TOO DRUNK TO CARE
ABOUT MY
HEARTBURN ... *and more*

"It's a Gut Feeling!"

ICIM -- March 2016 -- Atlanta

John Parks Trowbridge M. D., FACAM

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Fundamentals of Accurate Diagnosis and Effective Treatment of Frustrating Gut Symptoms of

The Yeast Syndrome

Scientific and Clinical Evidence for Patient Management

John Parks Trowbridge M. D., FACAM

Life Celebrating Health – Houston – Texas

Atlanta ICIM Meeting, March 2016: "It's a Gut Feeling!"

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THE YEAST SYNDROME

Basic Concept:

A “yeast **infection**” is ...

... an infection of the vagina with an overgrowth of a yeastlike fungus of the genus Candida (C. albicans) normally present in the vaginal flora and characterized by vaginal discharge and vulvovaginitis; broadly : **an infection (as thrush or tinea versicolor) caused by a yeast or yeastlike fungus**.

www.merriam-webster.com/medical/yeast%20infection [emphases added]

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THE YEAST SYNDROME

Some infections are recognized, others are not:

An **infection** may cause no symptoms and be subclinical, or it may cause symptoms and be clinically apparent.

www.medicinenet.com/script/main/art.asp?articlekey=12923 [emphases added]

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THE YEAST SYNDROME

Basic Concept:

A **syndrome** is ...

A **set** of signs and symptoms that appear together and characterize a disease or **medical** condition.

dictionary.reference.com/browse/syndrome [emphases added]

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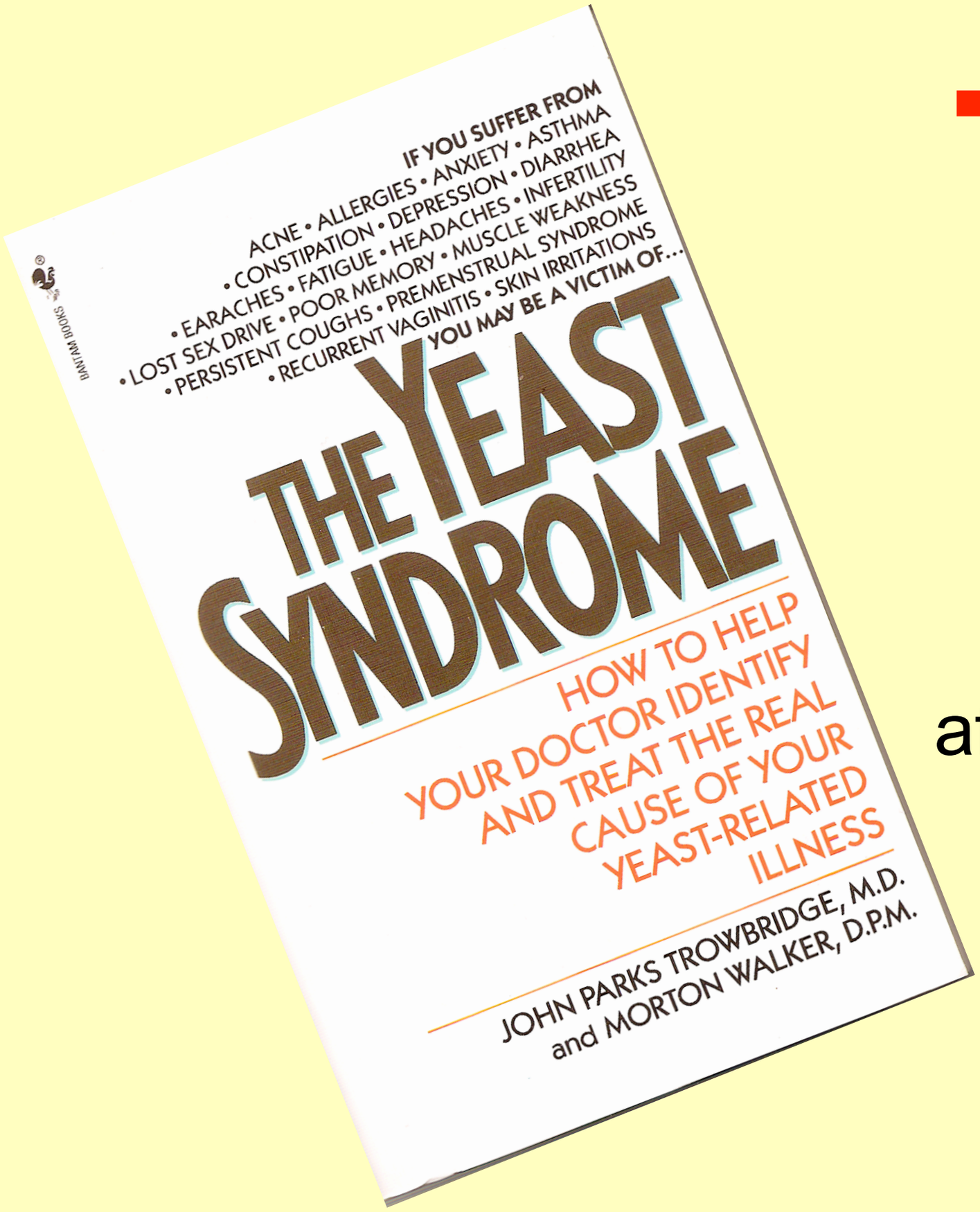

TYS

THE YEAST SYNDROME

affects 30 to 50%

of your patients

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THE YEAST SYNDROME

Basic Concept:

"The Yeast Syndrome" is ...

The yeast Candida has been **thought to cause** a syndrome with a number of **nonspecific problems** including fatigue, loss of appetite, headache, short-attention span, depression and ***all manner of intestinal irregularities***. **There is no scientific evidence to support the existence of the yeast syndrome** (also called the yeast connection).

http://www.medicinenet.com/script/main/art.asp?articlekey=6053 [emphases added]

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THE YEAST SYNDROME

Despite the ideas you will hear today ...

Fungal infections are of minor importance in general practice. ... Some exceptions are discussed. Candida spp. are **normally found** on the **surfaces** of the gastrointestinal and genital tract. The documentation of yeast from these sites is therefore **not an indication for antifungal therapy**. Esophagitis oral or vaginal thrush should however be treated promptly with oral fluconazole. Cutaneous mycoses are best treated topically or if too extensive with itraconazole or terbinafine. Treatment of **nail** infection, **if treatment is warranted at all** is best treated with terbinafine. **Any attempts to attribute uncharacteristic complaints to an intestinal colonization with yeasts or nutritional fungal toxins *lacks a scientific background*. The practice to diagnose from blood films obscure fungal infections is *quackery* at best.**

Schaffner A. [Fungal infections in primary care]. Praxis (Bern 1994). **1996** Oct 1;85(40):1236-9. [emphases added]

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THE YEAST SYNDROME

Despite the ideas you will hear today ...

The oral cavity and the oesophagus are the main sites of involvement in orointestinal candidosis. The clinical pictures of these manifestations are characterized. Involvement of the **stomach as well as the small and large intestine** is an **exceedingly rare** but possible manifestation. There are obviously no repeatedly occurring characteristical symptoms, neither have controlled studies confirmed such characteristics. Recently a discussion has arised -- undoubtedly to a large extent influenced by public media -- to explain a variety of in particular gastrointestinal symptoms as a consequence of an apparent "mycotic infection of the orointestinal tract" as "**a new mass disease**." These reports **lack any scientific basis** supported by experimental or clinical studies. There are similarities to the "candidiasis hypersensitivity syndrome" or "the yeast connection," the **existence of which has been critically denied by experts**. ... The author realizes the **necessity to oppose this public debate on a critical scientific basis** and to answer open questions by controlled studies.

Knoke M. [Clinical pictures of orointestinal candidiasis. Fiction or reality?]. Mycoses. 1996;39 Suppl 1:40-3. [emphases added]

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THE YEAST SYNDROME

In support of the “opinion” offered by the conventional medical community ...

Candida Cantadunitt was interviewed for this presentation and had this alibi to offer:

“I didn’t do it. ***I wasn’t there****.* ***No one saw me****. That’s my story, and I’m sticking to it.”*

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THE YEAST SYNDROME

GUILTY!

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THE YEAST SYNDROME

OF WHAT?

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THE YEAST SYNDROME

OF BEING LEGALLY DRUNK:

HER BLOOD ALCOHOL LEVEL = *0.18*

(BAC)

[DRIVING IMPAIRMENT: 0.08+]

A BAC of 0.1 (0.1% or one tenth of one percent)
means that there are 0.10 g of alcohol for every dL of blood

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THE YEAST SYNDROME

A woman of about 150 pounds

consuming 9 - **10+ "drinks"**

of table wine (148 ml, 5 fl oz each)

could show a BAC of about **0.30**

shortly *after* consuming ***all 10+***

http://www.brad21.org/bac_charts.html
(BeResponsibleAboutDrinking)

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THE YEAST SYNDROME

A BAC of **0.08** is considered “legally drunk”

Driving skills are affected when a BAC is between **0.04 - 0.06** (3 drinks)

Criminal penalties are associated with a BAC of **0.08 - 0.09** (4 - 5 drinks)

Legally intoxicated/criminal penalties are associated with a BAC of **0.10 - 0.28** (6+ drinks)

Death is possible with a BAC above ***0.30*** *(10+)*

http://www.brad21.org/bac_charts.html; Adapted from The ABCs of BAC, National Highway Traffic Safety Administration, 2005, and How to Control Your Drinking, WR Miller and RF Munoz, University of New Mexico, **1982**.

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THE YEAST SYNDROME

HOW DID SHE DO IT?

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THE YEAST SYNDROME

She had **one** glass of wine, finishing perhaps **2 hours** *before* being apprehended -- and her blood test was drawn about **4 hours later**

(Okay, so *maybe* it was *two?* glasses)

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THE YEAST SYNDROME

WHY?

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THE YEAST SYNDROME

She had no idea ***that*** she had done it ...

so she had no explanation as to “why”

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THE YEAST SYNDROME

“*She*” *didn’t* do it!

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THE YEAST SYNDROME

At her ***tested*** BAC of **0.18**, she should show ...

speech, **memory**, **coordination**, **attention**, **reaction time**, and **balance** significantly impaired

all **driving-related skills** dangerously impaired

judgment and decision-making dangerously impaired

vomiting and other signs of alcohol poisoning would be *common*

loss of **consciousness** *possible*

obviously an increased risk of harm to self and others

At .**400 --** Most people lose consciousness and *some* die
At .**450 --** Breathing stops. This is a fatal dose for *most* people.

http://awareawakealive.org/educate/blood-alcohol-content

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THE YEAST SYNDROME

SO ... HOW CAN WE EXPLAIN IT?

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THE YEAST SYNDROME

The effects of alcohol vary by individual and by how much alcohol they drink in one session. In fact, the effects and levels of alcohol in the body depend upon a number of factors:

- a person's size and weight
- individual metabolism rate
- related food intake
- the beverage consumed

When a person drinks an alcoholic beverage, about 20 percent of the alcohol is absorbed in the stomach and about 80 percent is absorbed in the small intestine.

NOTE: enterocolonic microbiota are ***not*** mentioned

http://www.sciencedaily.com/releases/2011/09/110915163508.htm

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Auto-Brewery Syndrome

Generally speaking, alcohol is absorbed into the blood relatively quickly and metabolized more slowly. In an average 150 pound person, for example, **each drink adds 0.02% to BAC** and **hour that passes removes 0.01%** from it. This is why alcohol concentrations build steadily throughout a drinking session. One serving of alcohol is fully absorbed into the blood stream **within 30 minutes to 2 hours** after intake.

The **liver is responsible for the elimination -** through metabolism - of **95% of ingested alcohol** from the body. The remainder of the alcohol is eliminated through excretion of alcohol in breath, urine, sweat, feces, milk and saliva. The body uses several different metabolic pathways in its **oxidation of alcohol to *acetaldehyde*** to acetic acid to carbon dioxide and water.

http://prevention.gwu.edu/alcohol-absorption. [emphases added]

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Auto-Brewery Syndrome

Alcohol burns off at a precise rate of .016 BAC per hour, about equal to **1 standard drink each hour** (depending on your weight). This rate is true regardless of the size of your body. A 5'2" female burns off alcohol at the same rate as a 6'1" obese male.

The **differences**, however, are in the **rate with which your BAC rises**. One drink in a small female of low weight constitutes a much larger percent of her BAC. It may take a male 5 drinks or more in an hour to reach a BAC of .08, while it may take a small female only 2 or three drinks.

http://www.intox.com/t-Physiology.aspx. . [emphases added]

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Auto-Brewery Syndrome

Retrograde extrapolation is the mathematical process by which someone's blood alcohol ***concentration at the time of driving* is estimated by *projecting backwards* from a *later* chemical test**. This involves estimating the absorption and elimination of alcohol in the interim between driving and testing. The **rate of elimination** in the average person is commonly **estimated at .015 to .020 grams per deciliter per hour (g/dl/h)** ...

In an increasing number of states, laws have been enacted to facilitate this speculative task: the **blood alcohol content at the time of driving is *legally presumed to be the same* as when later tested**. There are usually time limits put on this presumption, **commonly *two* or *three* hours**, and the defendant is permitted to offer evidence to rebut this presumption.

Montgomery, Mark R.; Reasor, Mark J. (**1992**). "Retrograde extrapolation of blood alcohol data: An applied approach." Journal of Toxicology and Environmental Health **36** (4): 281–92. [emphases added]

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Auto-Brewery Syndrome

By such a retrograde extrapolation ...

she could ***never*** have achieved a

BAC of 0.18 by consuming merely one

(or maybe *?two?*) glasses of wine

about ***6*** hours before the blood test

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Auto-Brewery Syndrome

So ... how much a “bad girl” was she?

If the **amount of alcohol consumed is known**, along with such variables as the weight and sex of the subject and period and rate of consumption, the **blood alcohol level can be estimated by extrapolating forward**.

Although subject to the same infirmities as **retrograde** extrapolation—guessing based upon **averages** and unknown **variables**—this can be relevant in **estimating BAC that was present when she last was drinking --** in her case, the test was taken **6 hours after** she finished drinking.

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Auto-Brewery Syndrome

At about 2 am, her BAC was 0.18

At about 8 pm, she finished drinking wine

Assuming each *drink* ***ADDS*** 0.02 and each *hour* ***REMOVES*** 0.01 - 0.02 ...

In 6 hours, she would have removed 0.06 - 0.12

So a total *starting* "**load**" would have to have amounted to about ...

(tested) **0.18** **+** (metabolized) **0.12** **=** (consumed) **0.30** "at 8 pm"

That would equal consumption of about ***10 - 15*** **drinks** (each contributes about 0.02), finishing by about 8 pm, **about 6 hours before blood testing**

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Auto-Brewery Syndrome

Unlike foods, which require time for digestion, alcohol needs no digestion and is **quickly absorbed**. ... About 20 percent is absorbed directly across the walls of an empty stomach and can reach the brain within one minute.

Alcohol is **rapidly absorbed** in the upper portion of the small intestine. The alcohol-laden blood then travels to the liver [and] affects nearly every liver cell. The **liver cells are the only cells in our body that can produce enough of the enzyme alcohol dehydrogenase** to oxidize alcohol at an appreciable rate. ...

reference on next slide

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Auto-Brewery Syndrome

Alcohol metabolism **permanently changes liver cell structure**, which impairs the liver's ability to metabolize fats. This explains why heavy drinkers tend to develop fatty livers.

The liver is able to metabolize about ½ ounce of ethanol **per hour** (approximately **one drink**, depending on a person's body size, food intake, etc.). If more alcohol arrives in the liver than the enzymes can handle, the **excess alcohol travels to all parts of the body**, circulating until the liver enzymes are finally able to process it.

http://www.alcohol-stuff.co.uk/guides/how-long-does-alcohol-stay-in-your-system.html. [emphases added]

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Auto-Brewery Syndrome

The term "auto-brewery syndrome" has been frequently used to describe patients who show **features of alcohol intoxication because of abnormal yeast proliferation after ingesting carbohydrate-rich meals.** We present a case of a 3-y-old girl with short bowel syndrome (SBS) who demonstrated signs of alcohol intoxication on repeated occasions. A blood test indicated an **ethanol concentration of 15 mmol/l**, and cultures from gastric fluid and faeces showed the presence of **Candida kefyr**. An association was found between the introduction of a **carbohydrate-rich fruit drink and the occurrence of symptoms**.

Jansson-Nettelbladt E, Meurling S, Petrini B, Sjölin J. Endogenous ethanol fermentation in a child with short bowel syndrome. Acta Paediatr. **2006** Apr;95(4):502-4. [emphases added]

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Auto-Brewery Syndrome

Conclusion: The possibility of **endogenous ethanol fermentation** should be considered in patients with SBS and the diagnosis of auto-brewery syndrome added to the differential diagnosis list for D-lactic acidosis. Management includes both **antifungal** treatment and special **diet modification**.

Jansson-Nettelbladt E[1], Meurling S, Petrini B, Sjölin J. Endogenous ethanol fermentation in a child with short bowel syndrome. Acta Paediatr. **2006** Apr;95(4):502-4. [emphases added]

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Auto-Brewery Syndrome

The concentration of ethanol in blood, breath or urine constitutes important evidence for **prosecuting drunk drivers**. ... One such argument for acquittal concerns the notion that alcohol could be produced naturally in the body, hence the term 'auto-brewery' syndrome. Although **yeasts such as Candida albicans readily produce ethanol in-vitro**, whether this happens to any measurable extent in healthy ambulatory subjects is an open question. Over the years, many determinations of endogenous ethanol have been made, and **in a few rare instances** (Japanese subjects with very serious yeast infections) an **abnormally high ethanol** concentration (> 80 mg/dl) has been reported. In these atypical individuals, **endogenous ethanol appeared to have been produced after they had eaten carbohydrate-rich foods**.

reference on next slide

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Auto-Brewery Syndrome

With reliable gas chromatographic methods of analysis, the concentrations of endogenous ethanol in peripheral **venous blood** of healthy individuals, as well as those suffering from **various metabolic disorders** (diabetes, hepatitis, cirrhosis) ranged from **0-0.08 mg/dl**. These concentrations are **far too low to have *any* forensic or medical significance**. The notion that a motorist's state of **intoxication** was caused by **endogenously produced ethanol *lacks merit*.**

Logan BK, Jones AW. Endogenous ethanol 'auto-brewery syndrome' as a drunk-driving defence challenge. Sci Law. **2000** Jul;40(3):206-15. [emphases added]

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Auto-Brewery Syndrome

Four common **yeasts** (Candida albicans, Candida tropicalis, Torulopsis glabrata and Saccharomyces cerevisiae) were combined with five **infant food formulas** and/or supplements (Isomil, Nutramigen, 5% glucose, Coca Cola and Similac) and incubated at 37 degrees C. Gas chromatography was used to measure ethanol production after 24 and 48 h incubation. The **quantities of ethanol produced suggest a *possible* explanation for patients exhibiting the 'Auto-Brewery Syndrome'** and raises interest in the role auto-produced ethanol could have in explaining the *etiology of Sudden Infant Death*.

Bivin WS, Heinen BN. Production of ethanol from infant food formulas by common yeasts. J Appl Bacteriol. **1985** Apr; 58(4):355-7. [emphases added]

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Auto-Brewery Syndrome

In some cases of sudden infant death syndrome (**SIDS**) the **intestinal** flora was found to be **dominated by Candida albicans**. Microbiologic investigations of the *various organs* showed the occasional presence of different Candida species, but not in the form of massive growth as in sepsis. **There is no basis to assume that the activity of yeasts, first of all of Candida albicans, is a contributory factor in the occurrence of SIDS**. Candida albicans was shown to produce alcohol from glucose at a rate of maximally 1 mg of alcohol per gram of intestinal content per hour. It is concluded that the intestinal production of alcohol in vivo from cases showing a Candida albicans dominated intestinal flora will *not be able to surpass the normal alcohol metabolizing capacity of the liver*.

Thus, **measurable concentrations of alcohol in the blood from such cases cannot be expected**.

Geertinger P, Bodenhoff J, Helweg-Larsen K, Lund A. Endogenous alcohol production by intestinal fermentation in sudden infant death. Z Rechtsmed. **1982**;89(3):167-72. [emphases added]

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Auto-Brewery Syndrome

The case of the gastrointestinal production of ethanol from **Candida albicans** and Saccharomyces cerevisiae in a Caucasian man with **chronic intestinal pseudo-obstruction** is reported. The patient, who declared to have **always abstained from alcohol**, was hospitalized for **abdominal pain, belching and mental confusion**. The laboratory findings showed the presence of **ethanol in the blood**. Gastric juice and faecal microbiological cultures were positive for C. albicans and S. cerevisiae. At home, **he was on oral antibiotic therapy** with amoxicillin plus clavulanic acid for a small bowel bacterial overgrowth, associated with a simple sugar-rich diet. ***Twenty-four hours after stopping both the antibiotic therapy and the simple sugar-rich diet, the blood ethanol disappeared***. A ***provocative test***, performed by giving amoxicillin plus clavulanic acid associated with the simple sugar-rich diet was followed by the ***reappearance of ethanol*** in the blood.

Spinucci G, Guidetti M, Lanzoni E, Pironi L. Endogenous ethanol production in a patient with chronic intestinal pseudo-obstruction and small intestinal bacterial overgrowth. Eur J Gastroenterol Hepatol. **2006** Jul;18(7):799-802. [emphases added]

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Auto-Brewery Syndrome

The **gut may prime inflammatory responses** following shock/trauma insults. **Ethanol (EtOH) use** is common in trauma patients and may **impair intestinal barrier function**. We compared varying concentrations of EtOH on proinflammatory cytokine production of Caco2 cell monolayers and the resultant changes in barrier function. ... Caco2 production of TNF-alpha increased in a dose-dependent manner when incubated with increasing concentrations of EtoH. ... In addition to alterations in gut permeability, **increasing concentrations of ethanol have a synergistic effect with *E. coli* on** Caco2 **production of proinflammatory cytokines TNF and IL-6**. The creation of a proinflammatory cytokine milieu with an altered barrier integrity may be a **mechanism by which ethanol may increase septic complications in the *injured* patient**.

Amin PB1, Diebel LN, Liberati DM. Dose-dependent effects of ethanol and E. coli on gut permeability and cytokine production. J Surg Res. **2009** Dec;157(2):187-92. [emphases added]

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Auto-Brewery Syndrome

ABS symptoms can include ***apparent acetaldehyde*** *effects*:

Many human colonic facultative anaerobic and aerobic bacteria are capable of alcohol dehydrogenase (ADH)-mediated ethanol oxidation. In this **bacteriocolonic pathway for ethanol oxidation** intracolonic ethanol is first oxidized by bacterial ADHs to acetaldehyde, which is further oxidized by either colonic mucosal or bacterial aldehyde dehydrogenases to acetate. ... Under aerobic conditions both [tested] E. coli strains oxidized ethanol. This study experimentally shows the potential of **certain bacteria representing normal human colonic flora to produce *acetaldehyde*** under various atmospheric conditions that may prevail **in different parts of the GI tract**. This bacterial adaptation may be an essential feature of the bacteriocolonic pathway to **produce toxic and carcinogenic acetaldehyde from either endogenous or exogenous ethanol**.

Salaspuro V, Nyfors S, Heine R, Siitonen A, Salaspuro M, Jousimies-Somer H. Ethanol oxidation and acetaldehyde production in vitro by human intestinal strains of Escherichia coli under aerobic, microaerobic, and anaerobic conditions. Scand J Gastroenterol. **1999** Oct;34(10):967-73. [emphases added]

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Auto-Brewery Syndrome

[I]ntracolonic ethanol levels are equal to those of the **blood ethanol levels**. In the large intestine, **ethanol is oxidized** by colonic bacteria, and this can lead to **extraordinarily high acetaldehyde levels** that might be responsible, in part, for ethanol-associated **carcinogenicity and gastrointestinal symptoms**. ... [I]n addition to ADH [alcohol dehydrogenases], part of the acetaldehyde produced in the large intestine during ethanol metabolism can be [bacterial] catalase dependent.

Tillonen J, Kaihovaara P, Jousimies-Somer H, Heine R, Salaspuro M. Role of catalase in in vitro acetaldehyde formation by human colonic contents. Alcohol Clin Exp Res. **1998** Aug;22(5):1113-9. [emphases added]
also
Jokelainen K[1], Siitonen A, Jousimies-Somer H, Nosova T, Heine R, Salaspuro M. In vitro alcohol dehydrogenase-mediated acetaldehyde production by aerobic bacteria representing the normal colonic flora in man. Alcohol Clin Exp Res. **1996** Sep;20(6):967-72.

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Auto-Brewery Syndrome

Her BAC was **0.18** some 6 hours after she finished consuming alcohol, with a projected **likely BAC of 0.30** shortly after taking her last drink. **Above 0.31, this is the mental level of coma or surgical amnesia**; the possibility of acute alcohol intoxication is very real, and death due to respiratory arrest is likely in 50% of drinkers.

In 1982, a 24-year-old woman was admitted to the UCLA emergency room with a serum alcohol concentration of 1.51% (1,510 mg/dL), corresponding to a BAC of **1.33**%. She was alert and oriented to person and place.

In February 2005, French gendarmes from Bourg-en-Bresse, France, conducted a breath test on a man who had lost control of his car. He had an alcohol content of **0.976**%. He was not injured in the accident but was charged with a €150 fine and his driving license was canceled.

In 2013, on July 26 a 30-year-old man from Alfredówka, Poland, was found by Municipal Police Patrol fromNowa Dęba lying in the ditch along the road in Tarnowska Wola. At the hospital there was recorded that the man had 13.74 permille of alcohol in the blood (**1.374**%). The man survived.

In South Africa, a man driving a Mercedes-Benz Vito light van containing 15 sheep, allegedly stolen from nearby farms, was arrested on December 22, 2010, near Queenstown in Eastern Cape. His blood had an alcohol content of **1.6**%.

Rebecca Martinson. These People Who All Managed To Have A BAC Over 1% Are Either My Heroes Or The Worst Drunks Ever. BroBible 10.06.14. http://brobible.com/life/article/worst-drunks-ever/ [emphases added]

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Auto-Brewery Syndrome

A woman who lives in upstate New York recently escaped drunk driving charges by convincing a judge that she hadn't been hitting the breweries—her body had literally become one, thanks to a rare disorder dubbed auto-brewery syndrome, in which an excess of intestinal yeast causes ingested food and drinks to ferment into alcohol.

According to *The Buffalo News*, the defendant, a 35-year-old schoolteacher, was pulled over in Erie County last year after tipsters reported she was driving erratically. While she said that she had consumed three drinks earlier that day, her Breathalyser results told a different story: Her blood alcohol content (**BAC**) was a **staggering .33 percent**—more than four times the state's legal limit of .08.

"She can register a blood alcohol content that would have you or I falling down drunk, **but she can function**," the defendant's attorney, Joseph Marusak, told *The Buffalo News*. (**The Erie County District Attorney's office is reportedly planning to appeal the judge's ruling.**) ... **[A]s many as 95 percent of sufferers might not even know they have it.** ... Some patients have used low-carb, low-sugar diets and anti-fungal drugs to treat their symptoms.

Kirstin Fawcett. Woman Beats Drink-Driving Charge by Proving That Her Body Brews Beer. Mental Floss. 06 . 01 . **16**. http://mentalfloss.com/uk/science/37394/woman-beats-drink-driving-charge-by-proving-that-her-body-brews-beer. [emphases added]

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Auto-Brewery Syndrome

Kaji H, Asanuma Y, Ide H, Saito N, Hisamura M, Murao M, Yoshida T, Takahashi K. The auto-brewery syndrome--the repeated attacks of alcoholic intoxication due to the overgrowth of Candida (albicans) in the gastrointestinal tract. Mater Med Pol. **1976**;8(4):429-35.

Barbara Cordell, Justin McCarthy (July **2013**). "A Case Study of Gut Fermentation Syndrome (Auto-Brewery) with Saccharomyces cerevisiae as the Causative Organism." International Journal of Clinical Medicine 4:309–312.

Auto-brewery syndrome has **never been convincingly reported outside Japan**. Why? It's all about enzymes. When the liver processes ethanol, the enzyme alcohol dehydrogenase first converts it to acetaldehyde. ... What's likely happening in the Japanese cases is a combination of high carb intake (which the Japanese diet is famous for), yeast infection, and a limited ability to metabolize the alcohol produced - any of these alone probably won't do the trick. My guess is that even without considering their BAC, auto-brewery sufferers might be in no condition to drive, since they'd be under the influence of acetaldehyde.

Cecil Adams. Designated drunk: Can you get intoxicated without actually drinking alcohol? The Straight Dope. October 20, **2006**. http://www.straightdope.com/columns/read/2677/designated-drunk. [emphases added]

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Auto-Brewery Syndrome

TAKE-HOME MESSAGE

Auto-brewery syndrome, where production of alcohol (and acetaldehyde) by an overgrowth of opportunistic enterocolic organisms -- predominantly *Saccharomyces* species and/or *Candida albicans* -- creates unexpected hazards and harm both to the patient and to society as a whole.

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THE YEAST SYNDROME

“How do *you*

spell R-E-L-I-E-F?”

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GERD

Gastroesophageal Reflux Disorder

"Heartburn"

Acid reflux

Acid indigestion

Water-brash

... leading to ***inflammation*** of esophagus

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GERD

Heartburn, also known as **acid indigestion**, is a burning sensation in the central chest or upper central abdomen. The pain often rises in the chest and may radiate to the neck, throat, or angle of the jaw.

Heartburn is usually associated with regurgitation of gastric acid (gastric reflux) which is the major symptom. It may also be a symptom of ischemic heart disease, though this is true for only 0.6% of those experiencing heartburn.

It is often called a "*disease*," but I prefer "*disorder*."

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GERD

As many as **30%** of chest pain patients **undergoing cardiac catheterization have findings which do not account for their chest discomfort**, and are often defined as having "***atypical*** chest pain" or chest pain of undetermined origin.

According to data recorded in several studies based on ambulatory pH and pressure monitoring, it is estimated that **25% to 50% of these patients** have evidence of abnormal GERD.

In other words, about **10 to 15% of patients undergoing cardiac cath** have adequate coronary circulation and have pathologic changes consistent with **GERD**.

Richard W. Tobin, M.D., Charles E. Pope II, M.D., and Deborah L. Greenburg, M.D. Heartburn and Regurgitation. University of Washington Division of Gastroenterology. http://www.uwgi.org/guidelines/ch_03/ch03txt.htm [emphases added]

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“NOT” GERD

“***Functional*** indigestion” (previously called “non-ulcer” dyspepsia) is indigestion “***without* evidence of an organic disease** that is likely to explain the symptoms.” It is associated with other “*functional* gastrointestinal disorders” like irritable bowel syndrome and is the **primary cause of lack of improvement** post treatment with proton pump inhibitors (PPIs).
PPIs are however “still the primary treatment” with response rates in about 50% of people. [Prevacid, Nexium, Prilosec -- all imid***azoles*** ... ***hint?***]
“*Functional* indigestion” is estimated to affect about **15%** of the general population in western countries.

Fass R (January **2009**). "Functional heartburn: what it is and how to treat it". Gastrointest. Endosc. Clin. N. Am. 19 (1): 23–33; Kato H, Ishii T, Akimoto T, Urita Y, Sugimoto M (April **2009**). "Prevalence of linked angina and gastroesophageal reflux disease in general practice". World J. Gastroenterol. 15 (14): 1764–8. [emphases added]

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“NOT” GERD

The diagnosis of “***functional*** heartburn” is one of elimination, based upon the Rome III criteria:

1) burning retrosternal discomfort
2) **elimination** of heart attack and **GERD** as the cause and
3) no esophageal motility disorders.

Functional **disease** (***functional*** **disorder**) a disease or disorder of physiological function having **no *known* organic basis**. Although not strictly correct, the term is often used in psychiatry for disorders that are psychogenic and in other branches of medicine to refer to any that are **idiopathic**. [*idiot* pathology = one’s own, private, peculiar]

Not organic in origin; denoting a disorder with **no *known* or detectable organic basis** to explain the symptoms.

Fass R (January **2009**). "Functional heartburn: what it is and how to treat it". Gastrointest. Endosc. Clin. N. Am. 19 (1): 23–33 [emphases added on all three references]
Miller-Keane Encyclopedia and Dictionary of Medicine, Nursing, and Allied Health, Seventh Edition. © 2003 by Saunders, an imprint of Elsevier, Inc. http://medical-dictionary.thefreedictionary.com/functional.
Farlex Partner Medical Dictionary © Farlex 2012. http://medical-dictionary.thefreedictionary.com/functional

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GERD

The **chest pain caused by GERD** has
a distinct 'burning' sensation,
occurs after eating or at night, and
worsens when a person lies down or bends over.

Common in pregnant women [*progesterone* promotes yeast proliferation]

May be triggered by consuming food in large quantities, or specific foods containing certain spices, high fat content, or high acid content.

Patients may undergo an **upper GI series** to confirm the presence of “acid reflux.” Heartburn or chest pain after eating or drinking ***and*** combined with difficulty swallowing may indicate esophageal spasms.

National Digestive Diseases Information Clearinghouse (NDDIC): Upper GI Series. Accessed May 18, **2010**. [emphases added]

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GERD

A "***complication"*** of GERD is **chronic inflammation** and tissue damage in the **esophagus**.

A bacterial, viral or fungal infection in tissues of the esophagus may cause esophagitis. **Infectious esophagitis is relatively rare** and occurs most often in people with *poor immune system function*, such as people with HIV/AIDS or cancer.

A fungus normally present in the mouth called **Candida albicans** is a common cause of infectious esophagitis. Such infections are often associated with *poor immune system function*, diabetes, cancer and ***antibiotic [or steroid] use***.

Mayo Clinic website. http://www.mayoclinic.org/diseases-conditions/esophagitis/basics/causes/con-20034313. [emphases added]

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GERD

A prospective study was conducted in 224 patients to determine the clinical **significance of esophageal colonization with yeasts** under different conditions. In accordance with the results of direct smear microscopic examination and culture of esophageal brushings, patients were divided into three groups: positive, negative, and the patients, in whom saprophytic forms were detected. A **higher prevalence of positive findings was noted in patients with predisposing factors for yeast invasion** than in patients free of underlying disease. **Eleven percent of patients with an endoscopically *normal appearing* esophagus were positive**. We have the impression that this situation **may represent a preclinical** condition of fungal esophagitis. **Patients treated with H2-blocking agents showed a significantly higher incidence of positive findings**, than did those without such treatment. Whether patients suffering from a **reflux esophagitis resistant to long-term treatment with H2 blockers, but with a significant colonization by yeasts, could benefit by an additional treatment with antimycotics** remains a ***controversial*** issue and should be studied in a controlled way.

Vermeersch B[1], Rysselaere M, Dekeyser K, Rasquin K, De Vos M, Elewaut A, Barbier F. Fungal colonization of the esophagus. Am J Gastroenterol. 1989 Sep;84(9):1079-83.

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GERD

A prospective search for gastro-oesophageal candidiasis was made by ***histological* examination** of all the biopsies taken from 465 patients endoscoped consecutively during a 12 month period. The criterion for diagnosis was the demonstration of **infiltration of tissue or ulcer slough by yeasts** and hyphae. Nineteen cases of candidiasis were found giving an overall incidence of 4%. There were **12 cases with *oesophageal* candidiasis, two with both oesophageal and gastric candidiasis, and five with *gastric* candidiasis**. ***In none of the patients was candidiasis suspected before endoscopy*.** **Symptoms** referable to the candidiasis were **uncommon** and **radiology was not helpful** in diagnosis. There was associated local pathology (particularly peptic ulceration and carcinoma of the stomach or oesophagus) in all except two patients, which suggests that the **candidiasis is usually secondary to *mucosal damage***.

Scott BB, Jenkins D. Gastro-oesophageal candidiasis. Gut. **1982** Feb;23(2):137-9. [emphases added]

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GERD

What's WRONG with conventional care?

Antacids such as calcium carbonate are often taken to treat the immediate problem, with further treatments depending on the underlying cause.

Medicines such as **H2** receptor antagonists [Tagamet, Zantac] or **proton pump inhibitors** [Prilosec, Prevacid,Nexium] are effective for gastritis and GERD, the two most common causes of heartburn.

Antibiotics are used if H. pylori is present (confirm with breath test).

About 42% of the United States population has had heartburn at some point.

Recurrent courses of treatment are **chronic**, sometimes slipping into **continuous** chronic medication.

A surprisingly large minority of patients complain of **frequent or persistent "heartburn,"** despite years of seeking intermittent or continuous "treatment."

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GERD

Chronic acid suppression therapy encourages intestinal seeding of Candida albicans and other microbes:

Quantitative **cultures** were carried out on samples from gastric juice obtained from 12 ambulatory patients with esophagitis before and one month after **omeprazole therapy**. An ***increase*** in the number of patients in whom gastric juice was culture-positive, as well as an ***increment*** in the bacterial counts were noted. The **spectrum of microorganisms isolated from gastric juice was identical to the normal flora of the oral cavity**, mainly alpha-hemolytic streptococci, corynebacteria, and **Candida** species. Thus, the counts of organisms within gastric contents are **simply a reflection of swallowed oral microflora** that were able to survive due to the less acidic environment.

Karmeli Y, Stalnikowitz R, Eliakim R, Rahav G. Conventional dose of omeprazole alters gastric flora. Dig Dis Sci. **1995** Sep;40(9):2070-3. [emphases added]

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GERD

Chronicity carries distinctive risks, because GERD sometimes causes injury of the esophagus, often one or more of the following:

- Reflux esophagitis – **necrosis of esophageal epithelium** causing ulcers near the junction of the stomach and esophagus
- Esophageal **strictures** – the **persistent narrowing** of the esophagus caused by reflux-induced inflammation, later progressing to dysphagia
- **Barrett's esophagus** – intestinal **metaplasia** (changes of the epithelial cells from squamous to intestinal columnar epithelium) of the distal esophagus which may progress to esophageal cancer in perhaps 20% of patients
- Esophageal **adenocarcinoma** – a form of cancer

https://en.wikipedia.org/wiki/Gastroesophageal_reflux_disease [emphases added]

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GERD

Diagnosis by **esophagoscopy**:

Mycology confirmed the presence of filamentous form of Candida albicans in 23 patients and pathology showed non-specific lesions of esophagitis, 20 with intramucous hyphae. HIV serology was positive in 19/23 patients (82.6%) and in 1/15 controls (6.6%). **Sensitivity and specificity of upper GI endoscopy for the diagnosis of Candida albicans were 100 and 83.3**% respectively; **positive and negative predictive values** were **88.5** and **100**%, respectively.

Upper digestive **endoscopy is a reliable method** for the diagnosis of Candida albicans esophagitis. However, mycological confirmation is warranted.

Redah D, Konutse AY, Agbo K, Dogbey EH, Napo-Koura G, Tchangai-Kao ST, Prince-David M, Amedegnato DM, Agbetra A. [Is endoscopic diagnosis of Candida albicans esophagitis reliable? Correlations with pathology and mycology]. Gastroenterol Clin Biol. **2001** Feb;25(2):161-3. [emphases added]
Redah D, Konutse AY, Agbo K, Dogbey EH, Napo-Koura G, Tchangai-Kao ST, Prince-David M, Amedegnato DM, Agbetra A. [Is endoscopic diagnosis of Candida albicans esophagitis reliable? Correlations with pathology and mycology]. Gastroenterol Clin Biol. **2001** Feb;25(2):161-3. [emphases added]

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GERD

The yeast Candida albicans is a harmless commensal in most healthy people, but it causes superficial as well as life-threatening systemic infections in immunocompromised patients. C. albicans can colonize or infect virtually all body sites because of its **high adaptability to different host niches**, which involves the **activation of appropriate sets of genes in response to complex environmental signals**. ... Our results demonstrate that SAP expression depends on the type of infection, with **different SAP isogenes being activated during systemic disease as compared with mucosal infection**. In addition, the activation of individual SAP genes depends on the **progress of the infection**. ... The in vivo expression technology allows the elucidation of gene expression patterns at different stages of the fungus-host interaction, thereby revealing **regulatory adaptation mechanisms that make C. albicans the most successful fungal pathogen** of humans and, at the same time, identifying the stage of an infection at which certain virulence genes may play a role.

Staib P, Kretschmar M, Nichterlein T, Hof H, Morschhäuser J. Differential activation of a Candida albicans virulence gene family during infection. Proc Natl Acad Sci U S A. **2000** May 23;97(11):6102-7. [emphases added]

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GERD

Could negative histologic samples ***misdirect*** clinical attention away from yeast?

Esophageal involvement was documented by mucosal lesions, microbiological findings of candidal infection in smears of brushing material, positive cultures of brushing material, and histological evidence of mucosal invasion by the yeast. For **21 of the 22** patients studied, there were **endoscopic and microbiological** findings of candidal esophagitis. **Cultures of the brushing** material from all 22 patients were **positive**, while ***histological evidence was found for [only] 14 patients.***

Samonis G, Skordilis P, Maraki S, Datseris G, Toloudis P, Chatzinikolaou I, Georgoulias V, Bodey GP. Oropharyngeal candidiasis as a marker for esophageal candidiasis in patients with cancer. Clin Infect Dis. **1998** Aug;27(2):283-6. [emphases added]

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GERD

The aim of the study was to demonstrate morphological traits of **Candida-induced upper alimentary tract mucosa inflammation**. The material for the study comprised 18 children aged 4 to 18 years treated at the I Department of Paediatrics and Gastroenterology, Institute Polish Mother Health Centre. In these children **positive mycologic cultures** were obtained from alimentary tract inflammed mucosa sections.

Candida fungi may be an etiopathogenetic factor of oesophageal, gastric and duodenal mucosa inflammation, **Candida-induced mucosa inflammation is most frequently of chronic nature**.

Fortak B, Płaneta-Małecka I, Trojanowska-Lipczyk J, Czkwianianc E, Dyńiska E, Kozieł B. [The role of Candida sp. in etiopathogenesis of esophageal, gastric and duodenal mucosa inflammation in children]. Wiad Parazytol. **2004**;50(3): 381-6. [emphases added]

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GERD

My clinical experience for successful treatment over the past 32 years:

Comprehensive treatment for The Yeast Syndrome is ***the key*** to long term remission and should not be postponed when indicated.

Locally:

Mycelex [clotrimazole] oral troches 10 mg #100 refill once --

Sig: dissolve 1 in mouth qid, ac and hs

Carafate [sucralfate] suspension 480 ml refill once --

Sig: slowly swallow 2 tsp po ac (after Mycelex) and hs -- do not rinse

[or tablets 1 g #100 refill only if calls -- 1 po qid ac and hs with minimal liquid; if desired, dissolve in small volume water]

aloe vera gel/papaya juice -- equal volumes, sip continuously for discomfort

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GERD

TAKE-HOME MESSAGE

Gastroesophageal reflux (GERD) patterns, usually treated by **unrealistically chronic acid-reducing medications** that impair digestion of proteins and preparation of minerals for absorption, can hasten development of debilitating chronic diseases; treatment of fungal overgrowth penetrating the esophageal epithelium can **resolve symptoms** and reduce risks for developing pre-cancerous Barrett's esophagus.

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THE YEAST SYNDROME

STRESS MAKES ULCERS?

If you ask what is the single most important key to longevity, I would have to say it is avoiding worry, stress and tension. And if you didn't ask me, I'd still have to say it.

– George Burns

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PEPTIC ULCERS

Peptic ulcer disease (PUD), usually treated by chronic acid-reducing medications that impair digestion of **proteins** and preparation of **minerals** for absorption--hastening development of **debilitating chronic diseases**--can be **resolved completely** in most patients by proper treatment of aggravating yeast ***and/or*** bacterial species and restoring more normal digestive processes.

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PEPTIC ULCERS

I'm a 35 year old woman. I have been using **Protonix** 20mg **since 2003** after being diagnosed with a moderate to severe **hiatal hernia**. A few years ago I developed a **yeast infection that would not go away or would go away but quickly return**. Last year I developed a **sinus infection** that was also **resistant to treatment** but finally got better with **steroid** treatment but I continue to have some discomfort. In April I developed **abdominal pain**. Ultrasound, CT scan & blood work all came back ok. My symptoms are **IBS** in nature (pain, constipation, diarrhea, bloating, etc) but the pain tends to stay in the upper portion of my intestine. Over the years I have also developed **dry eye**, **dry skin**, **dry brittle hair**, **fatigue** & occassional **sore joints**. I had originally thought I had an autoimmune disease like Sjogren's but my primary doctor ruled this out with blood work. I have read recent studies are showing that people on PPI medications, like **Protonix, long term can develop an overgrowth of bacteria from low acid amounts**. I'd like to know if my symptoms sound like this is a possibility. If so, I will insist upon having testing done with my doctor. If this is the case, is there a treatment?

Over-the-counter **PPIs** (omeprazole ...) should only be used as directed for 14 days for the treatment of frequent heartburn. **FDA warns that no more than three 14-day treatment courses should be used in one year.** ... Your symptoms suggest a **colonic cause** more than adverse effects of PPIs. You will do well to consult a gastro-enterologist and get yourself assessed. [He's "right" -- it's TYS!!!]

http://www.doctorslounge.com/gastroenterology/forums/backup/topic-46002.html. [emphases added]

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PEPTIC ULCERS

Candida albicans frequently inhabits the gastrointestinal tract of humans leading to **gastrointestinal candidiasis, especially following suppression of gastric acidity,** but studies on the relation between this fungal infection and gastric pathology are limited due to lack of convenient animal models resembling Candida infection in humans.

... The area of ulcers in control ***rats*** significantly decreased at day 15 and the ulcers disappeared almost completely after 25 days of their induction. In contrast, the **ulcers were present until day 25 in Candida-inoculated rats** followed by a fall in GBF [gastric blood flow] and a rise in plasma gastrin levels, these effects being significantly attenuated by the co-treatment with Lactobacillus. Candidiasis was **accompanied by up-regulation** of mRNA for IL-1beta, TNF-alpha, EGF and TGFalpha and a significant increment in plasma IL-1beta and TNF-alpha levels.

1) **Persistent colonization with Candida** could be achieved in rats treated with **antisecretory agents** or non-steroidal anti-inflammatory drugs (**NSAIDs**) such as ASA; 2) **candidiasis reduces gastric acid secretion**, while **delaying ulcer healing** possibly due to the impairment in GBF in the ulcer area and enhanced expression and release of IL-1beta and TNFalpha and 3) **probiotic therapy** could be useful in the treatment **against the deleterious action of fungal infection on the healing of pre-existing gastric ulcers**.

Brzozowski T, Zwolinska-Wcislo M, Konturek PC, Kwiecien S, Drozdowicz D, Konturek SJ, Stachura J, Budak A, Bogdal J, Pawlik WW, Hahn EG. Influence of gastric colonization with Candida albicans on ulcer healing in rats: effect of ranitidine, aspirin and probiotic therapy. Scand J Gastroenterol. **2005** Mar;40(3):286-96. [emphases added]

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PEPTIC ULCERS

This paper reports the results of a multicenter prospective study of 188 consecutive patients affected by **gastric ulcer**, verified by endoscopy, in whom the frequency of a mycotic infection of the lesion was evaluated as well as the eventual influence of such pathology on the efficiency of medical treatment, the healing rate, and the healing time. A **mycotic infection, defined as penetration of the periulcerous mucosa by the fungi**, was **found in only 13 patients** (**6.9%**). ***No significant differences were found in the healing rate and healing time among these patients treated with H2-receptor antagonists and a control group*** of 43 matched gastric ulcer patients treated in the same period with the same therapy. It would appear from the data that **mycotic infections of the gastric ulcer *do not modify* the efficiency of medical treatment.**

Di Febo G, Miglioli M, Calò G, Biasco G, Luzza F, Gizzi G, Cipollini F, Rossi A, Barbara L. Candida albicans infection of gastric ulcer frequency and correlation with medical treatment. Results of a multicenter study. Dig Dis Sci. **1985** Feb;30(2):178-81. [emphases added]

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PEPTIC ULCERS

Candida overgrowth in gastric juice of peptic ulcer subjects under **therapy with H2-receptor** (H2-R) antagonists has been detected in 21.4 and **53.8%** of cases after short- and **long-term treatment** respectively, and in 8% of controls. Both types of H2-R antagonists, ranitidine and cimetidine, were equally associated with production of yeasts. The **location of ulcers**, whether gastric or duodenal, seems to have **no influence on fungal growth**. Females were more susceptible than males to develop Candida in gastric juice. In the short-term course with H2-R blockers fungal colonization of gastric juice **was** associated with ***delay* of the rate of ulcer healing**. Fungal detection in gastric juice was **not associated with mucosal invasion** by Candida since in none of the patients who had a biopsy for gastric ulcer was Candida detected by **histology**.

Boero M, Pera A, Andriulli A, Ponti V, Canepa G, Palmas F, Duglio A, Molinaro GC, Toselli M, Riccardino N. Candida overgrowth in gastric juice of peptic ulcer subjects on short- and long-term treatment with H2-receptor antagonists. Digestion. **1983**;28(3):158-63. [emphases added]

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PEPTIC ULCERS

Acid-reducing drugs can cause increased growth of microbes, including fungus, because of high gastric pH. Our purpose was to evaluate the occurrence of mycotic infection in patients with duodenal ulcer on anti-ulcer therapy and to compare the effects of cimetidine, famotidine, and omeprazole. Eighty patients with duodenal ulcer (62 males and 18 female patients, 16-65 years old) were evaluated for mycotic infection before and after 6 weeks of therapy (cimetidine, 20 patients; famotidine, 40 patients; omeprazole, 20 patients). Mycotic infection was diagnosed by endoscopic biopsy from the ulcer edge subjected to smear, culture, and histopathology and by endoscopic brush samples and gastric aspirate. On the basis of these studies, patients were categorized as having no fungal growth, saprophytic growth, or significant fungal growth. Thirty-five (43.8%) patients had evidence of fungus before ulcer therapy, and 16 of the 35 (20%) had significant fungal growth. The fungal isolation rate was higher in older patients (> and = 45 years of age) and in those with an ulcer size > and = 2 cm. While there was no significant increase in the total number of patients with evidence of fungus after therapy (n = 36), there was a **significant increase in those with significant growth** (n = 27, $p < 0.05$) compared with pretreatment status. We found that **posttreatment gastric pH of > and = 4 was associated with a higher fungal positivity rate (59.4%)** than pH values < 4 (32.4%, $p < 0.05$). However, neither the type of drug used nor the response in terms of ulcer healing correlated with the presence of fungus. Regardless of the type of drug used, **acid-reducing therapy is associated with increased significant fungal growth.**

Goenka MK, Kochhar R, Chakrabarti A, Kumar A, Gupta O, Talwar P, Mehta SK. Candida overgrowth after treatment of duodenal ulcer. A comparison of cimetidine, famotidine, and omeprazole. J Clin Gastroenterol. **1996** Jul;23(1):7-10.

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PEPTIC ULCERS

Proton pump inhibitors (PPIs) remain the superior choice worldwide in antisecretory therapy in the evidence-based treatment of upper gastrointestinal disorders including gastroesophageal reflux disease, erosive esophagitis, dyspepsia and peptic ulcer disease. **PPI overutilization** in ambulatory care settings is **often a result of failure to re-evaluate** the need for continuation of therapy, or insufficient use of on-demand and step-down therapy. Nonjudicious use of PPIs creates both preventable financial as well as medical concerns. **PPIs have been associated with an increased risk of vitamin and mineral deficiencies** impacting vitamin B_{12}, vitamin C, calcium, iron and magnesium metabolism. While these risks are considered to be relatively low in the general population, they may be notable in **elderly** and **malnourished** patients, as well as those on chronic hemodialysis and concomitant PPI therapy. No current evidence recommends routine screening or supplementation for these potential vitamin and mineral deficiencies in patients on either short- or long-term PPI therapy. **Reducing inappropriate prescribing of PPIs can minimize the potential risk of vitamin and mineral deficiencies**.

Joel J. Heidelbaugh. Proton pump inhibitors and risk of vitamin and mineral deficiency: evidence and clinical implications. Ther Adv Drug Saf. **2013** Jun; 4(3): 125–133. [emphases added]

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PEPTIC ULCERS

Proton pump inhibitors (PPI) are now one of the **most widely used** classes of drugs. PPIs have proven to have a very favorable safety profile and it is unusual for a patient to stop these drugs because of side effects. However, **increasing numbers of patients are chronically taking PPIs** for gastroesophageal reflux disease and a number of other common persistent conditions, therefore the long-term potential adverse effects are receiving increasing attention. One area that is receiving much attention and generally has been **poorly studied, is the long-term effects of chronic acid suppression on the absorption of vitamins and nutrients**.
The data reviewed here support the importance of long-term investigations of the **possible effects of chronic PPI treatment** on absorption of important nutrients including calcium, vitamin B12, iron and magnesium. In general, the studies in each of these areas have led to differing conclusions, but when examined systematically, a number of the studies are showing consistent results that support the conclusion that **long-term adverse effects on these processes can have important clinical implications**.
What are badly needed for each of these nutrients, as well as studies of bone fractures, are more prospective studies.

Tetsuhide Ito, MD, PhD and Robert T. Jensen, MD. Association of Long-term Proton Pump Inhibitor Therapy with Bone Fractures and effects on Absorption of Calcium, Vitamin B12, Iron, and Magnesium. Curr Gastroenterol Rep. **2010** Dec; 12(6): 448–457. [emphases added]

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PEPTIC ULCERS

In my professional opinion, these factors will emerge as increasingly important in our understanding of gastric acidity and acid-suppression therapies:

1 -- **Helicobacter pylori** (and perhaps other opportunistic enterobacterial infections, such as Serratia marcescens), especially undiagnosed and untreated (or insufficiently treated) will steadily **gain in prominence**, especially in acid-suppressed patients

2 -- **Clostridium difficile** and other **bacterial**, **parasitic**, and **fungal** enterocolic infections will be appreciated as **resulting directly from** acid-suppression therapy

3 -- **Fungal infections** and **The Yeast Syndrome** will only **slowly be appreciated** by the conventional medical community

4 -- **Fungal infections** and **The Yeast Syndrome**, especially their ready innoculation in acid-suppressed patients and their profound contribution to oro-esophageal, gastric, enteric, and colonic pathologies will only **slowly be appreciated** and treated

5 -- Critical contributions of **biofilms** lining the digestive and respiratory systems might be better understood, developing strategies to reduce the chronicity of gut pathologies and reduce their repetitive or chronic treatments

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PEPTIC ULCERS

My clinical experience for successful treatment over the past 32 years:

Comprehensive treatment for The Yeast Syndrome is ***the key*** to long term remission and should not be postponed when indicated.

Locally, basically the same as for GERD:

Mycelex [clotrimazole] oral troches 10 mg #100 refill once --

Sig: dissolve 1 in mouth qid, ac and hs

Carafate [sucralfate] suspension 480 ml refill once --

Sig: slowly swallow 2 tsp po ac (after Mycelex) and hs -- do not rinse

[or tablets 1 g #100 refill only if calls -- 1 po qid ac and hs with minimal liquid; if desired, dissolve in small volume water]

aloe vera gel/papaya juice -- equal volumes, sip continuously for discomfort

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PEPTIC ULCERS

A critical caveat:

A primary reason for the continuation or reinstitution of acid-suppression therapies is simply that the **patient suffers recurrent symptoms soon after stopping** the medication. Both doctor and patient conclude: "Uh-oh, we haven't done enough treatment. Let's do another course of the drug."

When gastric acid production is suppressed, the stomach has no need to secrete and maintain a **thick mucinous layer** (like **rubber cement**) to protect its own tissue from digestion by the acid puddle because the threat has been reduced or removed. Thinning of the protective layer is appropriate -- until acid-suppression medication is discontinued. Resumption of acid production (even rebound hypersecretion) leads to **acid attack on the stomach lining**, hence recurrent pain (wrongly) suggesting that more acid-reduction is needed.

In my experience, treating with **Mycelex** and **Carafate**, along with a comprehensive program for **The Yeast Syndrome**, is ***almost 100% successful***. The acid-suppressing drugs are slowly reduced and withdrawn *by the patient*, as his symptoms improve and he no longer needs them.

Ksiądzyna D, Szeląg A, Paradowski L. Overuse of proton pump inhibitors. Pol Arch Med Wewn. **2015**;125(4):289-98. [emphases added]

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PEPTIC ULCERS

TAKE-HOME MESSAGE:

Peptic ulcer disease, usually treated by chronic acid-reducing medications that can **impair** digestion of **proteins** and preparation of **minerals** and **vitamins** for absorption as well compromise protection against entry of pathogens — hastening development of debilitating chronic diseases — can be **resolved completely in most** patients by proper treatment of aggravating **yeast *and/or* bacterial** species (and parasites?) and defining steps needed for restoration of more normal digestive processes.

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IRRITABLE BOWEL

The **state of the health of the individual** is equivalent to the state to the health of the colon.

-- Woody Harrelson

http://www.brainyquote.com/quotes/quotes/w/woodyharre555536.html?src=t_colon

"This study helps underscore the **importance of the microbiome in maintaining health** and demonstrates that good bacteria play an integral role in immune defenses against disease," says E. Albert Reece, M.D., Ph.D., M.B.A., Vice President for Medical Affairs at the University of Maryland and the John Z. and Akiko K. Bowers Distinguished Professor and Dean of the University of Maryland School of Medicine. "These findings also provide a potential **therapeutic model for other diseases that have been linked to changes in the human intestinal microbiota, such as obesity and diabetes**."

Restoring helpful bacteria of the gut and intestines may treat patients with RCDI, find scientists. News Medical: Life Sciences and Medicine. November 27, **2013**; http://www.news-medical.net/news/20131127/Restoring-helpful-bacteria-of-the-gut-and-intestines-may-treat-patients-with-RCDI-find-scientists.aspx.

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IRRITABLE BOWEL

Irritable bowel syndrome (IBS) is a common disorder that affects the large intestine (colon). Irritable bowel syndrome commonly causes cramping, abdominal pain, bloating, gas, diarrhea and constipation. IBS is a **chronic *condition*** that you will need to **manage long term**.

http://www.mayoclinic.org/diseases-conditions/irritable-bowel-syndrome/basics/definition/con-20024578

Inflammatory bowel disease (IBD) involves **chronic *inflammation*** of all or part of your digestive tract. IBD primarily includes ulcerative colitis and Crohn's disease. Both usually involve severe diarrhea, pain, fatigue and weight loss. IBD can be debilitating and sometimes leads to life-threatening complications.

Ulcerative colitis is an inflammatory bowel disease that causes long-lasting **inflammation and sores (ulcers)** in the innermost lining of your large intestine **(colon) and rectum**.

Crohn's disease is an IBD that cause inflammation of the lining of your digestive tract. In Crohn's disease, **inflammation often spreads deep** into affected tissues. The inflammation can involve **different areas** of the digestive tract — the large intestine, small intestine or both.

http://www.mayoclinic.org/diseases-conditions/inflammatory-bowel-disease/basics/definition/con-20034908

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IRRITABLE BOWEL

They haven't a clue!

There is no cure for *IBS*. Much about the condition remains unknown or poorly understood; however, dietary changes, drugs, and **psychological treatment** are often able to eliminate or substantially reduce its symptoms. ... IBS is the name people use today for a condition that was once called—among other things—**colitis**, **mucous colitis**, **spastic colon**, **nervous colon**, **spastic bowel**, and **functional bowel disorder**. ... No one knows for sure how many Americans suffer from IBS. Surveys indicate a range of 10-20%, with **perhaps as many as 30% of Americans experiencing IBS at some point in their lives**. ... **Women** with IBS outnumber men by two to one, for reasons that are not yet understood. IBS is **responsible for more time lost from work and school than any medical problem other than the common cold**. It accounts for a substantial proportion of the patients seen by specialists in diseases of the digestive system (gastroenterologists). Yet only half—possibly as few as **15%—of IBS sufferers ever consult a doctor**. ... Dietary changes, sometimes supplemented by drugs or **psychotherapy**, are considered the key to successful treatment.

http://medical-dictionary.thefreedictionary.com/irritable+bowel+syndrome [emphases added]

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IRRITABLE BOWEL

DANCING AROUND IT

There are diseases where **food is the primary factor in the pathogenesis**, such as food allergy and gluten-sensitive enteropathy. Both diseases are mediated by different types of immune reactions. Furthermore, **allergic reactions may play a role** in eosinophilic esophagitis and eosinophilic gastroenteritis. In chronic inflammatory bowel disease food influences the disease by the modulation of various factors such as intestinal bacterial flora or the inflammatory cascade. **Yeast antigens**, especially in people with a defect in the innate immune system, such as MBL-deficiency [mannose binding lectin protein deficiency, a complement component] may lead to an enhanced immune reactivity to mannan antigens of yeast. This **may have some significance in the perpetuation of intestinal inflammatory processes** in patients with Crohn's disease.

Seibold F. Food-induced immune responses as origin of bowel disease? Digestion. **2005**;71(4):251-60. [emphases added]

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IRRITABLE BOWEL

Yeasts, mainly Candida species, are present **in the gut of about 70%** of healthy adults.

Mucocutaneous Candida infections are due either to **impaired host defenses** or to **altered gene expression** in formerly commensal strains.

The expression of virulence factors enables yeasts to **form biofilms**, **destroy tissues**, and **escape the immunological attacks** of the host. **Yeast infections of the intestinal mucosa are of uncertain clinical significance**, and their ***possible connection to irritable bowel*** syndrome, while plausible, remains unproved.

Yeast colonization can trigger allergic reactions. Mucosal yeast infections are treated with topically active polyene antimycotic drugs [Nystatin, Amphotericin B]. The adjuvant administration of **probiotics** is justified on the basis of positive results from controlled clinical trials.

The *eradication* of intestinal yeasts is *advised only for certain clearly defined* indications.

Schulze J, Sonnenborn U. Yeasts in the gut: from commensals to infectious agents. Dtsch Arztebl Int. **2009** Dec; 106(51-52):837-42. [emphases added]

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IRRITABLE BOWEL

DANCING AROUND IT

Irritable bowel syndrome (IBS) is characterized by symptoms of abdominal pain and altered bowel habits. This common disorder is **managed by varying clinical styles as no dominant therapeutic strategy has emerged**. The pathophysiology of IBS remains **unknown**, but several lines of evidence link this disorder with the gut microbiota. Although controversy exists, **gut microbiota is likely contributing to symptoms** of IBS, at least in some patients, through an altered **fermentation** process, an impaired intestinal **barrier** function, a harmful **modulation of enteric sensorimotor** function, a promotion of low-grade **inflammation** without tissue damage, and a harmful **modulation of the brain-gut axis**. **Probiotic therapy** has a modest effect on IBS symptomatic relief, but the **actual evidence is not strong enough** to support a general recommendation of use.

Tojo González R, Suarez Gonzalez A, Rúas Madiedo P, Mancebo Mata A, Pipa Muñiz M, Barreiro Alonso E, Roman Llorente FJ, Moro Villar MC, Arce González MM, Villegas Diaz MF, Mosquera Sierra E, Ruiz Ruiz M. [Irritable Bowel Syndrome; gut microbiota and probiotic therapy]. Nutr Hosp. **2015** Feb 7;31 Suppl 1:83-8. [emphases added]

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IRRITABLE BOWEL

DANCING AROUND IT

IBS is a condition for which investigators have **long been in search of plausible underlying pathogeneses** and it is inevitable that altered composition or function of the gut microbiota will be considered as a potential aetiological factor in at least a subset of patients with IBS. This Review describes the **evidence implicating the gut microbiota** in not only the expression of the **intestinal manifestations** of IBS, but **also** the **psychiatric morbidity** that coexists in up to **80% of patients** with IBS. … The gut microbiota is subject to influences from a diverse range of factors including **diet**, **antibiotic** usage, **infection** and **stress**. These factors have previously been implicated in the pathophysiology of IBS and further prompt consideration of a role for the gut microbiota in IBS.

Collins SM. A role for the gut microbiota in IBS. Nat Rev Gastroenterol Hepatol. **2014** Aug;11(8):497-505. [emphases added]

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IRRITABLE BOWEL

STILL ENJOYING *THE DANCE*

A major functional **dysbiosis** was observed in **constipated-irritable bowel** syndrome gut microbiota, reflecting altered intestinal fermentation. **Sulphate-reducing population increased** in the gut of C-IBS and were accompanied by alterations in other microbial groups. This **could be** responsible for changes in the metabolic output and enhancement in toxic sulphide production which could in turn **influence gut physiology** and contribute to IBS pathogenesis.

Chassard C, Dapoigny M, Scott KP, Crouzet L, Del'homme C, Marquet P, Martin JC, Pickering G, Ardid D, Eschalier A, Dubray C, Flint HJ, Bernalier-Donadille A. Functional dysbiosis within the gut microbiota of patients with constipated-irritable bowel syndrome. Aliment Pharmacol Ther. 2012 Apr;35(7):828-38. [emphases added]

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IRRITABLE BOWEL

The gut flora carries out important functions for human health, … and an alteration of any of them, due to a condition of **dysbiosis, can lead to relevant pathological implications**. Commensal bacteria in the gut are essential for the preservation of the integrity of the mucosal barrier function and an alteration … has been implicated in the pathophysiologic process of different diseases. The gut microflora plays a role in modulating the intestinal immune system … The enteric flora represents a **potent bioreactor** which controls several metabolic functions, even if most of them are still unknown. ... This overview focuses briefly on the physiological role of the gut microbiota in maintaining a healthy state and the potential role played by **disturbances of both the function and composition of the gut microbiota** in determining important pathological conditions, such as ***irritable*** **bowel syndrome**, ***inflammatory*** **bowel** disease, **metabolic syndrome**, **obesity**, and **cancer**.

Festi D, Schiumerini R, Birtolo C, Marzi L, Montrone L, Scaioli E, Di Biase AR, Colecchia A. Gut microbiota and its pathophysiology in disease paradigms. Dig Dis. **2011**;29(6):518-24. [emphases added]

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IRRITABLE BOWEL

WILL THEY *EVER* SEE THE LIGHT?

Symptoms unrelated to the intestine (extraintestinal symptoms) are common in patients with IBS. These may include **headache**, **sleep** disturbances, post-traumatic **stress** disorder, **temporomandibular** joint disorder, **sicca** syndrome, back/pelvic **pain**, **myalgias**, **back pain**, and chronic **pelvic** pain. **Fibromyalgia** and **interstitial cystitis** are also frequently encountered in patients with IBS. In fact, Fibromyalgia occurs in up to 33% of patients with IBS and almost half of patients with fibromyalgia also have IBS.

http://www.hopkinsmedicine.org/gastroenterology_hepatology/_pdfs/small_large_intestine/irritable_bowel_byndrome_IBS.pdf. [emphases added]

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IRRITABLE BOWEL

Many patients with irritable bowel syndrome (IBS) are disillusioned by the lack of efficacy of treatments and suffer from numerous symptoms not covered by the Rome criteria for IBS, as the current empirical treatment regimens fail to address these persistent debilitating 'IBS associated symptoms'. These **symptoms are similar to other symptom complexes like chronic fatigue and the so-called 'candida syndrome'**, and many seek help from alternative medicine. The possible role of Candida and yeasts in non-immune compromised individuals is disputed and is the subject of this review. Even if the involvement of yeasts in the aetiology of IBS still remains unclear, there is **increasing evidence for yeasts being able to cause IBS-**symptoms in sensitized patients via Candida products, antigens and cross-antigens. But ***more research is needed*** before antifungal treatment can be recommended as a first line treatment for IBS.

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IRRITABLE BOWEL

TREATMENT IS USUALLY SIMPLE:

Comprehensive care for

THE YEAST SYNDROME

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IRRITABLE BOWEL

TAKE-HOME MESSAGE:

Irritable bowel syndrome, initially mistaken for inflammatory bowel disease and generally leading to unnecessary and expensive gastroenterologic testing -- then usually **inappropriately treated** by prescription medications aimed at **symptom relief** -- often can be **resolved by replacement of aggravating nutrient depletion** related to enterocolic overgrowth of yeast species, often *Candida albicans*.

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TAKE-HOME MESSAGE

Frustrating and common gastrointestinal conditions often can be **resolved easily, safely, and completely** by evidence-based clinical diagnosis and treatment leading to **reduction of abnormal overgrowth of enterocolic yeast** organisms and **replacement of nutrient depletion** resulting therefrom.

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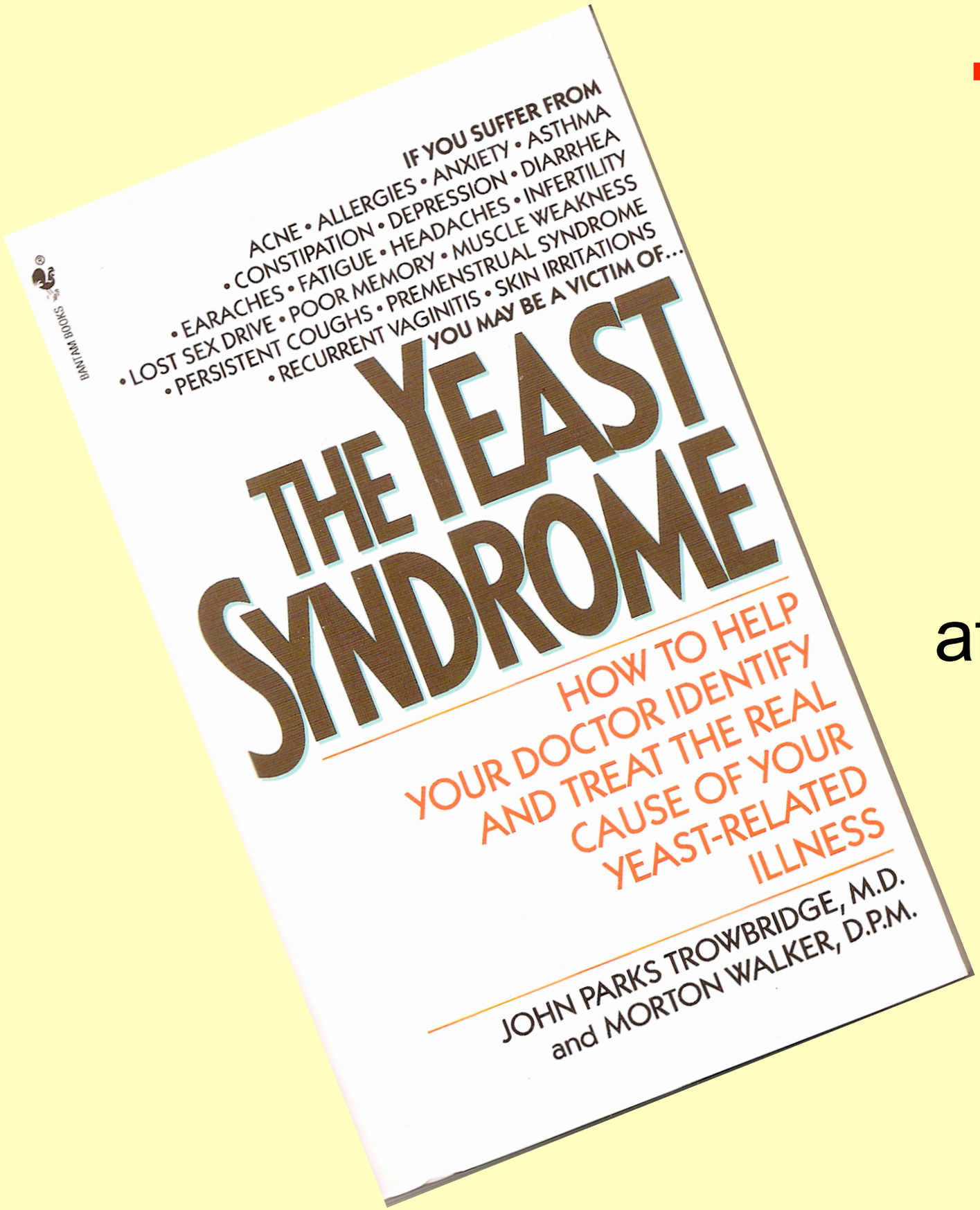

TYS

THE YEAST *SYNDROME*

affects 30 to 50%

of your patients

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