Irritable bowel syndrome: anxiety in gastroenterology

Irritable bowel syndrome is in many ways still a famous 'heartsinker' but over the past half century a significantly better understanding of the pathophysiology has evolved, improving overall treatment.

In the recent past poor understanding of irritable bowel syndrome (IBS) created opportunities for communication problems during the consultation process, resulting in mutual frustration for both patients and doctors, with limited treatment options leading to unsatisfactory outcomes.

Originally IBS was thought to represent a nervous disorder with augmented gastrointestinal motility occurring in anxious patients under stress. Alternatively it was seen as a primary end-organ disorder of intestinal function. Research has shown that IBS is a complex multifaceted disorder that could be described as a group of functional gastrointestinal disorders (FGID) with different pathophysiological mechanisms but some common clinical features. It can be conceptualized within the biopsychosocial model of illness as a dysregulation of the brain–gut axis and its relationships with psychosocial and environmental variables.

In many people, however, stress or feelings of anxiety, guilt or resentment still seem to trigger the symptoms. These 'non-intestinal' symptoms are important for a number of reasons. They can be more intrusive than the classical features of IBS, contributing to making it probably the most common abdominal complaint brought to the attention of doctors, affecting one-third to one-half of all patients who seek relief from gastrointestinal problems. This article aims to highlight some of the new findings in IBS and will try to persuade that there is much more to IBS than reaching a diagnosis and treating with fibre supplements and antispasmodics.

Epidemiology

IBS is the commonest FGID (Drossman et al, 1993). One-year prevalence rates range from 3% to 20% in Europe (Hungin et al, 2003) and the United States. It is estimated to affect about 9 million people in the UK. This disease is also common in Japan, China, South America and India, accounting for 20–50% of all referrals to gastroenterology clinics. Female sufferers outnumber males 1.9 to 1. It may present at any age and symptoms may emanate from the whole gut rather than just the colon.

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Presentation

IBS is actually a collection of symptoms. In many patients abdominal pain in the immediate postprandial period in the mornings is associated with either rhythmic contractions or high amplitude prolonged contractions – the so-called 'morning rush.' These alterations in the migratory motor complex (MMC) can either delay or accelerate intestinal transit (Kellow and Phillips, 1987). Abnormal small intestinal and colonic motility has been demonstrated and in some patients has been shown to correlate with symptoms. Abnormalities of intestinal motility may lead not only to the onset of pain but also to bloating and, if abnormal motility results in changes in intestinal transit, predominant constipation or diarrhoea, or alternating between the two. Symptoms are chronic, with remissions interrupted by relapses precipitated by stress or changes in bowel flora. Symptoms may begin following an episode of gastroenteritis.

Diagnosis

Because there is no biological marker to confirm the diagnosis of IBS, it is a diagnosis that has challenged clinicians for decades. In the past, IBS was a 'waste-basket' diagnosis given to patients with unexplained gastrointestinal symptoms. It was considered to be 'the diagnosis of exclusion' when extensive work-up for organic disease yielded no diagnosis.

Symptom-based criteria

In contrast to diagnosing by exclusion, published diagnostic guidelines from Drossman et al (2002) recommend using symptom-based criteria to diagnose IBS in clinical practice (*Tables 1* and *2*).

Alarm features

Using these criteria in conjunction with alarm features (*Table 3*) allows a physician to minimize the extent of diagnostic testing needed to make the diagnosis of IBS. Furthermore, in a systematic literature review Cash et al (2002) showed that performing a number of diagnostic tests did not result in a significant increase in the diagnosis of organic gastrointestinal disease.

The blind spot

To challenge clinicians further, the psychiatric co-morbidity of FGID is as high as 40–90% (Castle et al, 2004). Simon et al (1999), in an international study, showed that 69% of patients with depression and anxiety reported only physical symptoms as their reason for visiting the physician. Kirmayer et al (1993) showed that only 22% of patients suffering with depression and who presented in this way were correctly diagnosed as having depression, as opposed to 77% who presented with psychological complaints. This presentation 'blind spot' is an important factor in misdiagnoses and non-treatment of patients with co-morbid psychiatric disorders.

The brain–gut axis interactions

The brain–gut axis is the continuous back and forth interactions of information and feedback that takes place between the gastrointestinal tract, enteric nervous system (ENS), the autonomous nervous system (ANS) and the brain and spinal cord, i.e. the CNS. These interrelated feedback circuits can be activated by an external or internal factor or stimulus that makes a demand on the system, such as a stressful event, an injury, an emotional thought or feeling, or even the ingestion of food. This unifying hypothesis is that the symptoms of IBS result not just from afferent sensory signals arising from a disordered gut, but also from dysregulation of the brain–gut axis.

The varied influences of environmental stress, thought and emotions on gut function help explain the variation in symptoms of patients with these disorders. It also helps explain how psychosocial trauma (e.g. history of physical or sexual abuse) or poor coping style (e.g. 'catastrophising') profoundly affects symptom severity, daily function and health outcome (Drossman et al, 1995). It is no longer rational to try to discriminate whether physiological or psychological factors cause pain or other bowel symptoms. Both are operative, and the task is to determine the degree to which each contributes and is remediable.

Dysregulation may occur at any level in the brain–gut axis because of the non-site-specific nature of neurotransmitters. This explains how it can interact with other physiological (endocrine and immune function) and psychosocial (behaviour, personality, cognition, emotional arousal) systems (Drossman, 1999).

According to Porcelli (2004), '"Top-down" signals from the brain to the gut assure that the digestive function is optimized by modulating motility, secretion, immune function, and blood flow. For example, it was found that the primary motor cortex includes bilateral representation of the anal sphincter and pelvic floor muscles and transcranial magnetic stimulation can induce changes in anorectal function.

"Bottom-up" signals from the gut to the brain also play a role in reflex regulation. For instance, studies using positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) showed that patients with IBS had greater activation of the anterior cingulate cortex (ACC) and thalamus with painful distension than with nonpainful stimulation, unlike control subjects. ACC hyperactivity to visceral stimulation could explain the heightened pain sensitivity as well as altered descending reflexes that stimulate GI [gastrointestinal] motility. Subjects who by hypnotic suggestion perceived the experience as unpleasant had increased ACC activity on PET imaging compared to subjects who were given the suggestion that it was pleasant.'

Table 1. Rome II diagnostic criteria for irritable bowel syndrome

Abdominal pain or discomfort for at least 12 weeks (need not be consecutive) in the preceding 12 months, accompanied by two of the following three features:	Relieved with defecation
	Onset associated with a change in stool frequency
	Onset associated with a change in stool form (appearance)
Symptoms that support the diagnosis of irritable bowel syndrome	Abnormal stool frequency, which may be defined as greater than three bowel movements per day or fewer than three bowel movements per week
	Abnormal stool form (lumpy/hard or loose/watery)
	Abnormal stool passage (straining, urgency, or feeling of incomplete evacuation)
	Passage of mucus
	Bloating or feeling of abdominal distension

From Drossman et al (2002)

Life stress, the hypothalamus–pituitary–adrenal axis and psychoneuroimmunology

Creed et al (1988) showed severe life stress occurring immediately before the onset of FGID. In fact, longitudinal data reveal that chronic highly threatening stressors predicted IBS symptom intensity and clinical outcome, to a greater extent than the IBS symptom intensity predict stress.

Table 2. Rome II diagnostic criteria for different types of IBS

Diarrhoea-predominant IBS	Usual frequency of stool more than three times per day
	OR usual form of stool is loose
	AND not hard
	OR frequently feel the sense of urgency
	AND do not strain at the stools
Constipation-predominant IBS	Usual frequency of stool < three times per week
	OR usual form of stool is hard
	AND not loose
	OR often strain at the stools
	AND do not frequently feel the sense of urgency
Non-specific IBS	Those who do not fulfil the above criteria, i.e. neither diarrhoea- nor constipation-predominant IBS

IBS = irritable bowel syndrome. From Drossman et al (2002)

Table 3. Alarm features

History	Blood in stool
	New onset after the age of 50 years
	Unexplained weight loss
	Persistent diarrhoea
	Severe constipation
	Nocturnal symptoms
	Family history of colorectal cancer, inflammatory bowel disease or coeliac disease
	Travel history to locations with endemic parasitic diseases
Physical examination	Fever
	Abdominal mass
	Fecal occult or overt blood on rectal examination
	Evidence of anaemia
	Signs of bowel obstruction
	Signs of thyroid dysfunction
	Signs of malabsorption
	Active arthritis
	Dermatitis
Initial laboratory tests	Anaemia
	Leukocytosis
	Elevated erythrocyte sedimentation rate or C-reactive protein level
	Abnormal chemistries
	Abnormal thyroid-stimulating hormone

From Lucak (2004)

As mentioned above in neuroimaging studies, IBS patients were found to be differentiated from healthy controls by showing greater activation of an area of the brain essential for conscious pain (the anterior mid-cingulate component of the ACC). These effects may relate directly to both the hypersensitivity and hypervigilance associated with this disorder (Mertz et al, 2000). It is in this region in the brain where the systems concerned with emotion or feeling (e.g. the amygdala), attention and working memory interact.

This affects the endogenous corticotrophin-releasing factor (CRF) in the brain, which is the major mediating mechanism involved with the body's stress response system in which gastric emptying is inhibited (with possible loss of appetite). The inhibition of gastric emptying by CRF may be mediated by interaction with the CRF-2 receptor, while CRF-1 receptors are involved in colonic and anxiogenic responses to stress. Endogenous serotonin, peripherally released in response to stress, seems to be involved in stress- and central CRF-induced stimulation of colonic motility by acting on serotonin (5HT-3) receptors (producing a loose stool or a sensation of bowel urgency).

Altered stress responses of this kind modulate the responsiveness of the CNS that may modulate the immune response of the gut to infectious agents. In turn, gut-directed physiological stressors alter outputs of central stress circuits, such as the hypothalamus–pituitary–adrenal (HPA) axis and the sympathetic arousal of the ANS. In this way a variety of neuroimmune responses can lead to intestinal over-responsiveness (sensitization) and other clinical effects. These responses include direct toxicity to nerves that influence intestinal contractions, alteration in gut immune activation, abnormalities of serotonin metabolism, and persisting low-grade inflammation. For example IBS developing after infective gastroenteritis is associated with subtle increases in enteroendocrine and chronic inflammatory cells in the gut mucosa. The net effect may be to increase serotonin availability in the gut, and enhance secretion and propulsive motility patterns.

This can further produce effects of inflammation on the gut ENS, specifically involving the mast cells. In ENS tissue mast cells are found to accumulate around nerve endings of nerves that contain the neurotransmitter serotonin. The release of substances that can induce activity in excitable tissue (i.e. histamine, interleukin-1 (IL-1), and bradykinin) by mast cells can affect receptor and neurotransmitter function in the ENS. In other words, when mast cells in the intestinal lining empty their contents in response to inflammation, they activate nearby nerve endings, causing diarrhoea, abdominal discomfort and pain (Chadwick et al, 2002).

Similarly cytokine-mediated activation of neurons conveys messages from tissue to the brain (afferent neurons) through the vagus nerve. The vagus has both sensory and motor fibres that innervate nearly every internal organ. The gastrointestinal tract is richly supplied with vagal afferents that can signally affect its function. Such effects include symptoms of fever, increased sensitivity to pain, loss of appetite and decreased desire for social interaction. The process may provide the basis for a role of the vagus as an interface between the immune response and the brain that results in symptoms of altered mood, including anxiety or depression, that are associated with gastrointestinal disease (Bluthe et al, 1994).

This illustrates how, in the biopsychosocial model, illness is viewed as a multifactorial entity resulting from interaction of systems at cellular, tissue, organism, interpersonal and environmental levels. In such interacting systems, events do not occur in isolation.

Management

Having made the diagnosis, reassurance and explanation are vital, including frank explanation of the likely course of the disease. Many patients may have a fear of cancer, but only careful and often repeated explanations of the nature of the disease reduce this.

Make a full assessment of psychological and social factors as well as physical symptoms. Most effective management involves treating specific symptoms (e.g. diarrhoea, constipation) and/or modifying the central appreciation of pain (e.g. antidepressants, psychotherapy).

Of immediate relevance to clinicians is the relationship between high number of consultations and psychiatric comorbidity. Patients who consult frequently should alert the physician to check for comorbid psychiatric disorders and the possibility of sexual abuse. The physician may wish to set aside a specific period of time in the clinic to see such an individual and attempt to develop a rationale for referral to psychiatric services. Remember the presentation blind spot, which implies that if you don't look for psychiatric factors, you may miss them. Patients with diarrhoea who have failed to respond to conventional treatment should also receive a careful assessment to detect anxiety or depression.

Food intolerance is common with IBS (33–66%) although true allergies are rare. The literature suggests omitting any known food triggers, but a formal exclusion diet needs the support of a committed dietician. Lactose intolerance occurs in 10%, but a lactose-free diet often has no effect on symptoms (Gunn et al, 2003).

Constipation-predominant (IBS-C)

Encourage patients to eat at regular intervals and increase exercise. Increase dietary insoluble fibre (e.g. wholegrain cereals) and soluble fibre (other cereals, fruit and vegetables), but be aware that increasing the latter may increase bloating and wind. Ispaghula husk (soluble fibre – bulking agent) has been shown to increase stool frequency (better tolerated than wheat bran) and improves overall symptoms and IBS-related constipation (insoluble fibre only helps the constipation). Supplement if necessary with an osmotic laxative (e.g. lactulose or magnesium hydroxide) or stool softener (liquid paraffin) – avoid stimulant laxatives as these can cause cramping pains.

Selective serotonin-4 (5-HT4) receptor agonists have prokinetic activity for short term use in IBS-C. They appear to improve overall symptomatology, but there are few data on their effect on quality of life.

Diarrhoea predominant (IBS-D)

Symptoms may respond to a low fat diet, reduced caffeine intake and stopping smoking. Some fibre helps, but the patient may need to reduce a high fibre intake.

Loperamide (opioid analogue) reduces stool frequency and urgency, and improves stool consistency without the sedation and drug dependency of codeine. Titrate the dose carefully to avoid constipation. Low dose tricyclic antidepressants help pain, and as they also tend to cause constipation, they may have particular benefit in IBS-D. Here too a selective serotonin 3 (5-HT3) receptor antagonist is being developed and is showing promise as a potential future pharmacological treatment.

Abdominal spasms

Smooth muscle relaxants (e.g. mebeverine and alverine) appear to improve global rating of symptoms and reduce pain in IBS patients. They have relatively few adverse effects but only benefit some patients. Antimuscarinics (hyoscine and dicycloverine/dicyclomine) are occasionally helpful, but anticholinergic side-effects limit their use.

Peppermint oil is commonly recommended as a smooth muscle relaxant. It may help symptoms of abdominal pain, distension and stool frequency, but more evidence is needed. Transcutaneous nerve stimulation (TENS) may be effective for pain, but usually has no effect on other symptoms.

New drugs, such as cholecystokinin, neurokinin and corticotropin receptor antagonists, are being developed.

Psychotherapies

The most important aspect of treatment is the patient's acceptance of the need for treatment and his/her motivation to engage in it. This can be enhanced if the gastroenterologist and psychologist or psychiatrist help the patient accept the treatment as part of an overall plan of care.

Empirical support for psychotherapies

Most of the research to date involved various combinations of cognitive-behavioural, relaxation, hypnosis, psychodynamic and biofeedback approaches, making assessment of the effectiveness of the specific approaches difficult. In terms of reduction of bowel symptoms at the end of treatment, 10 of 13 studies showed significant superiority of psychological over conventional medical treatment. Of the nine studies with follow-up data (duration 9–40 months), eight showed superiority of psychological treatment. Therefore, psychological treatment appears superior to conventional medical treatment and there were no differences in outcome based on technique. The psychotherapist should use the technique with which he/she is most experienced.

In addition, people with IBS should engage in regular physical exercise. This helps relieve the symptoms of anxiety and also promotes good bowel function. In general efforts should be made to deal with any stresses that may be contributing to the problem and in people without marked psychiatric abnormalities, consider relaxation therapy, biofeedback and hypnotherapy.

Anxiety and depression is co-morbid in 20–60% of IBS patients, so where there is marked psychiatric illness, cognitive-behavioural therapy, dynamic psychotherapy and psychiatric referral may be more appropriate (Drossman et al, 1999).

Psychopharmacotherapy

Silk (2003) describes that 'as well as treating the underlying depression, some antidepressants have been shown to modify gut motility and alter visceral responses. The tricyclic antidepressant imipramine has been shown to normalize the rapid small-bowel transit in diarrhoea-predominant IBS patients, possibly by reducing the propagation velocity of the phase III of the MMC. The converse is true for paroxetine (a selective serotonin re-uptake inhibitor), which shortens orocaecal transit time in patients with IBS, again presumably on account of its direct mode of action

on small intestine motility, which is to reduce the duration of the phase III MMC and increase its propagating velocity. Importantly, the end-organ effects of the two drugs are seen long before any effect on mood.'

Low doses of amitriptyline and particularly clomipramine at night can be highly effective in combating urgency and frequency, and are particularly effective at alleviating the 'early morning rush'. Song et al (2005) also illustrated that in patients with sleep disorders, the administration of melatonin significantly diminished abdominal pain and rectal urgency.

Further the rationale for using psychotropic agents lies in the high comorbidity: roughly half of the patients with a FGID also have depression and/or anxiety disorders which may respond to psychopharmacological intervention. Data supporting the efficacy of antidepressants in FGID is growing. At least five studies have shown efficacy that is independent of change in mood, or anticholinergic effects. Prescribing psychopharmacological agents is best accomplished in the context of a strong doctor–patient relationship, where they complement an overall multidisciplinary treatment plan. The physician needs to explain the rationale, possible side effects, and expected benefits from the medication and address them in the context of the patient's beliefs and expectations relative to psychopharmacological treatment.

The response to antidepressant therapy is highly patient specific – side effects and therapeutic effects vary across individuals, making sensible change of drugs appropriate. However, it is better to ensure consistent treatment at an appropriate dose over a longer period of time (2–3 months) than change rapidly from one drug to another.

Anxiolytic agents are effective for reducing anxiety in the short term, but their CNS depressant effect, including mild transient cognitive dysfunction and the risk of addiction with the benzodiazepines, leads to the recommendation that a psychiatrist be consulted to evaluate patients before prescribing benzodiazepines on a long-term basis. Alternative strategies for the treatment of anxiety should be used.

The therapeutic value of the medical interview

Data from primary care studies now show that active listening, facilitating the individual patient's expression of thoughts and feelings, and working together on a mutually agreed plan of care is associated with greater patient satisfaction, adherence to treatment and symptom reduction. It also leads to improvement in health outcomes and reduced health-care utilization.

Drossman (2001) provides some good guidelines for physicians on how to more effectively implement a physician–patient interaction, which include:

1. Listen actively
2. Be aware of questioning style and non-verbal messages. 'It's not what you say, but how you say it that makes the difference'
3. Elicit, identify and communicate the agenda(s) (both the patient's and the physician's) and work toward a mutually specified set of goals
4. Acknowledge the pain and provide empathy
5. Validate the patient's feelings
6. Don't overreact
7. Educate (i.e. elicit the patient's understanding, address misunderstandings, provide information that is consistent with the patient's frame of reference or knowledge base, and check the patient's understanding of what was discussed)
8. Reassure (identify the patient's worries and concerns, acknowledge or validate the concerns, respond to specific concerns, and avoid 'false' reassurances, e.g. 'Don't worry, everything's fine', particularly before the medical evaluation is complete, since this may be viewed as a doctor's lack of commitment)
9. Negotiate
10. Help the patient take responsibility (e.g. rather than asking a patient: 'How is your pain?' one might ask: 'How are you managing with your symptoms?' The former question tends to leave the responsibility for dealing with pain with the physician, while the latter acknowledges the patient's role)
11. Establish boundaries (this would include setting time limits and guidelines for narcotic use).

Conclusions

In the specialist gastroenterology clinic, the response 'I am pleased to be able to tell you there is nothing seriously wrong with you' should no longer be acceptable. Referred patients should be assessed properly after organic disease has been excluded, and should have access to a patient-focused multidisciplinary care team, including the gastroenterologist, a dietician, a clinical psychologist and, if indicated, a psychiatrist. Health-care managers should be alerted to these future needs, which should not surprise them, as 40–50% of all patients seen by gastroenterologists in their clinics have IBS.

Current symptom-based diagnostic criteria recognize that IBS is a group of functional bowel disorders sharing some prominent clinical features rather than one single disorder. The similar bowel symptom expression may be a common final pathway resulting from the limited number of perceptual (pain, discomfort) and behavioural (bowel movements) responses to gut stimulation, regardless of the underlying mechanisms. Several pathophysiological mechanisms directly related to gastrointestinal functioning have been postulated.

A likely unifying hypothesis is that IBS results from a dysregulation that may occur at any level of the interactions, both within the bidirectional brain–gut axis and between this and other physiological (direct) and psychosocial (indirect) systems (*Figure 1*). From a psychological perspective, IBS may be conceived as a summarization process. In particular, it may be viewed as an abnormal cognitive processing (illness belief) of emo-

tional and visceral stimuli, a tendency to perceive somatic stimuli as evidence of symptoms of disease and to seek repeated and often unnecessary medical care.

Psychosocial factors such as somatosensory amplification, psychopathology (anxiety, mood, and somatoform, disorders), past and/or current life stress (e.g. sexual and physical abuse, parental reinforcement of sick role in early life), social support, persistent summarization, and alexithymia interact with the brain–gut system and contribute to stepping up from gut sensations to IBS symptoms. Degree of quality-of-life impairment, abnormal illness behaviour and belief, and concurrent psychopathology may mediate the referral to primary (mild IBS, higher prevalence) or tertiary (severe IBS, lower prevalence) care settings.

This review has indicated the importance of biopsychosocial variables in IBS but further research is required in the use of well-designed, randomized, controlled psychological and psychopharmacological treatment trials with standardized assessment to further understanding and determine treatment effects on symptoms mediated by changes in gut and CNS physiology. BJHM

Conflict of interest: none.

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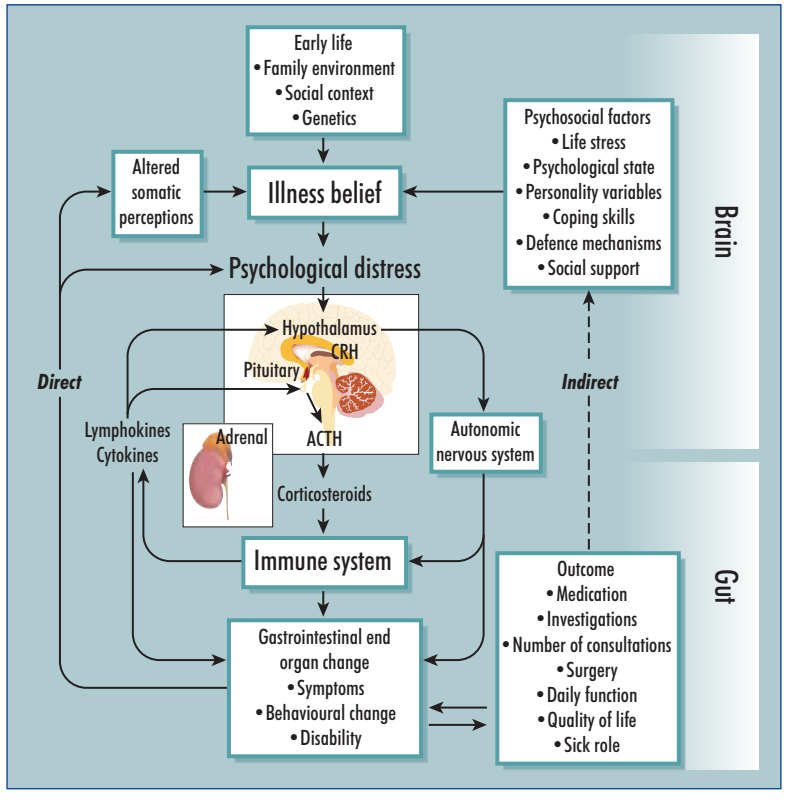
Figure 1. The brain–gut axis. ACTH = adrenocorticotropic hormone; CRH = corticotropin-releasing hormone.



KEY POINTS

- The psychiatric co-morbidity in functional gastrointestinal disorders is very high.
- In patents who present with physical complaints remember the 'presentation blind spot' – if you don't look for psychiatric factors, you may miss them.
- Ongoing research is improving our understanding of the pathophysiology of irritable bowel syndrome and is opening up greater treatment options.
- In irritable bowel syndrome mental states affect physical states, as physical states affect mental states via brain–gut axis dysregulation through a psychoneuroimmunological mechanism.
- For effective management patents should have access to a patient-focused multidisciplinary care team addressing both physical and psychological aspects of the disorder.
- End-organ effects of antidepressant treatment in irritable bowel syndrome are seen before their effect on mood, implying a direct impact on abdominal symptoms.
- The most important aspect for treatment is the patient's acceptance of the need for treatment and his/her motivation to engage in it.