Digestive diseases
Functional gastrointestinal disorders (FGID)

The most common = functional disorders of the oesophagus, irritable bowel syndrome

Prevalence: 10-20% (developed countries)!
FGID criteria

The symptoms should have appeared at least 6 months before and should have been active for at least three months.

Few of the affected individuals seek for help. However, as many as 2,2 mil. prescriptions / year are filled with a FGID diagnosis (USA), with important consequences on health costs and work force.

More frequent in women.
Etiology and pathogeny

1. neurohormonal factors:

- lower pain threshold (more frequent in women; could explain their more frequent appeal to doctor help);
  would be connected to the impairment of the downregulation mechanism of pain perception and its replacement with a positive feedback mechanism;

- the involvement of the central nervous system is proven by:
  - EEG changes at FGID patients;
  - connection of FGID to being awake.

- hormones involvement: malfunction of the hypothalamo-pituitary-corticosuprarenalian axis (high levels of ACTH = connected to high digestive motility).
2. psychosocial factors:

- low social acceptance of FGID:
  Patients with FGID are often not taken seriously. They fear to come to the doctor, as a common myth regarding them says that they typically exaggerate symptoms or / and attempt to obtain a benefit from their disease.

- a pathogenic role is played by the lack of perceived social support (especially daily quarells are associated with a higher prevalence of FGID).
Etiology and pathogeny (III)

3. psychoindividual factors:

- high anxiety, alexithymia:
  These patients tend to amplify or to interpret in a wrong way their somatic sensations, or to have difficulties in disentangling normal body reactions to emotional circumstances from abnormal ones;

- coping style: inefficient, dominated by hypochondria, phobias and somatic preoccupations.
  A common coping mechanism is avoidance, this leading to catastrophic beliefs and a low feeling of control over symptoms. Both play a key role in triggering depression on a long run.
Etiology and pathogeny (IV)

4. genetic vulnerability:

- there is a literature consensus over the possibility of FGID aggregation within the same family;

- confusing factor: shared diet routines.
Common myths

1. "FGID are always correlated to depression or other psychiatric diseases". In fact, if psychiatric symptoms occur they rather stem from FGID than cause FGID.

2. "FGID patients are difficult". If so, this is also caused by stigmatization, labeling or not being taken seriously. In fact, some patients have a history of abuse, with a direct implication on the onset of FGID. Typically these patients do not complain more than other patients (self blame, self-silence).
Difference FGID - somatoform disorders (SFD):

SFD = multiple, recurrent, variable somatic symptoms, present for more than 2 years, not intentionally produced or feigned, not explained by an organic disease.

- association of:
  - 4 pain symptoms: a history of pain related to at least 4 different sites or functions (head, back, joints, thorax, etc.);
  - 2 gastrointestinal symptoms other than pain (e.g. nausea, bloating, vomiting, etc.);
  - a sexual symptom other than pain (e.g. sexual indifference, impotence, ejaculatory dysfunction);
  - a pseudoneurologic symptom other than pain (paralysis, localized weakness, deglutition difficulties, etc.)
Examples of FGID

1. Gastroesophageal reflux disease (GERD)

Gastric acid or, occasionally, gastric content, flows back into esophagus, generating most typically heartburn and interference with daily life and habits (threshold: twice a week). Modifications of esophageal mucosa are common in advanced GERD (esophagitis). A subcategory of patients do not have esophagitis, however they complain of an "oversensitive esophagus".
Examples of FGID (II)

2. Irritable bowel syndrome (IBS)

- > 12 weeks of abdominal discomfort / pain, with irregular changes in peristalsm.

- brain-gut axis;

- feelings of shame and fear: self-fulfilling prophecy
GERD and IBS are often associated.

(a) Same prevalence in developed countries: 10–20%.

(b) Shared physiopathological mechanisms:

- reduced pain or discomfort threshold in response to visceral stimulation;

- motor abnormalities (overall dysfunction of smooth muscle throughout the GI tract);

- central neural mechanisms (e.g. anxiety would modulate the degree of responsiveness / sensitization of the GI tract);

- similar response patterns to psychototropic drugs that modulate visceral pain: e.g. tricyclic antidepressives, SSRI.
Treatment

Difficulties:

- polymorphic symptoms and report of symptoms (need for personalization of therapy) (difficulties in doing statistics and drawing conclusions);

- lack of a precise understanding of mechanisms responsible for symptoms (i.e. how much is psychoindividual / psychosocial);

- overlap with other diseases (e.g. pure FGID vs. SFD).
1. Pharmacological treatment

Disadvantages:

- is strictly centered on symptoms and rather unselective (e.g. proton pump inhibitors can dramatically decrease gastric secretion, on the expense of increasing the risk of gastric cancer);
- can create psychological addiction, thereby increasing the feeling of invalidity.

However, some psychotropic drugs, e.g. tricyclic anti-depressives, SSRI, offer a window of opportunity, by acting both on psychological and somatic symptoms, as they target cholinergic / serotoninergic receptors all over the body.
2. Psychotherapy

- relaxation: progressive muscle relaxation, exercises centered on respiration, meditation;

- hypnotherapy: acts positively on visceral motility, but also on self perception, self-esteem and on the conviction that symptoms can be controlled.
Cognitive-behavioral therapy

- change of irrational thoughts regarding the disease (e.g. "my disease will never heal"), the ability to cope (e.g. "I can not do anything to feel better") or being the bearer of the disease (e.g. "everybody perceive me as being weird").

- passing from avoidance to a more active behavior, exploring new ways to cope with daily stress, working on (re)establishing personal relationships, progressive exiting from the sick role.

Results = more positive at patients open to the idea that the psychological factors could have played a role in their illness and willing for change;
= weaker at patients with psychiatric comorbidity (e.g. severe anxiety) and in those patients that have frequent (daily) symptoms.