The bile acid (BA) pool size is preserved in the enterohepatic circulation (EHC) by means of an efficient intestinal absorption\(^1\).

The BA, which enter the small intestine after gallbladder emptying, take part in the digestion and absorption of fat and fat-soluble vitamins in the jejunum and proximal ileum; then they are absorbed, mainly in the distal ileum\(^2,3\).

Two modalities of bile acid absorption have been identified: passive absorption and active absorption, the former mechanism operating in the proximal small intestine and the colon, the latter accounting for the absorption in the distal ileum.

As far as the passive absorption mechanism is concerned, this inversely correlates with the number of hydroxyl groups and the degree of ionization\(^4,5\).

The hydroxyl groups increase the binding of BA to water; therefore, the partition coefficient of the BA into the lipid membranes of the cells is decreased.

Passive ionic diffusion now is thought to account for little of the total BA absorption, while the protonate molecule, i.e. non-ionized, is considered the major determinant of the passive absorption.

Therefore, since the \(\text{pK}_a\) of unconjugated BA present as 'monomers' is about 5, i.e. more similar to the pH of the intestinal lumen than that of the conjugated forms, unconjugated BA, mainly dihydroxy, are candidates for passive absorption.

The active BA intestinal transport is operating in the terminal ileum, though the exact mechanism is not yet known and its histochemical localization is still to be fully determined.

Active absorption follows Michaelis–Menten kinetics\(^6\): \(V_{\text{max}}\) increasing with the number of OH groups, \(K_m\) being, on the contrary, related to conjugation, either with taurine or glycine\(^7\).

The colon helps maintain the enterohepatic circulation of BA by means of passive absorption\(^8\): its contribution to the total absorption may be important in cases of BA malabsorption (BAM) in the small intestine\(^9\).
BILE ACID MALABSORPTION

In health, only about 10-15% of the total BA pool is lost daily in the faeces; in presence of interruption of the EHC, with or without mucosal damage, the BA are malabsorbed\(^{10}\).

Various conditions have been so far described in association with BAM, though a cause-effect relationship has not been identified for each of them; three types of BAM have been listed\(^{11}\): the first is due to ileal resection or disease\(^{10,12,13}\); the second is regarded as a primary BAM; the third is found in association with clinical conditions, such as cholecystectomy, fenformine treatment, or renal failure\(^{11}\).

The first type of BAM, which is pathognomonical for the disease, is related either to Crohn’s disease or to ileal resection: the severity of the malabsorption depends on the extent of the ileal involvement.

The second type of BAM, firstly described by Thaysen and Pedersen\(^{14,15}\) and later studied by us\(^{16}\), seems to be related either to an increased BA synthesis rate, overloading the ileal transport system or to impaired intestinal absorption of a normal BA load, due probably to a defect in the carriers in the ileal mucosa.

The third type has been found in uraemic patients\(^{17}\), patients on biguanydes therapy, though this second case has not been confirmed, and in cholecystectomy, possibly because of the increased recycling frequency of the BA pool.

Since ileal resection has been almost always found in association with changes in the enterohepatic circulation of bile acids at the level of intestinal absorption, two syndromes have been described, depending upon the length of the resection\(^{10}\):

(1) Bile acid diarrhoea: the increased concentration of bile acids in the colon induces diarrhoea in resections under 100 cm. In this syndrome usually none or slight steatorrhoea is present (8-20 g/day); BAM is present, and hepatic bile acid synthesis is increased\(^{18}\) to maintain a normal or only slightly decreased jejunal bile acid concentration. The concentration of the bile acids in the faecal water is increased; in particular, dihydroxy bile acids are present in concentrations similar to those which have been shown to cause water and electrolyte secretion during colonic perfusion in man\(^{19}\). Cholestyramine\(^{20}\) is considered to have abolished diarrhoea by binding BA, thus preventing their secretory effect in the colon. Although a cholestyramine-induced steatorrhoea may develop, this is usually of no caloric or symptomatic consequence\(^2\).

(2) Fatty acid diarrhoea: in cases of resection over 100 cm, steatorrhoea is usually severe (> 20 g/day); bile acid malabsorption is severe and jejunal bile acid concentration is usually decreased, despite an increased BA synthesis rate. Because of a decreased bile acid secretion, the concentration of the BA in the faecal water of colonic content is normal and bile acids do not induce water secretion in the colon. In this case, hydroxy fatty acids may induce water and electrolyte secretion in the colon\(^{22,23}\). Cholestyramine treatment is of no sympto-