phagocytosed. This means most of human iron homeostasis is dependent on iron recycling [4]. Tight regulation of iron metabolism is imperative for its homeostasis.

This review assesses the roles of the intestine, the bone marrow, the spleen, and the liver in iron metabolism. Furthermore, a consideration of tissue-specific pathological situations and their effects on iron homeostasis is presented. Finally, management of these iron-associated disorders is included.

2. Intestine

from A Short Review of Iron Metabolism and Pathophysiology of Iron Disorders.2019

An average western-world diet includes the ingestion of about 15–20 mg of iron, 10% of which is present in the haem form and the remainder in the non-haem/ionic form. Only about a tenth of the consumed iron is absorbed, predominantly within the duodenum and lesser amounts in the jejunum [2]. Most of the ingested haem-iron is found in haemoglobin and myoglobin of meat proteins. The low gastric pH releases these proteins from meat, and subsequent protease action in the stomach and the intestine releases free haem [5]. It has been proposed that enterocytes absorb haem via the Haem Carrier Protein 1 transporter (HCP1) localised on their brush-border membrane [6]. Evidence from a later study showed that this protein is a proton-coupled folate transporter (PCFT), and therefore the acronym PCFT/HCP1 is sometimes used for the transporter [7]. Once haem enters the enterocyte, it can be degraded via the action of haem oxygenase (HO-1) to release free iron, which joins the intracellular labile iron pool (LIP), as depicted in Figure 2. Moreover, intact haem may also be absorbed into the circulation via two exporter proteins found on the enterocyte basolateral membrane: the breast cancer resistant protein (BCRP) and the feline leukaemia virus subgroup C (FLVCR) [8].

need for acid