EXPERT REVIEWS

A common cause of irritable bowel syndrome and diverticulitis: chronic distal colon distention from sedentary behavior and excessive dietary fiber

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A multidisciplinary analysis restricted to validated reports was applied to the cause and management of irritable bowel syndrome and diverticular formation and subsequent diverticulitis. There is evidence that they are linked – both caused by attenuation of gravitational aid to distal intestinal motility, resulting in damaging chronic intestinal distention. Both irritable bowel syndrome and diverticular formation and subsequent diverticulitis have worsened in recent years owing to excessive dietary fiber intake. Potential solutions include augmenting weight-bearing time, moderating dietary fiber consumption, stimulating distal colon evacuation through chemical means and developing pharmaceuticals to block the reflexive distal colon distention associated with fiber consumption. Amplified intestinal distention commenced when all classes of Renaissance Europeans became the first group in human history to wear shoes, which led to a sedentary lifestyle that moderates gravitational aid to colon motility and evacuation.

Keywords: descending colon • dietary fiber • distention • diverticulitis • diverticulosis • historical diets • irritable bowel syndrome • sedentary

Attention has recently been focused on expanding the analysis of health problems beyond the arbitrary traditional boundaries imposed by subspecialties and even traditional health sciences through a multidisciplinary approach. By expanding the pool of relevant verified reports, it has been hoped that new insight could be gained, particularly in areas that have stagnated. Certainly, our understanding of the cause or causes of irritable bowel syndrome (IBS) and diverticular formation and subsequent diverticulitis (DFSD) seems particularly suited for this approach, since it remains as obscure as it was when these conditions were first identified ages ago.

The cause or causes of IBS and DFSD may be related. They affect mainly the distal descending and sigmoid colon [1–5]. Both are similar in prevalence, although they commence at differing ages – IBS usually beginning in late adolescence, whereas DFSD incidence begins to rise in middle age [1–5]. There are regional disparities in the incidence of DFSD, and perhaps IBS, that are poorly explained by differences in dietary fiber consumption. The incidence of DFSD and perhaps IBS is highest in the western industrialized countries and lower in Asia [6,7]. The incidence of both may have increased [6–8]. Diverticulitis commences at a younger age now compared with decades ago, and IBS and perhaps DFSD are more common in sedentary individuals [9]. Since the majority of the population have at least modest symptoms of IBS, most sufferers of DFSD probably suffered from IBS symptoms earlier in life. This suggests that over time, IBS might lead to DFSD.

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Prevention of IBS and DFSD is not possible since successful interventions require understanding of cause, and no plausible causal mechanisms have been advanced for either, let alone one that explains both [1–5]. Contemporary standards of proof of causality of a disorder require both a statistical association between disorder and proposed cause together with a plausible (best proven) causal mechanism [10]. Proof of causality is further advanced when it satisfies the principle of 'parsimony' (referred to sometimes as 'Ockham's razor'), which, in the present context, is considered a single causal hypothesis which explains both IBS and DFSD [10]. It would be further strengthened if it also explained other related issues [10], such as why the incidence of both IBS and DFSD appears to be increasing [8], why augmenting dietary fiber has failed to prevent and perhaps exacerbated both conditions [8], regional disparities in DFSD [6,7], why DFSD incidence is increasing in a younger age group [9] and why IBS is more prevalent in sedentary individuals [11].

Dietary fiber consists of cellulose, which humans digest poorly. Diets rich in fiber were introduced for the management of IBS and DFSD decades ago [12,13] without any scientific reports that meet high standards supporting their effectiveness in preventing and managing those conditions [12]. They continue to be recommended despite reports indicating that augmenting dietary fiber fails to prevent DFSD and is less effective than prunes in dealing with symptoms of IBS [14,15]. Their use was based on the presumption that humans evolved consuming foodstuffs richer in fiber; consequently, human intestines are adapted to this condition [12,15,16]. The most popular mechanism proposed for DFSD has been intestinal wall damage from excessive interluminal pressure caused by low-fiber diets [12,13,15–17].

This article first deals with the validity of the assumptions upon which the practice of augmenting fiber was based. A single causal explanation is then presented that explains both IBS and DFSD and is consistent with recent developments in the study of historical diets and intestinal pathophysiology. It meets contemporary standards of proof of causality. Furthermore, it explains why increasing dietary fiber consumption in contemporary populations has failed to prevent, and may actually have exacerbated, IBS and DFSD. Most importantly, it leads to improved treatment and portends prevention of these troubling disorders.

Pertinent pathophysiology

The physiology of visceral pain and intestinal motility as it applies to the large intestine is complex and evolving, yet the issues relevant to this discussion are uncontroversial. Distention, inflammation and ischemia are sufficient stimuli to induce intestinal pain [18–22]. Pain results from central processing of afferent information from high-threshold stretch receptors (nociceptors) or via these receptors, nerves and intestinal ganglia acted upon by a variety of chemical agents released from damaged intestinal tissues or cells that mediate the inflammatory response [20].

Intestinal motility (organized contraction that propels contents) is controlled by two nervous systems. Central control is exerted by the autonomic nervous system, which slows motility unless overridden by the independent second 'enteric nervous system' [19–23]. This network utilizes mainly serotonin-based neurotransmitters produced by enterochromaffin cells in the intestine (90% of human serotonin is based in the intestine) [21]. This secretion amplifies motility in response mainly to distention of the intestine [18]. Contractility of intestinal muscle is also strongly influenced by the same chemical agents discharged from damaged intestinal tissue and products of the inflammatory process that cause pain; however, this contraction is disorganized (spasms), and therefore does not involve motility [19–22].

The intestinal skeleton (IS) is considered to be a structure that strongly maintains tubular integrity. It is composed of collagen of the mesenteric plexus and collagen network intrinsic to (and created by) the robust muscularis propria of the large intestine [24]. The IS is vulnerable to damage from distention caused by chronic constipation, regardless of cause [24]. Diverticula are harmless (in themselves) small bubble-like breaches of the IS that are found almost exclusively in the distal descending and sigmoid colon (the last 50 cm of the intestine). They possess normal intestinal epithelium but only remnants of muscularis propria and IS, presumably due to damage to the IS from chronic excessive distention. Therefore, they are devoid of peristalsis, and poorly resist distention from even mild pressure exerted by intestinal contents [24]. Diverticula are of health concern only because they often become infected (diverticulitis), burst and seed the abdominal cavity with fecal bacteria, resulting in a medical emergency [1].

IBS is a group of signs and symptoms involving disordered distal intestinal motility that commences in adolescence, affects all individuals to some degree when considering mild symptoms such as periodic bloating and cramping, and involves essentially the same region of the large intestine subject to DFSD [1]. IBS is characterized by abdominal distention, variable constipation and 'overflow diarrhea', cramping pain and nausea – identical to the signs and symptoms of partial distal intestinal obstruction, yet the similarity of the two is rarely mentioned [1]. Since few humans are totally free of symptoms of IBS, its diagnosis is based mainly on just the frequency and intensity of the symptoms, individual intolerance of symptoms and availability of medical professionals to make the diagnosis [1]. Mediators of pain and localized intestinal contraction tend to be present in high concentrations in sufferers of IBS [1,23]. This is consistent with anatomical evidence of minute areas of inflammation [23]. The disordered contractility found in IBS is accounted for by high concentrations of various chemicals released when the muscularis propria is damaged – therefore, it is simply a response to damage caused by distention from obstruction [21]. It seems conceivable that, over time, damage signaled by symptoms of IBS causes sufficient IS damage to result in DFSD. What has been lacking in the understanding of both IBS and DFSD is the cause of this partial obstructive state.

Influence of gravity on intestinal motility

It has become apparent that upright members of the genus *Homo* rely on gravitational force for normal function of the intestines, as the zero-gravity conditions experienced during space travel predictably result in intestinal obstructive symptoms [25]. Gravitational force acting upon the human body is necessary for normal distal intestine motility, although not entirely, as every physician can attest to the fact that nearly all bedridden patients become constipated, which explains standard orders in nearly every hospital for the use of laxatives in all patients confined to the recumbent posture. Normal distal intestinal function requires gravity but also a considerable time spent upright.

Under normal circumstances, large intestinal contents are liquid at least until the beginning of the descending colon. Thus they require modest peristaltic force to propel them. Intestinal contents tend to be semisolid in most of this distal colon. The descending colon is aligned parallel to the spinal column and, hence, is vertical when upright. This allows gravity to maximally aid in transit and evacuation. Since formed stool requires far greater force generated by intestinal muscle to effect transit compared with liquid contents, when upright, gravitational force assists when it is needed the most. This helps avoid overwhelming the ability of the distal colon intestinal muscle, which results in overdistention and, over time, permanent damage to contractility, which further interferes with motility and eventual evacuation.

Paradoxical negative relationship between distention & interluminal pressure

When dietary fiber intake varies, the relationship between intestinal interluminal pressure and fiber intake is negative – diets rich in fiber moderate distal colon interluminal pressure [12,13,16]. Perhaps health professionals recommend fiber-rich diets for the management of IBS and DFSD based on the assumption that the pains of IBS and intestinal damage of DFSD are caused by interluminal pressure alone, or because they assume that the relationship between interluminal pressure and damaging intestinal distention is also positive, as would be predicted if the intestine behaved passively as a viscoelastic tube whereby flow and interluminal pressure follow Bernoulli's principle, and wall tension and luminal diameter are related following the Young–Laplace equation [2–4,12,16]. This assumption seems unlikely considering the neurological systems that effect large intestinal mechanics and substantial muscle elements in the distal colon wall.

From the outset, it was apparent that this simplistic model was inadequate for understanding distal colon mechanics. Lowering interluminal pressure through dietary fiber counterintuitively resulted in radiologically distal intestinal distention and symptoms of bloating. Contrary to predictions based on the Young–Laplace equation, the relationship between interluminal pressure and distention is negative – lowering pressure through dietary fiber results in greater distention [16,17]. This is best explained as behavior in response to sensing solidity of distal large intestinal contents, probably via intestinal wall stretch receptors. When less solid (softer and more liquid) contents typical of a diet rich in fiber enter the distal colon, symptoms of impending colon damage and microtrauma to the intestinal wall that is typical of IBS are initiated in the form of delayed evacuation through moderated motility and amplified distention. Conversely, when fiber-poor intestinal contents reach the distal colon, interluminal pressure is amplified without distention resulting in immediate peristalsis and evacuation. This delay in evacuation and distention associated with high-fiber ingestion can perhaps be explained from an evolutionary perspective as a mechanism to preserve water homeostasis through dehydrating distal colon contents – a mechanism used by many species. This adaptation to relative water scarcity would rarely, if ever, have caused intestinal damage until modern times because, as will be mentioned later, data regarding historical diets indicate that humans never consumed sufficient dietary fiber to cause exaggerated distention [18–21]. Regardless of the explanation, when more fiber is consumed, the distal colon becomes distended, resulting in symptoms of impending and localized damage from distention that are identical to IBS, which over time probably result in damage to the IS – the precondition of DFSD. This explains why agents that force evacuation through chemical means have been shown to more effective than dietary fiber in the form of psyllium seed husks (Metamucil®, Proctor and Gamble, OH, USA) in moderating symptoms of IBS [15].

Humans evolved consuming a low-fiber diet

The genus *Homo* emerged perhaps 2.5 million years ago in sub-Saharan Africa, probably as nomadic hunter–gatherers. *Homo sapiens* separated from a common ancestor perhaps 250,000 years ago. Isotope methods are now able to reconstruct diets of humans from ancient skeletal remains. These investigations have shown that humans evolved consuming a low-fiber diet. From their origins until the early Holocene period (~10,000 years ago), they consumed a diet consisting of 80% animal flesh, which has essentially no dietary fiber value, and 20% material of plant origin, much of which was rich in dietary fiber [26–29]. In the early Holocene period (10,000 years ago), *H. sapiens*, the sole extant member of the genus, began to develop sufficient technology in agronomy and animal husbandry to remain fixed in location and grow crops. Those who ceased to be nomadic continued with a diet rich in animal flesh (60%) [26–29]. The above information refutes the notion that humans evolved eating a diet rich in fiber and, therefore, are well adapted to it. Rather, humans evolved consuming less fiber than is found in typical contemporary diets [26–29]. This suggests that humans might be poorly adapted to the augmentation of dietary fiber that is currently popular because they never experienced this condition in their evolutionary history, and this might partly account for present distal colon disorders.

The recent origin of human sedentary behavior

Chairs were uncommon in the home and workplace prior to the European Renaissance. They were used almost exclusively as a symbol of power and prestige in many societies [30]. Chairs became an article in general use for the first time in human history

in all European countries following the Renaissance. In English, French, Italian and Spanish, the equivalent of the word 'sedentary' was created to describe people habituated to sitting. It was a neologism (without equivalence in earlier forms of each respective language), which suggests that a word was needed to describe the phenomenon commencing across Europe consisting of spending a significant amount of wakeful time in the semirecumbent position (seated – sedentary).

The transition to the sedentary lifestyle coincides closely with the introduction of footwear use for the first time in human history by all classes. Pre-Renaissance Europe was essentially barefoot, notwithstanding small numbers from elite classes that wore them. This remained until the 'Black Death' ravaged southern Europe. To avoid contact with the presumed contaminated substrate according to reintroduced Hellenistic health beliefs, Europeans became the first group in human history to adopt the practice of wearing shoes in all social classes. Footwear produce instability [31–34]. Mechanisms that humans use to maintain stable equilibrium take precedence over adaptations to sustain plantar skin during prolonged standing (weight-bearing without locomotion) via constantly repositioning plantar load [35]. The pain associated with impending plantar skin damage forced Renaissance Europeans to change from almost continual wakeful weight-bearing to being largely semirecumbent. The tradition of footwear by all classes outside of Europe can be traced to European colonization and cultural imperialism.

Hypothesis: role of sedentary behavior in distal intestinal disorders

IBS and DFSD were probably rare conditions in humans prior to the European Renaissance because they spent most of their wakeful time upright and consumed a low-fiber diet, both of which aid evacuation and minimizes lower intestinal distention [26–29]. Renaissance Europeans became the first group in human history to spend a significant amount of wakeful time in the semirecumbent position [30], a condition humans had never reviously experienced in their evolutionary history. The sedentary lifestyle of Europeans resulted in less gravitational force aiding distal colon motility and evacuation – a state of partial distal colonic obstruction with symptoms now considered to constitute IBS. Over time, this distention resulted in intestinal wall damage and DFSD [24]. Increases in the incidence of IBS and DFSD in recent years can be attributed to amplification of sedentary behavior and the introduction of augmenting dietary fiber, which results in further distal colon distention in already partially obstructed individuals [16].

This sedentary and dietary fiber hypothesis is parsimonious in that it provides vast explainability. It explains the relationship between IBS and DFSD. IBS is seen as symptoms and signs of partial distal intestinal obstruction via a proven mechanism consisting of altered gravitational influence on intestinal function through sedentary behavior exacerbated by excessive dietary fiber intake. This results in a multitude of microtraumas from overdistention, and eventually DFSD from breaches in the IS caused by microtraumas over time. Other aspects of explainability of this hypothesis will be mentioned briefly. The hypothesis explains why dietary fiber has not improved both conditions and probably worsened them through the positive relationship between dietary fiber consumption and distal colon distention. It explains the reported association of IBS and sedentary behavior [9], why IBS commences in an younger age group than DFSD [9] and the increased incidence of both IBS and DFSD in recent times and DFSD in younger-aged groups [9,12]. It explains why humans evolved with a distal intestine inadequate for contemporary life. The difference in diverticulitis incidence between Asians and North Americans is perhaps explained by greater weight-bearing time in Asian populations [6–8].

Rethinking management of IBS & DFSD

Primary prevention of permanent IS damage that results in the chronic obstructive state that is associated with the sedentary nature of modern life would require avoiding a sedentary lifestyle from birth – remaining upright most wakeful hours. This is presently unrealistic given the explicit social norms requiring footwear use in many situations and implicit social norms inducing footwear use. Furthermore, technology in daily use has been designed to be used in the seated position, although enlightenment about the hazards of footwear portends making daily life more amenable to weight-bearing. Since essentially all humans presently alive presumably have sustained some permanent intestinal damage from being sedentary, and will maintain suboptimal weight-bearing time in the immediate future, the relevant issues are secondary and tertiary prevention.

Public education may have significant secondary and tertiary prevention value. The populace needs to be informed about the risk of a sedentary existence and the hazards of consuming greater than modest amounts of fiber-rich foods. This may be no easy task considering not only the well-meaning but misguided convictions of so many health professionals, but also the vested interest of industry in the business of manufacturing and marketing foods rich in fiber.

What alternatives are available to mitigate intestinal damage that would otherwise be inevitable? Stimulation of distal colon motility and evacuation by chemical means might moderate symptoms of IBS the avoid DFSD. This harkens back to the 'pre-fiber' era when prunes and indigestible oils were considered healthful. Prunes contain neochlorogenic acids, chlorogenic acids and sorbitol, which have mild laxative properties and are considered harmless. A recent report has indicated that prunes are more effective in aiding intestinal evacuation than fiber, which is consistent with the above analysis [15]. Use of prunes or equivalent agents could moderate distention, which damages the large intestine IS.

One problem associated with reducing dietary fiber content is that many fiber-rich foods are low in calories. Reducing dietary fiber might come at a cost of aggravating obesity. It may be possible to moderate the distention caused by fiber (the negative relationship between fiber and distention) and thereby prevent IBS and DFSD through pharmaceutical means.

This would necessitate development of drugs that eliminate or moderate the reflexive distention of the distal intestine associated with high-fiber intestinal contents – a neurological adaptation of the lower colon that probably evolved to maintain water homoeostasis. The investigators have identified many of the autonomic and enteric neurotransmitters in the colon. Perhaps a false transmitter could be developed that specifically blocks the distention and delay in motility associated with fiber-rich colon contents. Considering that essentially the whole population is in need of primary, secondary and tertiary prevention of these conditions, an effective pharmaceutical intervention has enormous commercial value.

Conclusion

All members of genus *Homo* were probably free of IBS and DFSD when they were weight-bearing for most of their wakeful hours and eating a low-fiber diet. Perhaps the recent popularity of a diet richer in fiber would have been without health consequences so long as humans remained mainly upright. With the European Renaissance, for the first time in human history, a large group began sitting for a considerable amount of their wakeful hours as a consequence of them being also the first group in history to wear shoes. Footwear interfere with inherent mechanisms humans use to continuously redistribute plantar load that allows them to stand for long periods. Humans have been unable to adapt to the attenuation of the effect of gravitational force that aids descending colon motility. The chronic partial obstruction resulting from a sedentary lifestyle causes symptoms of IBS. Colon distention over time causes DFSD. The fashion of augmenting fiber may have exacerbated the problem. Increase in weight-bearing is the essential element in prevention and management of these disorders of modern life. Since suboptimal weight-bearing will continue, consideration should be given to moderating dietary fiber consumption and stimulating distal colon motility and evacuation through chemical means.

Expert commentary

There has been a rush to set up multidisciplinary research departments in health science institutions with hopes of attaining novel insights in areas that have stagnated. The results of these efforts have not been remarkable, perhaps because the model used for these departments has been 'research by committee' – a group of expert individuals from various fields attempting to combine elements from their area of expertise in order to achieve an original result. This approach fails to grasp that important discovery typically comes from an individual willing to stray beyond the arbitrary boundaries of a single discipline – the approach followed in this report. This approach was applied to IBS and DFSD, both of which affect the distal descending colon and appear to be increasing in incidence. They are of unknown cause when the analysis has been restricted to traditional medical analysis. Dietary fiber continues to be recommended for both conditions, despite a lack of conviction from practitioners, and rather a difficulty in admitting error to patients and unwillingness to face the void of ideas once fiber is appreciated for what it is – a pseudoscientific folk remedy. There is a desperate need for evidence-based explanation for IBD and DFSD, which might lead to more satisfactory prevention.

Verified scientific reports from anthropology, evolutionary biology, biomechanics and neurophysiology are added to more typical data drawn from epidemiology, gastroenterology and pathology. A unified IBS and DFSD cause emerges from this multidisciplinary analysis that meets contemporary scientific standards for validity. Loss of gravitational force aiding distal colon motility and evacuation resulted from the change in Renaissance Europeans from weight-bearing to sedentary for most of their wakeful time for the first time in human history. This causes signs and symptoms of mild chronic partial distal colon obstruction with excessive distention typical of IBS. Distention over time is known to result in damage to the IS which explains DFSD. The popularity of augmenting dietary fiber may have worsened both conditions because of reflexive delay in emptying of the distal colon and further distention when intestinal contents are fiber rich – a concept that is well known but never clearly articulated. This article presents a solid foundation for future investigations and preventive and treatment interventions.

Five-year view

Public education regarding myths about dietary fiber will be essential. There is a need to begin the process of informing the public that there is no quality research indicating that augmenting dietary fiber is beneficial to humans. Also, people must be prepared for the possibility that it may be hazardous, based on recent epidemiological reports. Even before definitive scientific reports are available, there seems little risk in preparing the public for the possibility that increase in weight-bearing time and mild stimulation of motility and evacuation by chemical means, such as through the use of prunes, may be superior to augmenting dietary fiber. Outcomes of interventions that diminish chronic distal intestinal distention must be tested objectively through population studies. Results for this type of research dealing with chronic problems and secondary and tertiary prevention typically take years; therefore, it is important to embark on these projects as soon as possible.

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Key issues

- Management of irritable bowel syndrome (IBS) and diverticulitis is currently unsatisfactory, at least in part because the cause of both is unknown.
- No convincing reports indicate that dietary fiber is useful in managing IBS and diverticular formation and subsequent diverticulitis (DFSD).
- Dietary fiber augmentation may be hazardous because there appears to have been an increase in incidence of both IBS and DFSD since its introduction.
- The scientific basis for the introduction of augmented dietary fiber diets has been refuted.
- A unified hypothesis, explaining both IBS and DFSD, is presented that meets contemporary scientific standards of proof of causality.
- These conditions are caused by by attenuation of the effect of gravity on distal colon motility and evacuation – from the change from weight-bearing most wakeful hours to sedentary, which commenced with Renaissance Europeans.
- Chronic overdistention damages the intestinal skeleton of the colon.
- Addition of dietary fiber worsened both conditions by causing further distention.
- Fiber intake should be moderated.
- Prevention of both conditions requires more weight-bearing time and moderating chronic distal colon distention through chemical means.
- A drug could be developed that blocks the reflexive distal colon distention associated with high-fiber intestinal contents.

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