraction or closure)

Dysbiosis and GERD

"In the human distal esophagus, inflammation and intestinal metaplasia are associated with global alteration of the microbiome. These findings raise the issue of a possible role for dysbiosis in the pathogenesis of reflux-related disorders."

Gastroenterology. 2009 Aug; 137 (2): 419-21

Dysbiosis*

*A State of Disordered Microbial Ecology that Causes Disease (Overgrowth of Potentially Harmful Bacteria when present in excess quantities)

- Reduces friendly bacteria
  - Vitamin Deficiencies
  - SCFA deficiency
- Inflammation/Irritation
- GI Permeability
- Toxins
- Hydrogenates polyunsaturated fatty acids (PUFA's)
- B-glucuronidase de-conjugates hormones allowing resorption

The Causes of Intestinal Dysbiosis, A Review. Alter. Med Rev 2004; 9(2) 180-197

Dysbiosis and GERD

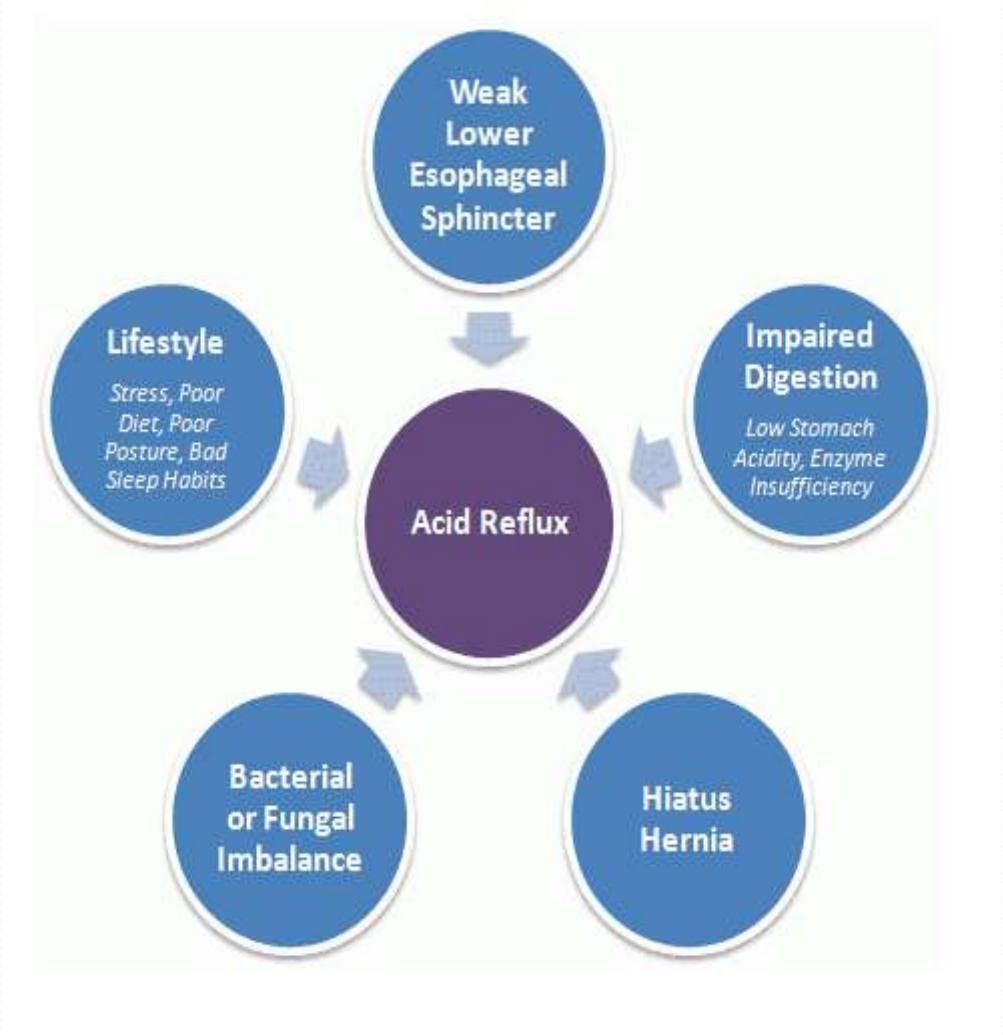
"Lipopolysaccharides (LPS), a major structure of the outer membrane in Gram-negative bacteria, can up-regulate gene expression of pro-inflammatory cytokines via activation of the TLR4 and NF-kB pathway. The potential impact of LPS on reflux esophagitis may be through relaxation of the lower esophageal sphincter via iNOS and by delaying gastric emptying via COX-2. Chronic inflammation may be play a critical role in the progression from benign to malignant esophageal disease.

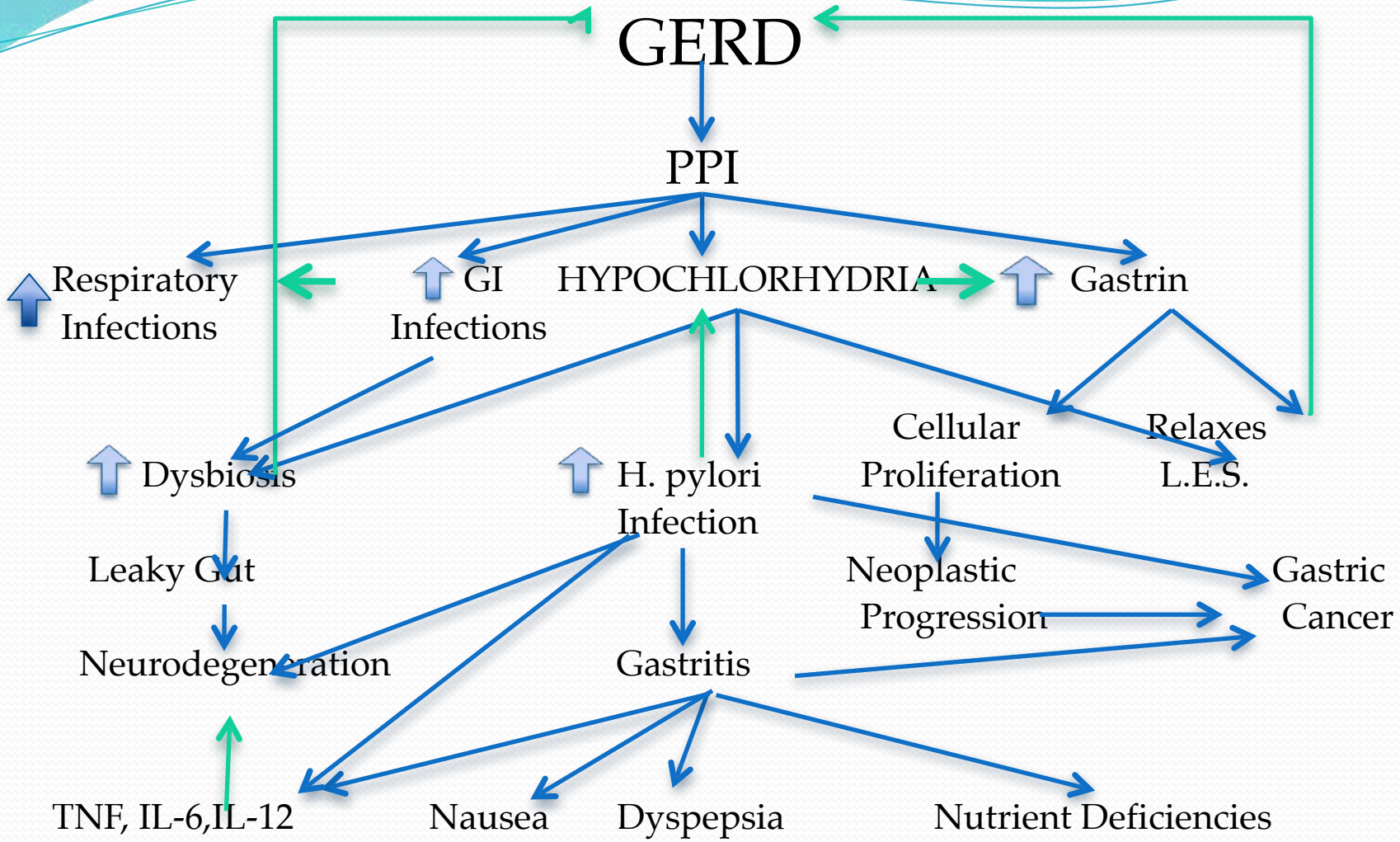
Clinical Cancer Research (Online Pub. 2/16/12)

Lipoteichoic Acid and Inflammation

- Glycolipid from Gram-positive bacteria
- Shares many inflammatory properties with bacterial Lipopolysaccharides (LPS)
- Abundant in Lactobacillus acidophilus
- Increases IL-12 and TNF
- Activates pro-inflammatory subsets of Dendritic cells
- Activates pathogenic CD4+ T-cells

Journal of Inflammation 2012, **9:7**


Weak Lower Esophageal Sphincter
Lifestyle
Stress, Poor Diet, Poor Posture, Bad Sleep Habits
Impaired Digestion
Low Stomach Acidity, Enzyme Insufficiency
Acid Reflux
Bacterial or Fungal Imbalance
Hiatus Hernia


GERD
PPI
Respiratory Infections
GI Infections
HYPOCHLORHYDRIA
Gastrin
Dysbiosis
H. pylori Infection
Cellular Proliferation
Relaxes L.E.S.
Leaky Gut
Neoplastic Progression
Gastric Cancer
Neurodegeneration
Gastritis
TNF, IL-6,IL-12
Nausea
Dyspepsia
Nutrient Deficiencies

Melatonin and GERD

- Melatonin protects the GI mucosa from oxidative damage
- Study involved 36 patients
- Oral melatonin improved GERD
- Reduced epigastric pain and heartburn

BMC Gastroenterol. 2010; 10: 7

Melatonin and H.pylori Ulcers

- Melatonin 5 mg. B.I.D. or tryptophan 250 mg. B.I.D. with PPI
- Initial low levels of melatonin
- Increased with treatment
- Increased rate of ulcer healing over PPI alone

J. Physiol Pharmacol 2011 Oct; 62 (5): 521-6

Hypochlorhydria

- HCL production declines with age
- More prevalent over 45

Blackman et. al. 1970 Am. J. Dig. Dis. 15 (9): 783

Hypochlorhydria

(AKA low stomach acid)

- 30-50% of population over 65 is hypochlorhydric
- Underlying infections/dysbiosis often root cause
- May be autoimmune in nature (Atrophic gastritis)

Townsend Newsletter (July 2004)

Hypochlorhydria & Gastric Flora

- 15 healthy people 80-91 years old
- 12 were hypochlorhydric
- Average resting stomach pH 6.6
- 10^8 CFU/ml in hypochlorhydric patients*
- $<10^1$ CFU/ml in normalchlorhydric patients*

Gut 1992; 33: 1331-7

* note: CFU (colony forming units. 1 CFU = 1 bacteria). The normal stomach contains very few bacteria. patients in this study had 10,000,000 more bacteria in their stomach/small intestine, indicative of Small Intestinal bacterial overgrowth (SIBO)

"The hypochlorhydric stomach serves as reservoir of microbes from saliva and ingested materials that continuously seeds the small intestine. A close relationship has been shown between intestinal stasis and bacterial overgrowth, and impaired fasting motility of the small intestine has been reported in patients with bacterial overgrowth syndrome"

Gut 1992; 33: 1331-7

Atrophic Gastritis

- Typically non-erosive
- Loss of HCL secretion
- High prevalence-marked increase with age
  - 15% of adults over 25
  - >30% of adults over 60

Townsend Newsletter (July 2004)

Atrophic Gastritis Signs / Symptoms

- Dyspepsia
- Abdominal pain
- Distention / Bloating / Burping
- Nausea / Vomiting
- Hypochlorhydria
- Increased TNF, IL-1, IL-6
- Increased enteric infections
- Intestinal dysbiosis
- Nutrient Malabsorption
- Chipped fingernails
- Anemia
- Thinning hair

Atrophic Gastritis Causes

- Enteric infections
  - H. pylori
- NSAID overuse
- Autoimmune
  - Gluten
  - Food Sensitivity
  - GI Permeability

Helicobacter pylori

- Infects stomach
- First identified in 1982 by Barry Marshall, MD
- Primary cause of gastric and duodenal ulcers
- May cause stomach cancer
- 80% of infected are asymptomatic
- Adequate stomach acid helps prevent infection
- Lives in parietal cells under mucus

Wikipedia (Helicobacter pylori)

H. pylori Effect on HCL Production

"Helicobacter pylori plays major causative roles in peptic ulcer disease and gastric cancer. Elevated acid secretion in patients with duodenal ulcers contributes to duodenal injury, and diminished acid secretion in patients with gastric cancer allows carcinogen-producing bacteria to colonize the stomach. Eradication of H. pylori normalizes acid secretion both in hyper-secreting and hypo-secreting relatives of gastric cancer patients."

Yale J Biol Med. 1999 Mar-Jun; 72(2-3): 195–202

H. pylori and Gastric Acidity

"Corpus-predominant gastritis and corpus atrophy are accompanied by Hypochlorhydria and carry the highest risk for gastric cancer, whereas antrum-predominant gastritis with little involvement of the corpus-fundic mucosa is associated with hyperchlorhydria and predisposes to duodenal ulcer disease."

Dig Dis 2011;29:459-464

H. pylori and Gastric Atrophy

"The progression of the gastric pre-cancerous lesions, glandular atrophy and intestinal metaplasia in superficial gastritis, gastric erosion, erosive gastritis and gastric ulcer was strongly related to H pylori infection."

World J Gastroenterol, 2005 Feb14; 11 (6):791-6

H. pylori and Dyspepsia

"Helicobacter pylori eradication provided significant benefits to primary care patients with functional dyspepsia"

Arch Intern Med. 2011;171(21):1929-1936. doi:10.1001/archinternmed. 2011.533

H. pylori and Parkinson's

"The two-stage neuroinflammatory process, containment and progression, proposed to underlie neurodegeneration may predicate on systemic inflammation arising from the gastrointestinal tract. Helicobacter infection has been described as one switch in the pathogenic-circuitry of idiopathic Parkinsonism (IP): eradication modifies disease progression and marked deterioration accompanies eradication-failure."

Gut Pathogens 2009, 1:20doi:10.1186/1757-4749-1-20

H. pylori Antibiotic Resistance

"Recent studies suggest that eradication rates achieved by first-line treatment with a proton pump inhibitor (PPI), clarithromycin, and amoxicillin have decreased to 70–85%, in part due to increasing clarithromycin resistance."

American College of Gastroenterology Guideline on the Management of Helicobacter pylori Infection (2007)

H. pylori and PPI's

H. pylori-positive patients on long-term acid inhibition displayed three features: non-H. pylori bacterial growth; increased cytokine levels; and a higher risk of atrophic gastritis. We suggest that double infection with H. pylori and non-H. pylori bacteria is a major factor in the development of atrophic gastritis during gastric acid inhibition.

Aliment Pharmacol Ther. 2001 Aug;15(8): 1163-75

Vit. C Effect on H. pylori

"In a prospective, controlled study involving 214 consecutive patients infected with Helicobacter pylori, supplementation with vitamin C (250 mg, twice a day) was found to reduce the dosage of clarithromycin needed (from 500 mg, twice a day, to 250 mg, twice a day) to successfully eradicate H. pylori."

Acta Gastroenterol Belg, 2009 April-June; 72(2): 222-4.

H. pylori and Botanical Oils

- "… three different strains of H. pylori were highly sensitive to some mixtures of essential oils, particularly oregano. The most effective was a mixture of oregano, clove, wormwood and ginger oils."
- "Feedback from practitioners throughout the USA and UK indicates that eradication rates with Pyloricin is >70% and this is now being confirmed in independent human clinical trials using the radioactive labeled urea/Urease breath test, which is rapidly becoming the gold standard for diagnosis."

Townsend Newsletter (July 2004)

Natural Antimicrobials

"… the antimicrobials allicin from garlic, cinnamaldehydes from cinnamon, and also berberine from goldenseal have been found to profoundly inhibit overgrowth organisms such as Staphylococci, Coliforms, and yeasts without affecting Lactobacilli and Bifidobacteria."

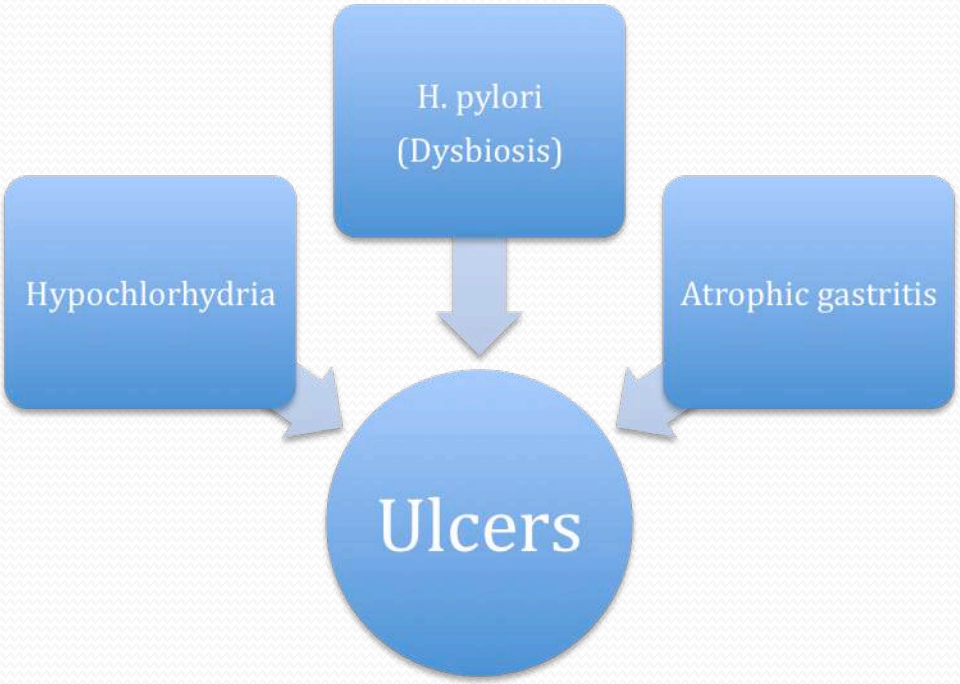
(Rees et al., 1993).

Mastica Gum

"Our results show that administration of mastic extract may be effective in reducing *H. pylori* colonization and that the major triterpenic acids in the acid extract may be responsible for such an activity."

Antimicrob Agents Chemother. 2007 Feb;51(2):551-9.

Ulcers


H. pylori
(Dysbiosis)
Hypochlorhydria
Atrophic gastritis
Ulcers

Summary

- Stomach acid production decreases with age
- GERD increases with age
- PPI do not treat the root causes of GERD but rather suppress symptoms of heartburn
- PPI increases rate of GI infection and dysbiosis
- PPI increases gastrin levels
- Increased gastrin decreases LES pressure
- Dysbiosis decreases LES pressure
- HCL increases LES pressure

Summary

- Bacterial products LPS and Lipoteichoic acid cause inflammatory changes though NF-KB, TNF, IL-6 and IL-12
- Hypochlorhydria increases dysbiosis
- H. pylori infection is a major cause of atrophic gastritis
- Atrophic gastritis is a major cause of Hypochlorhydria
- Hypochlorhydria increases H. pylori risk
- H. pylori is the major cause of GI ulcers

Heidelberg Gastric pH Analysis

Normal Gastrogram

A HEIDELBERG NORMAL pH PROFILE WITH ALKALI CHALLENGE SHOWING pH MEASUREMENTS AND TIMING

1) Capsule Administration
1-2) Normal resting stomach pH
2) Alkali challenge with 5cc. Of sodium bicarbonate
2-3) Normal reacidification time
3) Normal rapid reacidification

Time to reacidify will determine if the patient is normochlorhydric, hypochlorhydric, or hyperchlorhydric. As age increases, time to reacidify increases. The average normal reacidification time for all age groups, falls between 15 to 26 Minutes.

Normal Gastrogram

A HEIDELBERG NORMAL pH PROFILE WITH ALKALI CHALLENGE SHOWING pH MEASUREMENTS AND TIMING

Heidelberg University clinical study on 1000 patients, found that the normal re-acidification time, for young healthy adults is, between 20 to 22 minutes.

Normal HCL Secretion

- Resting stomach secretion = 10% of maximal rate
- Resting pH range 1.8-2.8 (Heidelberg 0.5-1.5)
- Feedback loop prevents over-acidification
- Normal full stomach pH 3.5-4.5 (Heidelberg 4.5-5.5)
- Following meal gradually decreases to resting pH

Townsend Newsletter July 2004

Achlorhydria

HEIDELBERG ACHLORHYDRIA pH PROFILE (Obvious)

Achlorhydria: Complete absence of free hydrochloric acid.

- May have Pernicious Anemia, due to Vitamin B-12deficiency
- Megaloblastic anemia
- Multiple essential nutrient deficiencies
- Atrophic Gastritis

Hypochlorhydria

HEIDELBERG HYPOCHLORHYDRIC pH PROFILE (Obvious)

Obvious hypochlorhydria: The diminished secretion of HCL.

- Reflex stomach emptying (Dumping Syndrome)
- Loss of most of first phase of digestion, including:
  - Sterilization
  - Incomplete protein digestion
  - Failure to activate Pepsinogen
  - Increased possibilty of food antigenicity

Hidden Hypochlorhydria

Hidden hypochlorhydria: Normal resting acidity but failure to produce adequate acid in response to alkali challenge.

- Normal fasting pH 0.5–1.3
- When challenged, will *neutralize* to pH 6.0– 8.0
- Will not reacidify prior to 26 minutes
- In many instances reacidification takes much longer
- Inadequate HCL production
- Most common HCL production dysfunction
- 68% of patients with type II diabetes suffer from hypochlorhydria or achlorhydria

Hyperchlorhydria

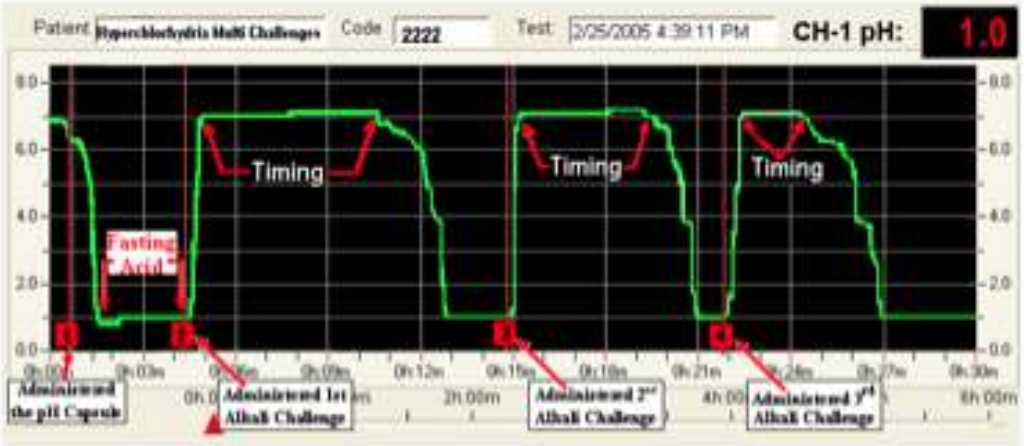
HYPERCHLORHYDRIA, WITH RAPID REACIDIFICATION
(Multi-challenged with Alkali Drink)

Hyperchlorhydria: Excess production of HCL

- Causes delayed stomach emptying time
- May retain food up to 24 hours
- Untreated will cause excoriation of stomach and intestinal mucosa
- Increased risk of gastric cancers.
- Symptoms of virtually identical to hypochlorhydria.

Hyperchlorhydria

HYPERCHLORHYDRIA, WITH RAPID REACIDIFICATION
(Multi-challenged with Alkali Drink)



- Alkaline challenge leads to rapid reacidification under 15 minutes
- Two or additional alkaline challenges to verify
- Subsequent Challenges will consistently result in re-acidification in the same time or progressively faster
- Does not require the use of a Proton Pump Inhibitor
- Treated with 2:1 sodium/potassium bicarbonate-mimics the normal physiologic buffering produced by pancreas

Hyperchlorhydria

HYPERCHLORHYDRIA, WITH RAPID REACIDIFICATION
(Multi-challenged with Alkali Drink)

- Chyme (partially digested food) absorbs and neutralizes HCL during digestion
- Normal pyloris relaxation and stomach emptying occurs at pH 4.5-5.5
- With hyperchlorhydria, excess acid reduces pH, delaying emptying
- Emptying pH will be lower (1.5 to 2.8)
- Ideal small intestine ph is 5.8 to 7.0
- Acidic chyme in proximal Duodenum Exit Region is an irritant leading to ulcers
- Nutrients, supplements, and medications subjected to caustic effect of prolonged exposure to high acid level, reducing their effectiveness
- Intestinal digestion does not occur in proper pH environment
- Pancreatic digestive enzyme activity dimininshed with lower pH

Summary

Stomach pH

Proper stomach pH is important for:

- Initiation of protein digestion
- Food sterilization
- Retention of nutrients
- Effectiveness of medications
- Proper stomach emptying
- Proper pH for small intestine digestion

Digestion Phases

- Cephalic- Sight, smell and thought of food
- Parasympathetic Vagal stimulation- 20% of HCL
- Chewing- Mechanical increase in surface area
- Amylase pH 7.0-7.4-initiates carbohydrate digestion
- Most food neutral (pH 7.0)
- HCL & Pepsin initiates protein digestion
- Mucin provides protective coat for stomach
- Stomach churning- mechanical digestion

Digestion Phases

Slight duodenal acidity (pH 4.5-5.5) stimulates:

- Duodenum to produce Secretin and CCK
- Proximal 10" of duodenum resorbs HCL, increasing pH
- Pancreas to produce digestive enzymes and bicarbonate
- Gall Bladder to secrete bile
- Set proper pH for digestive enzymes to function

Case History Heidelberg Results

Hidden Hypochlorhydria graph

Heidelberg Graph Interpretation

- Minute 0-1, start of test. Mouth pH ~7.
- Minute 1-2, capsule swallowed and enters stomach
- Minute 2-4, normal pH of ~0.5 in resting stomach
- Minute 4, challenge with 5ml. of saturated bicarbonate solution given
- Minute 4-6, pH increases back to ~6.5, indicating minimal acid in fasting stomach
- Minute 6-44, no change in pH, indicating no stomach acid production.
- Minutes 44, 49 and 53, 500mg. capsule of Betaine HCL given with gradual decrease in pH of stomach
- Minute 58, stomach returns to baseline pH (reacidifies)

Interpretation: This is hidden hypochlorhydria. It is "hidden" because the resting pH of the stomach is normal. Lack of acid production in sufficient quantity in appropriate time frame shows almost no acid production. Trial of betaine HCL may be indicated, absent any contraindications.

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