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BOWEL NO 6 - FUNCTIONAL GASTRO-INTESTINAL DISORDERS V DYSFUNCTIONAL HUMAN BEINGS; MICROMANAGEMENT OF MILIEU INTERIOR PART III – *SUBLATA CAUSA, TOLLITUR EFFECTUS* (REMOVING CAUSE, REMOVES EFFECT)

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ABSTRACT

Functional gastro-intestinal disorders (FGIDs) portray Westernisation of life. Issues like gut motility, microbiota, visceral sensitivity, mucosal immune system, role of nervous systems, pathophysiological role of microbiome (probiotics) and prebiotics (carbohydrates), laws of physics impacting gastrointestinal physiology, role of physiotherapy and fasting in gut health; are subtle causes needing modification and rectification.

KEYWORDS: Functional Gastro Intestinal Disorders – FGIDs, Disorders of Gut-Brain Interactions – DGBIs, Functional Dyspepsia – FD, Irritable Bowel Syndrome – IBS; Diverticular Disease – DD; Symptomatic Uncomplicated Diverticular Disease – SUDD.

(1) INTRODUCTION

Functional gastro-intestinal disorders (FGIDs) are chronic malady *apparently* drenched in Western lifestyle explained by Rome Group alongwith '*gas and bloating*' nonissues; due to diet mostly of animal protein, high in sugars and saturated fats, devoid of natural fiber, leads to gut microbial dysbiosis. Lack of gut synbiotics in nutrition and metabolism causes irritable bowel syndrome (IBD), diverticular disease (DD); even inflammatory bowel disease (IBD). Role of multivitamins, Laws of Physics regulating gastrointestinal (GI) physiology, role of physiotherapy in FGIDs and fasting in gut health are addressed. Surgery in FGIDs is limited to serious complications only.

(2) Preliminary Prologue: Humans are only 'Dysfunctional Human Beings (DHBs)' contracting FGIDs. Food must not be eaten for tongue delight; with its caloric value to supply micronutrients and energy but to keep gut healthy alongwith good *health related quality of life* (HRQOL), (Teoli and Bhardwaj[1]). This entails watching of *milieu intérieur* and intake of proper fiber (*prebiotics)* for trillions of bowel microbiomes (probiotics); effectors of our wholesome health including gut motility, mucosal immune system and defecation. Burkitt[2] drew attention in 1973, working in Africa. Had Burkitt's advice been heeded perhaps there might not have been FGIDs phenotypes today; remove the cause, to remove the effect' (*sublata causa, tollitur effectus).* Interestingly Drossman[3] asserts, "Psychosocial processes were considered important but only as secondary phenomena, because if cause of a disease could be found and treated, then certainly any psychosocial difficulties would disappear. Unquote." This prudent presage by leading authorities nullify "Brain-Gut Axis" hypothesis in FGIDs.

(3) Westernisation and FGIDs: Rome Group asserts that they have tried to move from Western Society based FGIDs criteria to more inclusive approach with a focus on multicultural orientation but a lot more is needed (Schmulson and Drossman[4]). It is good start but a lot of pointed approach is needed to find a definite solution by studying Eastern sociocultural lifestyle for FGIDs. They assert, Rome approach was based on Western knowledge to understand patients' symptoms, which has limitations for other countries' cultures. This led to inclusion of a chapter devoted entirely on multi-cultural information to address the global perspective on these disorders. Most characteristic symptom report is *bloating*, a term confined primarily to English language but absent in Spanish or Italian. Also, differentiation of bloating from postprandial fullness imposes a linguistic and cultural issue. In China, postprandial fullness is limited to epigastrium while bloating refers to a *sensation of gas* that is present in most, if not entirely, all over abdomen... Attempt is praiseworthy but large part of South-East Asia with Indian Peninsula remains out-with their exclusive lifestyle and culinary mores.

(4) Is Western Lifestyle the Deadbeat: Comparing **"***Western diet"* that mainly consists of animal protein, high in sugar and saturated fat, to an *agrarian diet* that *mainly* consists of low animal protein and saturated fat with high carbohydrates and simple sugars; composition of intestinal microbiota showed drastic differences. When analyzing microbiota of people consuming Western diet, there was an increase in *Firmicutes* and *Proteobacteria* besides some *Bacteroides* but people consuming agrarian diet had increased amounts of *Actinobacteria*, mostly *Prevotella*. In people consuming agrarian diet, there was an increase in SCFA levels and reduced SCFAs in people consuming Western diet (DeGruttola *et al*[5]). Many studies have found change in gut microbiota composition between countries, which is mainly related to local eating habits. Therefore, in different populations, elemental composition of microbiota in healthy individuals considered to be norm will vary (Zapala et al[6]).

(5) Visceral hypersensitivity: Visceral hyper/sensitivity expressed as discomfort or pain, vague, mostly in midline or locally if severe. By contrast *somatic pain* is sharply localised and relates to skin, muscle, bones and outer body parts. Moshiree *et al*[7] define Visceral hyperalgesia as reduced pain threshold and/or an exaggerated response to a painful stimulus. Two types of hyperalgesia include, *primary* (at site of tissue injury/inflammation) and *secondary*, viz, *allodynia*, means prior non-painful nociceptive stimuli now evoke pain. Precise etiopathology is not known for these phenotypes. Camilleri *et al*[8] deliberated, "Visceral hypersensitivity is currently the holy grail (sic)....it is widely regarded as reason for development of functional gastrointestinal diseases, including functional dyspepsia and irritable bowel syndrome. Unquote" Author thinks, visceral pain or discomfort is secondary clinical phenotype, refers to ill-understood biophysiopathological traits in DHBs. Maryam *et al*[9] feel, organic IBD is also sister disorder or coexisting with FGIDs. Šojat *et al*[10] claim, "Up to today, inflammatory bowel diseases (IBDs), ulcerative colitis (UC), and Crohn's disease (CD), which are classified within autoinflammatory diseases; have been considered the only chronic inflammatory diseases that affect gastrointestinal tract. The increasing evidence suggests that development of these diseases may be triggered by a disturbance in balance between gut commensal microflora and mucosal immune system. Unquote" Noddin *et al*[11] stated, "Many patients with IBS have dyspepsia; likewise, many patients with dyspepsia also have overlapping symptoms of IBS. These 2 groups of patients are similar in that symptoms are typically chronic in nature, may wax and wane, are aggravated by psychosocial stressors and are often *worsened by meals*. Unquote" This statement often *worsened by food* must be qualified for type of foods to remove confusion. Li *et al*[12] mandated, "Each part of GIT can be considered a unique physical environment with characteristic pH values, motility, digestive juices and microbiomes. For example, stomach has low pH, small intestine has optimal drug absorption, and colon is rich in microbiota... Lifestyle and diet affect occurrence and development of gastrointestinal diseases. Unquote." Amazingly, "Why do patients lose appetite or develop pyoderma gangrenosum in skin and subcutaneous tissues in cases of IBD (Marzano *et al*[13])? In another study, Weizman *et al*[14] described, "Pyoderma gangrenosum, neutrophilic dermatosis affecting skin, is one of the more common extra-intestinal manifestations of inflammatory bowel disease (IBD). Unquote." Li *et al*[15] found, "Research highlights that diet, lifestyle, environmental exposures, genetic predisposition, and gut microbiota significantly influence IBD development. In recent years, dietary trace elements, such as vitamins C, D, and K have garnered increasing research interest due to commonly observed nutritional deficiency in IBD patients. These vitamins are considered to be beneficial to health due to their antioxidant, immune-regulatory, and anti-inflammatory effects, and their deficiency may be related to various inflammatory diseases, including IBD. Unquote." Such clinical mishmash is baffling.

(6) Intestinal Microbiota (probiotics) and fibers (prebiotics)-Synbiotics: Goal of synbiotic is to improve survival, growth and metabolic activity of probiotics. Here are titbits from Roberfroid *et al*[16], "(1)... original idea of prebiotic concept, defined as: 'The selective stimulation of growth and/or activity(ies) of one or a limited number of microbial genus(era)/species in gut microbiota that confer(s) health benefits to the host.' (2) Changes in gut microbiota composition are classically considered as one of many factors involved in pathogenesis of either inflammatory bowel disease or irritable bowel syndrome. (3) As a result of research activity that followed publication of prebiotic concept 15 years ago, it has become clear that products that cause selective modification in gut microbiota's composition

and/or activity(ies) and thus strengthen normobiosis could either induce beneficial physiological effects in colon and also in extra-intestinal compartments or contribute towards reducing the risk of dysbiosis and associated intestinal and systemic pathologies. Unquote." GOS (galacto-oligosaccharides) and FOS (fructo-oligosaccharides) are vital prebiotics that support intestinal flora (probiotics), crucial for gut and overall health. Probiotics or Microbiota are about 100 trillion in colon; surpass our body cells by 3 to 4 times. Thursby and Juge[17] update, "However, a recently revised estimate has suggested that the ratio of human::bacterial cells is actually closer to 1:1. As a result of vast number of bacterial cells in body, host and microorganisms inhabiting it are often referred to as a 'superorganism' Unquote." Their function is biophysiologically useful for our health. *Dysbiosis* or *dysbacteriosis* causes change in microbiota from healthy pattern (physiological) to a disease one (pathological). *Microbiota diversity* is a measure of how many different species and how evenly distributed they are in community. *Lower diversity is considered a marker of dysbiosis (microbial imbalance)* in gut and is found in autoimmune diseases, obesity, cardiometabolic conditions and elderly people. *Xenobiotics* are chemical products being foreign to an organism and elicit toxic (oxidant) reactions through mechanisms involving 'biologically reactive chemical species'. Present therapy of probiotics has lot of difficulties in clinical practice to find *compatible strain* to suit individuals besides its therapeutic application. Human genome consists of ±23,000 genes and microbiome has over three million genes producing thousands of metabolites replacing many functions of host affecting fitness, phenotype and health.

(7) Synbiotics Physiopathology consideration: Carbohydrates meet criteria of prebiotics; few compounds not classified as carbohydrates may also act as prebiotics, such as cocoa-derived flavanols poorly absorbed in intestine, promotes bacterial growth and exert anti-inflammatory effects contributing to overall health. Consuming flavanol-rich foods may enhance gut microbial diversity and positively influence immune function and metabolic markers and stimulate lactic acid bacteria. Slavin[18] thinks dietary components that stimulate fermentation leads to an increase in bacterial mass and consequently fecal mass and thus have stool bulking effect. It is estimated that ± 30 g of bacteria are produced per ± 100 g of carbohydrates fermented. Colonic epithelial cells specially use *butyrate* as energy source, even when substrates such as glucose and glutamine are available. Butyrate is key nutrient towards metabolic activity and growth of colonocytes and may function as a primary protective factor against colonic disorders. Together with gut immune system, colonic and mucosal microflora adds significantly to barrier that prevents pathogenic bacteria from invading gastrointestinal tract. Unquote." Stressing, Holscher[19] yelled on microbiota, "The human gastrointestinal microbiota-one of the most densely populated microbial communities on earth contain highly diverse microbial communities that provide metabolic, immunologic and protective functions that play a crucial role in human health. Unquote" There are several divisions of bacteria found in normal intestinal microflora dominant being *Bacteroidetes* and *Firmicutes*. Gut houses bacteria that are protective (commensals) and harmful (pathogenic) to host. Microbiota protects host from foreign microbes known as "*barrier effect* or *colonization resistance*".

(8) Pathophysiological Role of Microbiota: Microbiotas possess various metabolic functions, ie, breaking down complex carbohydrates and generating short-chain fatty acids (SCFAs) to benefit host. Surprisingly, gut microbiota are also capable of interacting with distant organs, such as brain (*psychobiotics*) which has led to studies of gut microbiota on mental disorders like autism and Alzheimer's. Dysbiosis has been associated with diseases such as IBD, Obesity, Type 1 and 2 Diabetes, allergic disorders and certain GI cancers. DeGruttola *et al*[5] explain, "The three types of dysbiosis are: (1) Loss of beneficial bacteria (2) Overgrowth of potentially pathogenic bacteria (3) Loss of overall bacterial diversity. In most cases these types of dysbioses occur at the same time. Unquote." *A decrease in fiber is associated with a higher risk of advanced CRC* (Colo-rectal carcinoma). Patients with advanced CRC were shown to have decreased butyrate-producing bacteria in gut microbiota, which causes less SCFA fermentation in gut, which may lead to formation of advanced CRC. In contrast, increased fiber intake has shown to have an increase in butyrate-producing bacteria and an overall balanced gut microbiota as compared to individuals with decreased fiber, indicating fiber's important role in decreasing CRC risk as well as its role in maintaining gut microbial homeostasis. Butyrate is vital SCFAs, showing anti-tumorigenic effect by inhibiting proliferation of cancer cells, restricting tumor angiogenesis and inducing apoptosis. Diets high in whole grains and fiber may influence gut microbiota at phylum level, specifically causing a decrease in *Bacteroides* when consumed long-term and increasing *Bfidobacteria* and *Roseburia*, both are butyrate-producing bacteria suggest to influence immunity and inflammatory functions in animal models. In studies probing roles of high-protein/low-carb (HPLC) versus high-protein/moderate-carb (HPMC) diets, presence of fiber and non-digestible carbohydrates contribute to prevention of CRC. This is believed to be due to increased SCFA fermentation that affect colon by promoting mucus secretion and modulating inflammatory immune response. An increase in fruit and vegetable fiber consumption is shown to be chemoprotective and is associated with increased gut microbial diversity and decreased CRC risk. People with high vegetable diet are shown to be less likely to develop IBD and CRC. Along with decreasing cancer risk, polyphenols present in many foods, are also associated with lowering blood pressure, cholesterol levels, lower

risk for cardiovascular disorders, stroke, and aortic aneurysm (DeGruttola *et al*[5]).

(9) Role of Gut microbes (probiotics) in nutrition and metabolism: This aspect of microbiota involved in nutrition and metabolism has become a lead topic of research and development and some of the authors involved in this research are Soto-Martin et al[20]; Magnúsdóttir *et al*[21]; Morowitz et al.[22] Bacteria able to produce growth factors are called *prototrophs*, whereas bacteria cannot produce them are *auxotrophs* and need to obtain them from external sources including diet or cross-feeding by other bacteria (Soto-Martin *et al*[20]). B-vitamin biosynthesis pathways seem prevalent in human gut microbiota genomes and gut bacteria have capacity to produce sufficient B-vitamins for daily human requirements. Magnúsdóttir *et al*[21] predicted B-vitamin biosynthesis by 256 human gut microorganisms based on their genome annotations alone. Morowitz *et al*[22] explain that drugs which block acid secretion and affect GI motility can indirectly alter microbiota. Physiobiochemical function in humans have shown that gut microbiota are able to synthesize vitamin K, most of the water-soluble B vitamins including amino acids are produced by 40–65% of 256 human gut microbes. In contrast to dietary vitamins, which are adsorbed in small intestine; predominant uptake of microbially produced vitamins occurs in colon. The genus *Bifidobacterium* currently encompasses 39 species; represent key components of human gut microbiota. Many reports have pointed importance of bifidobacteria in regulating intestinal homeostasis, modulating local and systemic immune responses and protecting against inflammatory diseases and infections. Animals, plants and fungi are incapable of cobalamin production and are only vitamin exclusively produced by microorganisms notably by anaerobes. Daily need for vitamin K is fulfilled by dietary phylloquinone that is present in plants and to an extent by bacterially produced compounds known as menaquinones synthesized in human gut. B-vitamins are necessary cofactors for many aspects of human metabolism including fat and carbohydrate metabolism and DNA synthesis. Humans are not capable of producing B-vitamins in sufficient amounts; thus they must obtain such vitamins either from diet or by gut microbiota. Current studies are clarifying important role of microbiota in carbohydrate metabolism and providing new evidence that highlights role of bacteria in protein and lipid homeostasis. Researches from past decade have finally proved that host-microbe relationship in humans is far more complex than earlier appreciated. Superb reviews are written on role of probiotics in health and disease (Liu *et al*[23], Amara and Shibl[24], Sarita *et al*[25]).

(10) IBS and IBD are closely faceted: There are lot of studies which have linked IBS and IBD, nay even two phases of same etiopathological phenotype. Walker *et al*[26] stated, "Inflammatory bowel disease is a chronic inflammatory condition of gastrointestinal tract. It comprises two disorders: Crohn's disease (CD) and ulcerative colitis (UC). There can be clinical similarities between presentations of IBD and FGIDs. Both IBD and FGID have similar bowel symptoms that occur with variable severity and both conditions follow chronic relapsing remitting courses. Unquote". They[26] continued, "It raises the question as to whether the strong correlation in our patients with IBD and FGIDs is a result of direct causation alone, or in fact down to shared predisposing factors... It is likely therefore that this distinction is purely arbitrary and more to do with expediency for academic community rather than addressing real issues facing people and practitioners. Unquote". This is testified by Vasant and Ford[27], "Indeed, recent data have shown that there is often a poor correlation between symptoms and mucosal inflammation in IBD. Although potential for co-existence of "functional" gastrointestinal symptoms in a proportion of patients with quiescent IBD was first described over 30 years ago, this group of patients has received minimal attention in medical literature until relatively recently. Unquote". This is cryptic view on IBS and IBD, "Are/could they be two faces/stages of same phenotype"? Author has few cases labelled UC elsewhere for last couple of decades treated as IBS successfully leading active life.

(11) Dietary factors, multivitamins, microbiome and IBD: Li *et al*[28] highlights role of various vitamins especially vitamin D and others: (1) In recent years, dietary trace elements, such as vitamins C, D, and K have garnered increasing research interest due to commonly observed nutritional deficiency in IBD patients. (2) Meanwhile, we need more evidence, to test therapeutic effects of vitamin D in prevention and treatment of IBD. Furthermore, future studies should investigate associations of vitamin C, vitamin K, and other dietary trace elements with IBD risk. Unquote." In a review, Reich *et al*[29] reported on Vitamin D deficiency in patients of IBD. Patients with IBD are at risk of low bone density and increased fractures due to low vitamin D levels, long standing disease and frequent steroid exposures; it is well known that adding vitamin D in this cohort is important. There is role of vitamin D in fortifying *innate immunity* by acting as an immunomodulator and reducing inflammation in IBD. Active form of vitamin D acts on T cells to promote T helper responses; suppresses dendritic cell inflammatory activity; induces antibacterial activity; and regulates cytokine production in favor of an anti-inflammatory response. Vitamin D is also believed to be a factor in regulating gut bacterial homeostasis.

Vemulapalli and Thomas[30] also expressed that Vitamin D influences gastrointestinal homeostasis, regulate microbial diversity and modulate immune responses. Interestingly diet, nutrition and physical activity is also linked to cancer, said Clinton *et al*.[31] In yet another work, Aggeletopoulou *et al*[32] opined, "Autoimmune diseases and IBD in particular, show a predisposition to vitamin D deficiency, leading to alterations in

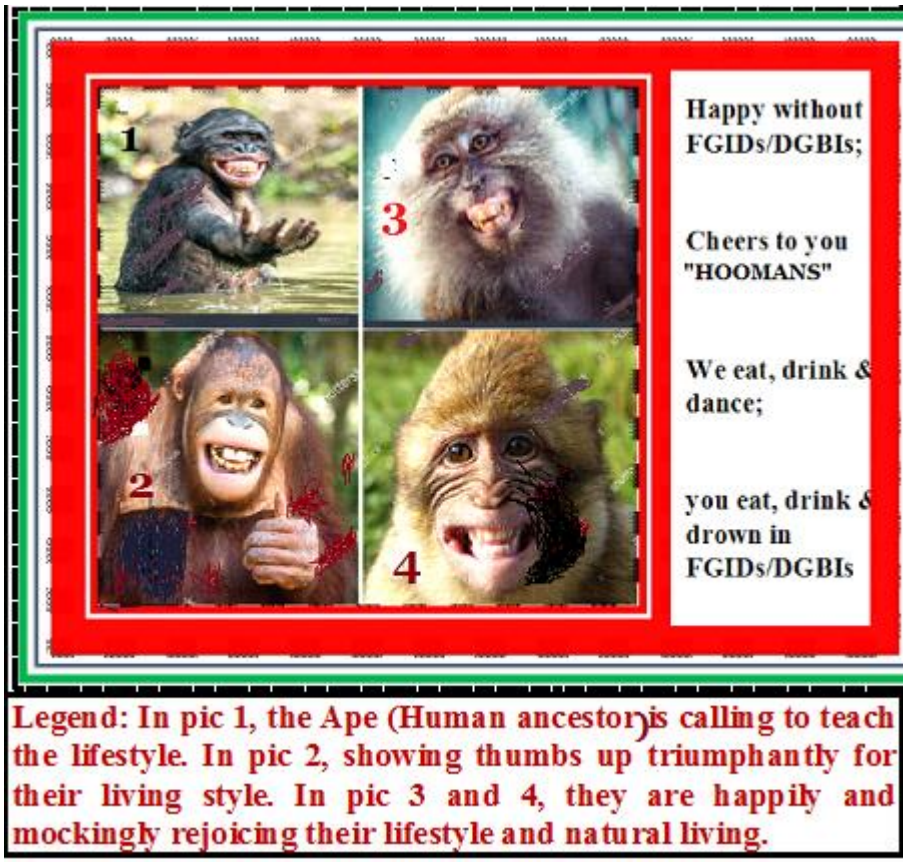
microbiome and disruption of intestinal epithelial barrier integrity. This review examines the role of gut microbiome in IBD and discusses how vitamin D–vitamin D receptor (VDR)–associated molecular signaling pathways contribute to development and progression of IBD through their effects on gut barrier function, microbial community and immune system function. Unquote." Whole grains are rich in phenolic compounds, most abundant being *ferulic acid* (FA). Fiber in whole grain foods delivers FA to gut, where they are slowly and continuously released by gut microbiota, which also convert FA into circulating dihydroferulic acid (DHFA). FA and DHFA have been considered to be protective against neoplastic changes in colonic epithelial cells and an increase in immune response and have been shown to possibly decrease the risk of obesity-related diseases when whole grains are consumed long-term. Alongwith these acids, whole grain consumption have shown to decrease TNFα (tumour necrosis factor-alpha) production besides increased Bacteroides and Lactobacillus, help subclinical inflammation.

(12) Laws of Physics and GI Physiology: Basic mechanisms of living and nonliving things in nature are guided by laws of pure sciences, ie, Physics, Chemistry and Mathematics (Srivastava *et al*[33]). Laws of physics in gastroenterology physiopathology make important contribution in FGIDs phenotypes in many aspects. Cecum, because of its larger diameter compared to rest of the bowel, requires least amount of pressure to distend according to *law of Laplace*. In ordinary situations, cecal diameter is less than 9 cm. Ischemia risk increases after 10 cm for perforation if diameter is greater than 12 cm for longer than six days. It is not only diameter and duration of distension but rate of acceleration of distension also increase the risk of perforation. *Law of Laplace* states, "Larger the vessel radius, larger the wall tension required to withstand given internal fluid pressure." Wall tension is the force on container wall that resists the force trying to expand it.

(13) Role of Physiotherapy and FGIDs: Physical exercise of all kinds should be encouraged as it is shown to improve IBS symptoms, particularly constipation. It also improves fatigue, depression, anxiety, and HRQOL in IBS patients. In IBD, recreational exercise is shown to reduce fatigue. Although there are no studies that have specifically investigated the effects of exercise in FGIDs with IBD, it is likely that exercise in presence of both disorders have beneficial effect, save in acute stage. It is well known that physical fitness needs regular physical exercises and our GIT being a smooth muscle organ, also needs energization by regular physical exercises. In this context, 'Yoga Āsan' plays important role. Wilderman and Sinha[34] state, "Yoga is a mind, body, and spiritual practice that originated nearly ±5,000 years ago in India, though precise chronology is debatable. It is known to relieve stress, enhance athletic performance, increase strength, flexibility as well as assist in achieving inner peace, focus and improved mental wellness and self-confidence. In recent years, practice of yoga has grown exponentially in the United States, with about 37 million practitioners in 2016. Unquote" In a project, Castaneda[35] stated (edited by Author for practicality without diluting its essence): (1) Yoga knows no creed. Though Yoga began in East and is considered to be founded by Eastern religions like Hinduism, Buddhism and Jainism, Yoga doesn't ask you to worship anything or anyone. I am not required to believe in Shakti or Buddha to know my body, meditate and open my mind to the world. That's the beauty of Yoga practice, it truly is for everyone. (2) It is in Pantajali's Yoga Sutra that *Ashtanga Yoga*, the most widespread form of yoga in America, is defined: *Ashtanga*, meaning "eight (astha) limbs (anga)" is composed of eight *guidelines* or steps to live a purposeful life. The limbs serve as "prescription for moral and ethical conduct and self-discipline; they direct attention toward one's health; and help acknowledge spiritual aspects of nature." (3) Starting with, *Yama*, the first limb; and *Niyama* is second limb; *Asanas*, or postures make third limb; *Pranayama*, or breath control, is fourth limb of Ashtanga Yoga; fifth limb is *Pratyahara*; this limb is one of withdrawal (unfortunately there is no exact English translation of this limb but it needs constant practice to withdraw 'oneself from oneself and surroundings' to concentrate on Supreme Lord whom one trusts; better on 'Formless'!); *Dharana* is sixth limb –mind is a busy place, learning how to focus our attention on a single 'formless' point is sometimes hardest part of Yoga practice; seventh limb of Ashtanga is *Dhyana*, or meditation. In Ashtanga, each limb builds on the next and by the time practitioner addresses *Dhyana* s/he should draw on previous experiences perfecting initial limbs. At Dhyana stage, mind has been calmed, external distractions eliminated, breath controlled and body prepared through Āsan. Eighth and final limb of Ashtānga is *Samadhi*; state of *ecstasy* is not correct translation that is a state of profound mental stillness and focus that can lead to a 'deep sense of connection with oneself, and potentially with something larger than oneself'. This is a state of complete mental stillness and mind becomes fully silent. To describe this in words is like detailing your dream that is a subjective phenomenon; difficult to put in words. It's a goal for those seeking spiritual growth and liberation through meditation. Let us ponder on, "What are the health benefits of exercise"? Regular exercise and physical activity may: (a) Help you get and stay at a healthy weight. (b) Reduce your risk of heart diseases. (c) Help your body manage *blood glucose* and insulin levels. (d) Help you manage a chronic health condition. (e) Help quit smoking. (f) Improve your mental health, and mood. (g) Help keep your thinking, learning, and judgment skills sharp as you age. (h) Strengthen your bones and muscles. (i) Reduce your risk of some cancers, (j) Reduce your risk of *falls*. (k) *Improve your sleep*. (l) Boost your energy. (m) Improve your sexual health. (n) Increase your chances of living longer. (o)

Above all, it brings a sense of selfesteem that improves the work performance, self-esteem and confidence. Lastly, 'Do It Yourself" to believe it. Studies show that physical activity can reduce your risk of dying early from the leading causes of death, like heart disease and certain cancers, MedlinePlus [Internet].[36] Lee et al[37] observed, "Worldwide, we estimate that physical inactivity is responsible for between 6% and 10% of the major NCDs (non communicable diseases) of CHD (coronary heart disease), type 2 diabetes, and breast and colon cancers. And, this unhealthy behaviour is responsible for 9% of premature mortality, or >5.3 of the 57 million deaths in 2008. By eliminating physical inactivity, life expectancy of the world's population may be expected to increase by 0.68 years. Unquote." Zhang *et al*[38] elaborated, "Moderate exercise (around 30 min of moderate-intensity activity five to seven times a week) can provide multiple health benefits, although amount of exercise varies among individuals." While elaborating on, 'Effects of Exercise on Brain Health Through the Gut–Brain Axis', they went on, "Many studies have suggested that moderate exercise can promote healthy brain function, including enhancing memory, improving mood and cognitive performance. One study provides direct evidence that exercise alters gut microbiota profiles and plays a role in cognitive disorders. Indeed, exercise plays a key role in a variety of neurological diseases and can promote the reduction of oxidative stress and release of anti-inflammatory factors, regulate microglia, promote production of neurotransmitters, etc. Unquote."

(14) Author's illustrative anecdotes: Author treating these patients over half a century feels, "We must retune mindset focused on, *pills, potions, powders, pastes, pricks, petridish physiopathology mania*, leading to *effects* rather than deal with *cause* and learn to *peep in the platter plate (ie, diet, exercise, potable water etc)*"! Dictum goes, "As we eat and drink, so we become". Hippocrates posited, "Let food be thy medicine and medicine be thy food."



Legend: In pic 1, the Ape (Human ancestor) is calling to teach the lifestyle. In pic 2, showing thumbs up triumphantly for their living style. In pic 3 and 4, they are happily and mockingly rejoicing their lifestyle and natural living.

We have been ignoring valuable and important remedy that lies in our *dinner dish*, "What we *eat and drink* builds and transforms our body and mind?" Author always stresses on food and drinks as most important remedy. This is *res ipsa loquitur* (thing speaks for itself). The area Author is living in now, is historically called, "*Kaalaa Paani or Black Water*". Groundwater is very unhealthy. What we eat and drink has profound impact on physical and mental well-being that is not directly obvious but leads to *liberandi causa* (liberative prescription).

(15) Role of Fasting in Gut Health: Fasting intermittently is known and now studied by various groups showing positive effects. Mindikoglu *et al*[39] observed, "Based on the findings of studies conducted on human subjects, dawn-to-sunset fasting has potential to be a cost-effective intervention for obesity, metabolic syndrome and NAFLD. Unquote." The dawn-to-sunset fasting might be a cost-effective intervention to prevent obesity, metabolic syndrome, 'metabolic-dysfunction associated fatty liver disease' (MAFLD, 2020), 'non

alcoholic fatty liver disease' (NAFLD), 'non-alcoholic steato-hepatitis' (NASH), hepatocellular carcinoma and cirrhosis; is definitely seen advantageous. Regular rhythmic fasting is routinely practiced by devout people in India for times immemorial, though formal study for health benefit has not been carried out. Gofton *et al*[40] stated, "Since the introduction of the term, there have been key areas in which the superiority of MAFLD over traditional NAFLD terminology has been demonstrated, including for the risk of liver and extrahepatic mortality, disease associations, and for identifying high-risk individuals. Unquote." Visioli *et al*[41] conclude that the manipulation of dietary intake, in the form of calorie restriction, intermittent fasting, dietary restriction with the exclusion of some nutrients, prolonged fasting, and etc, is anthropologically engraved in human culture possibly because of its positive health effects.

(16) Role/Scope of Surgery in FGIDs: Normally there is no role of surgery in FGIDs, unless complicated and depends upon the situation and decision for surgical interference, availability of skilled surgeon, shall depend upon cohort's imminent phenotype.

(17) CONCLUSION

FGIDs are due to Western lifestyle leading to gradual transition of cohorts as explained by Rome Group alongwith 'gas and bloating', due to diet mainly consisting of animal protein, high in sugars and saturated fats, devoid of natural fiber, resulting in gut microbial dysbiosis, role of gut synbiotics in nutrition and metabolism resulting in syndromes like IBS, DD, or even IBD. Role of multivitamins, Laws of Physics modulating GI physiology, physiotherapy in FGIDs, role of fasting in gut health is seen effecting positively. Lastly role of surgery in FGIDs is limited unless some serious complications arise, eg, symptomatic complicated diverticular disease (SCDD) syndromes. Visceral hypersensitivity is result of change in the milieu interior of gut leading to dysbiotic physiopathology, disturbed motility and its remedy lies in reversal of faulty phenotype in lifestyle issues.

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To my parents and my patients, who made me what I am?

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REFERENCES

1. Teoli D and Bhardwaj A. Quality Of Life. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing, 2025 Jan-. Last Update: March 27, 2023. https://www.ncbi.nlm.nih.gov/books/NBK536962/#:~:text=For%20example%2C%20common%20facets%20of,social%20belonging%2C%20and%20physical%20surroundings. (Accessed 27/11/25)
2. Burkitt, Denis Parsons. Some diseases characteristic of modern Western civilization. Br Med J., Feb. 3, 1973; 1(5848): 274–278. https://www.bmj.com/content/bmj/1/5848/274.full.pdf (Full article - pdf) doi: 10.1136/bmj.1.5848.274 PMCID: PMC1588096 PMID: 4568142 https://pmc.ncbi.nlm.nih.gov/articles/instance/1588096/pdf/brmedj01541-0038.pdf (Accessed 17/06/25)
3. Drossman D A. Functional Gastrointestinal Disorders: History, Pathophysiology, Clinical Features, and Rome IV. History of the Functional Gastrointestinal Symptoms and Disorders and the Role of Psychosocial Factors. SECTION I: FGIDS: BACKGROUND INFORMATION | FUNCTIONAL GI OVERVIEW E2., May 2016; 150(6): 1262-1279. DOI: 10.1053/j.gastro.2016.02.032 Also available on ScienceDirect https://www.gastrojournal.org/article/S0016-5085(16)00223-7/fulltext (Accessed 20/02/2025)
4. Schmulson M J, Drossman D A. What Is New in Rome IV. J Neurogastroenterol Motil., Apr. 1, 2017; 23(2): 151–163. doi: 10.5056/jnm16214 PMCID: PMC5383110 PMID: 28274109. https://pmc.ncbi.nlm.nih.gov/articles/PMC5383110/ (Accessed 18/07/25)
5. DeGruttola A K, Low D, Mizoguchi A, Mizoguchi E. Current understanding of dysbiosis in disease in human and animal models. Inflamm Bowel Dis., May 2016; 22(5): 1137–1150. doi: 10.1097/MIB.0000000000000750 PMCID: PMC4838534 NIHMSID: NIHMS751286 PMID: 27070911 https://pmc.ncbi.nlm.nih.gov/articles/PMC4838534/#:~:text=Dysbiosis%20is%20defined%20by%20an,bacterial%20distribution%20within%20the%20gut. (Accessed 13/08/25)
6. Zapała B, Pustelnik J, Dudek A and Milewicz T. Differences in the Composition of *Akkermansia* Species and Families of *Christensenellaceae* and *Ruminococcaceae* Bacteria in the Gut Microbiota of Healthy Polish Women following a Typical Western Diet. *Diversity*, 2023; *15*(10): 1103. https://doi.org/10.3390/d15101103 https://www.mdpi.com/1424-2818/15/10/1103 (Accessed 5/10/25)
7. Moshiree B, Zhou Q, Price D D, Verne G N. Central sensitisation in visceral pain disorders. Gut., Jul. 2006; 55(7): 905–908. doi: 10.1136/gut.2005.078287 PMCID: PMC1856337 PMID: 16766744 https://pmc.ncbi.nlm.nih.gov/articles/PMC1856337/#:~:text=General%20mechanisms%20of%20visceral%20hyperalgesia&text=Allodynia%20however%20refers%20to%20a,processing%20of%20ascending%20afferent%20input. (Accessed 29/06/2025)
8. Camilleri M, Coulie B, Tack J F. Visceral hypersensitivity: facts, speculations, and challenges. Occasional viewpoint. Gut., 2001; 48: 125-131. https://doi.org/10.1136/gut.48.1.125

https://gut.bmj.com/content/48/1/125 (Accessed 29/06/2025)

9. Maryam S; Ghasemi C T; Babak T; Nazila K; Rahim K M; Peyman A. Relative Frequency of Gastrointestinal Functional Disorders in Patients with Inflammatory Bowel Disease Based on Rome IV: A Case-Control Study. *Advanced Biomedical Research, May 2024,* 13(1): 43. | *DOI:* 10.4103/abr.abr_369_23 https://journals.lww.com/adbm/pages/articleviewer.aspx?year=2024&issue=07120&article=00043&type=Fulltext (Accessed 29/04/25)
10. Šojat D, Volarić M, Keškić T, Volarić N, Cerovečki V, M ajnarić L T. Putting Functional Gastrointestinal Disorders within the Spectrum of Inflammatory Disorders Can Improve Classification and Diagnostics of These Disorders. Biomedicines, Mar. 21, 2024; 12(3): 702. doi: 10.3390/biomedicines12030702 PMCID: PMC10967747 PMID: 38540315 https://pmc.ncbi.nlm.nih.gov/articles/PMC10967747/ (Accessed 27/08/25)
11. Noddin L, Callahan M, Lacy B E. Irritable Bowel Syndrome and Functional Dyspepsia: Different Diseases or a Single Disorder With Different Manifestations? MedGenMed., Aug. 26, 2005; 7(3): 17. PMCID: PMC1681633 PMID: 16369243 https://pmc.ncbi.nlm.nih.gov/articles/PMC1681633/#:~:text=%5B2%5D%20Many%20patients%20with%20IBS,are%20often%20worsened%20by%20meals. (Accessed 27/08/25)
12. Li S, Wu T, Wu J, Chen W, Zhang D. Recognizing the biological barriers and pathophysiological characteristics of the gastrointestinal tract for the design and application of nanotherapeutics. Drug Deliv., Oct. 15, 2024; 31(1): 2415580. doi: 10.1080/10717544.2024.2415580 PMCID: PMC11485891 PMID: 39404464. https://pmc.ncbi.nlm.nih.gov/articles/PMC11485891/ (Accessed 28/09/25)
13. Marzano A V, Borghi A, Stadnicki A, Crosti C, Cugno M. Cutaneous manifestations in patients with inflammatory bowel diseases: pathophysiology, clinical features, and therapy. Review: Inflamm Bowel Dis., Jan. 2014; 20(1): 213-27. DOI: 10.1097/01.MIB.0000436959.62286.f9 PMID: 24105394. https://pubmed.ncbi.nlm.nih.gov/24105394/ (Accessed 29/06/2025)
14. Weizman AV, Huang B, Targan S, Dubinsky M, Fleshner P, Kaur M, Ippoliti A, Panikkath D, Vasiliauskas E, Shih D, McGovern D PB, Melmed G Y. Pyoderma gangrenosum among patients with inflammatory bowel disease: a descriptive cohort study. J Cutan Med Surg. Author manuscript; available in PMC: 2016 Nov 1. *Published in final edited form as:* J Cutan Med Surg, Oct. 2014; 18(5): 361. PMCID: PMC5089702 NIHMSID: IHMS822201 PMID: 25277124. https://pmc.ncbi.nlm.nih.gov/articles/PMC5089702/#:~:text=Pyoderma%20gangrenosum%20is%20a%20severe,absence%20of%20active%20underlying%20IBD. (Accessed 27/09/25)
15. Li H, Li W C & Hu X R. Association between vitamin C, D, and K intake and inflammatory bowel disease risk: findings from, 2009 to 2010 NHANES *BMC Gastroenterology*, 2025; 25: 177. Cite this article https://bmcgastroenterol.biomedcentral.com/articles/10.1186/s12876-025-03747-9#citeas (Accessed 17/11/25)
16. Roberfroid M, Gibson G R, Hoyles L, McCartney A L, Rastall R, Rowland I, Wolvers D, Watzl B, Szajewska H and Stahl B. Prebiotic effects: metabolic and health benefits. *British Journal of Nutrition*, 2010; 104(S2): S1-S63. DOI: https://doi.org/10.1017/S0007114510003363 https://www.cambridge.org/core/journals/british-journal-of-nutrition/article/prebiotic-effects-metabolic-and-health-benefits/F644C98393E2B3EB64A562854115D368 (Accessed 28/09/25)
17. Thursby E, Juge N. Introduction to the human gut microbiota. Biochem J., May 16, 2017; 474(11): 1823–1836. doi: 10.1042/BCJ20160510 PMCID: PMC5433529 PMID: 28512250 https://pmc.ncbi.nlm.nih.gov/articles/PMC5433529/ (Accessed 02/12/25)
18. Slavin J. Fiber and Prebiotics: Mechanisms and Health Benefits. Nutrients, Apr. 22, 2013; 5(4): 1417–1435. doi: 10.3390/nu5041417 PMCID: PMC3705355 PMID: 23609775 https://pmc.ncbi.nlm.nih.gov/articles/PMC3705355/ (Accessed 01/09/25))
19. Holscher H D. Dietary fiber and prebiotics and the gastrointestinal microbiota. Gut Microbes, Feb. 6, 2017; 8(2): 172–184. doi: 10.1080/19490976.2017.1290756 PMCID: PMC5390821 PMID: 28165863 https://pmc.ncbi.nlm.nih.gov/articles/PMC5390821/ (Accessed 01/09/25)
20. Soto-Martin E C, Warnke I, Farquharson F M, Christodoulou M, Horgan G, Derrien M, Faurie J, Flint H J, Duncan S H, Louis P. Vitamin Biosynthesis by Human Gut Butyrate-Producing Bacteria and Cross-Feeding in Synthetic Microbial Communities. Research Article 14 July 2020. https://doi.org/10.1128/mbio.00886-20 https://journals.asm.org/doi/10.1128/mbio.00886-20 (Accessed 03/10/25)
21. Magnúsdóttir S, Ravcheev D, de Crécy-Lagard V, Thiele I. Systematic genome assessment of B-vitamin biosynthesis suggests co-operation among gut microbes. Front. Genet., 20 April 2015; 6: 2015. https://www.frontiersin.org/journals/genetics/articles/10.3389/fgene.2015.00148/full. https://doi.org/10.3389/fgene.2015.00148 (Accessed 03/10/25)

22. Morowitz M J, Carlisle E, and Alverdy J C. Contributions of Intestinal Bacteria to Nutrition and Metabolism in the Critically Ill. Surg Clin North Am. Author manuscript; available in PMC 2012 Aug 1. Published in final edited form as:Surg Clin North Am., Aug. 2011; 91(4): 771–785. doi: 10.1016/j.suc.2011.05.001 PMCID: PMC3144392 NIHMSID: NIHMS296280 PMID: 21787967 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3144392/ (Accessed 03/10/25)
23. Liu M, Chen J, Dharmasiddhi I P W, Chen S, Liu Y and Liu H. Review of the Potential of Probiotics in Disease Treatment: Mechanisms, Engineering, and Applications. *Processes*, 2024; *12*(2): 316. https://doi.org/10.3390/pr12020316 https://www.mdpi.com/2227-9717/12/2/316 (Accessed 16/10/25)
24. Amara A A and Shibl A. Role of Probiotics in health improvement, infection control and disease treatment and management. Saudi Pharm J., Jul. 18, 2013; 23(2): 107. 114. doi: 10.1016/j.jsps.2013.07.001 PMCID:PMC4421088 PMID: 25972729 https://pmc.ncbi.nlm.nih.gov/articles/PMC4421088/ (Accessed 16/10/25)
25. Sarita B, Samadhan D, Hassan M Z, Kovaleva E G. A comprehensive review of probiotics and human health-current prospective and applications. REVIEW article. Front. Microbiol., 06 January 2025; 15: 2024. | https://doi.org/10.3389/fmicb.2024.1487641 https://www.frontiersin.org/journals/microbiology/articles/10.3389/fmicb.2024.1487641/full (Accessed 16/10/25)
26. Walker C, Boland A, Carroll A, O'Connor A. Concurrent functional gastrointestinal disorders in patients with inflammatory bowel disease. REVIEW article. Front. Gastroenterol., 12 August 2022; 2022 | https://doi.org/10.3389/fgstr.2022.959082 https://www.frontiersin.org/journals/gastroenterology/articles/10.3389/fgstr.2022.959082/full (Accessed 23/11/25)
27. Vasant DH, Ford AC. Functional gastrointestinal disorders in inflammatory bowel disease: Time for a paradigm shift? *World J. Gastroenterol*, 2020; 26(26): 3712-3719. PMCID: PMC7383849 PMID: 32774052 DOI: 10.3748/wjg.v26.i26.3712 ORCID number: Dipesh H Vasant (0000-0002-2329-0616); Alexander C Ford (0000-0001-6371-4359. Corresponding author: dipesh.vasant@mft.nhs.uk https://www.wjgnet.com/1007-9327/full/v26/i26/3712.htm (Accessed 29/08/25)
28. Li, H., Li, WC. & Hu, XR. Association between vitamin C, D, and K intake and inflammatory bowel disease risk: findings from 2009 to 2010 NHANES. *BMC Gastroenterol*, 2025; 25: 177. https://doi.org/10.1186/s12876-025-03747-9 https://bmcgastroenterol.biomedcentral.com/articles/10.1186/s12876-025-03747-9#citeas (Accessed 16/10/25)
29. Reich K M, Fedorak R N, Madsen K, Kroeker K I. Vitamin D improves inflammatory bowel disease outcomes: Basic science and clinical review. World J Gastroenterol, May 7, 2014; 20(17): 4934–4947. doi: 10.3748/wjg.v20.i17.4934PMCID: PMC4009525 PMID: 24803805 https://pmc.ncbi.nlm.nih.gov/articles/PMC4009525/ (Accessed 25/09/25)
30. Vemulapalli V, Thomas A S. The Role of Vitamin D in Gastrointestinal Homeostasis and Gut Inflammation. Int J Mol Sci., Mar 26, 2025; 26(7): 3020. doi: 10.3390/ijms26073020 PMCID: PMC11988781 PMID: 40243631 https://pmc.ncbi.nlm.nih.gov/articles/PMC11988781/ (Accessed 25/09/25)
31. Clinton S K, Giovannucci E L, Hursting S D. The World Cancer Research Fund/American Institute for Cancer Research Third Expert Report on Diet, Nutrition, Physical Activity, and Cancer: Impact and Future Directions. J Nutr., Nov. 23, 2019; 150(4): 663–671. doi: 10.1093/jn/nxz268 PMCID: PMC7317613 PMID: 31758189 https://pmc.ncbi.nlm.nih.gov/articles/PMC7317613/ (Accessed 25/09/25)
32. Aggeletopoulou I, (iaggel@upatras.gr), Marangos M, Assimakopoulos S F, Mouzaki A, Thomopoulos K, Triantos C. Vitamin D and Microbiome: Molecular Interaction in Inflammatory Bowel Disease Pathogenesis. REVIEW, June 2023; 193(6): 656-668. DOI:10.1016/j.ajpath.2023.02.004 Also available on ScienceDirect External Link https://ajp.amjpathol.org/article/S0002-9440(23)00055-X/fulltext (Accessed 25/09/25)
33. Srivastava A, Sood A, Joy P S, Mandal S, Panwar R, Ravichandran S, Sarangi S, Woodcock J. Principles of Physics in Surgery: The Laws of Mechanics and Vectors Physics for Surgeons—Part 2. Indian J Surg, Nov. 16, 2010; 72(5): 355–361. doi: 10.1007/s12262-010-0155-8 PMCID: PMC3077132 PMID: 21966132 (Accessed 19/11/25)
34. Wilderman, C T. and Sinha, M. "Namaste in the USA: The Growing Pains that Traditional Yoga Faces in America – An International Marketing Case Study," The Kennesaw Journal of Undergraduate Research, 2019; 6(2): 5. DOI: https://doi.org/10.62915/2474-4921.1161 Available at: https://digitalcommons.kennesaw.edu/kjur/vol6/iss2/5 https://digitalcommons.kennesaw.edu/cgi/viewcontent.cgi?article=1161&context=kjur (Accessed 31/07/2025)
35. Castaneda, C. "America the Yogiful: Insights Into American Yoga Culture Today" (2014). A Project Submitted in Partial Fulfillment of the Requirements for the Degree of Master of Liberal Studies. May, 2014. Rollins College, Hamilton Holt School,

Master of Liberal Studies Program. http://scholarship.rollins.edu/mls/55 Correspondence: Carolina Castaneda; ccastaneda@rollins.edu https://scholarship.rollins.edu/cgi/viewcontent.cgi?params=/context/mls/article/1056/&path_info=auto_convert.pdf (Accessed 31/07/25)

36. MedlinePlus [Internet]. Bethesda (MD): National Library of Medicine (US); Benefits of Exercise. [updated Jun 24; cited 2020 Jul 1]. Available from: https://medlineplus.gov/. https://medlineplus.gov/benefitsofexercise.html#:~:text=For%20example%2C%20it%20can%20strengthen,heart%20disease%20and%20certain%20cancers. (Accessed 17/11/25)
37. Lee I M, Shiroma E J, Lobelo F, Puska P, Blair S N, Katzmarzyk P T, for the Lancet Physical Activity Series Working Group. Impact of Physical Inactivity on the World's Major Non-Communicable Diseases. *Published in final edited form as:* Lancet, Jul. 21, 2012; 380(9838): 219–229. doi: 10.1016/S0140-6736(12)61031-9 PMCID: PMC3645500 NIHMSID: NIHMS385288 PMID: 22818936 https://pmc.ncbi.nlm.nih.gov/articles/PMC3645500/ (Accessed 17/11/25)
38. Zhang L, Liu R, Song Z and Zhang X. Exercise, Diet, and Brain Health: From the Perspective of Gut Microbiota Regulation. *Nutrients*, 2025; *17*(10): 1686; https://doi.org/10.3390/nu17101686 https://www.mdpi.com/2072-6643/17/10/1686#:~:text=4.-,Effects%20of%20Exercise%20on%20Brain%20Health%20Through%20the%20Gut%E2%80%93Brain,mood%2C%20and%20improving%20cognitive%20performance. (Accessed 12/11/25)
39. Mindikoglu A I, Opekun A R, Gagan S K, Devaraj S. Impact of Time-Restricted Feeding and Dawn-to-Sunset Fasting on Circadian Rhythm, Obesity, Metabolic Syndrome, and Nonalcoholic Fatty Liver Disease. Gastroenterol Res Pract, Nov. 19, 2017; 2017: 3932491. doi: 10.1155/2017/3932491 PMCID:PMC5733887 PMID: 29348746 https://pmc.ncbi.nlm.nih.gov/articles/PMC5733887/ (Accessed 01/10/25)
40. Gofton C, Upendran Y, Zheng M H, George J. MAFLD: How is it different from NAFLD? Clin Mol Hepatol, Nov. 29, 2022; 29: S17–S31. doi: 10.3350/cmh.2022.0367 PMCID: PMC10029949 PMID: 36443926. https://pmc.ncbi.nlm.nih.gov/articles/PMC10029949/#:~:text=%E2%80%9CMetabolic%20dysfunction%2Dassociated%20fatty%20liver%20disease,related%20to%20systemic%20metabolic%20dysregulation. (Accessed 17/12/25)
41. Visioli F, Caretta C M, Anile F, Panaite S A. Traditional and Medical Applications of Fasting. Nutrients, Jan. 19, 2022; 14(3): 433. doi: 10.3390/nu14030433. PMCID: PMC8838777 PMID: 35276792 https://pmc.ncbi.nlm.nih.gov/articles/PMC8838777/ (Accessed 29/11/25)