

provide preliminary evidence that  $Mg^{2+}$  and  $Mn^{2+}$  compete for the same binding sites located on the inner mitochondrial membrane. This assumption explains the inability of  $Mg^{2+}$  to act once  $Mn^{2+}$  has been added to the mitochondrial suspension.

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## S8

### Iron Transport and Accumulation in Mammalian Cells

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Iron is essential for all cells and mammalian cells are no exception. The daily requirements for iron are provided to a small extent from absorption of dietary iron essentially in the upper part of the intestine, to a large extent mediated by mucosal transferrin, and by reutilisation of tissue iron, to a major degree furnished by the breakdown of effete red blood cells, and assured by the serum  $\beta$ -globulin transferrin. In fact of the 35 mg or so of iron which exchanges between different body compartments each day, only 1 mg is contributed by mucosal iron absorption, and this latter is compensated by an equivalent amount of iron excretion (also about 1 mg/day). We shall concern ourselves here with three aspects of iron metabolism in mammalian cells: (i) iron uptake by cells from serum transferrin (ii) accumulation of iron in the intracellular storage protein ferritin (iii) intracellular transport of iron to sites where it is required for essential cellular functions such as haem synthesis.

*Iron uptake from serum transferrin.* It has been well established for many years that many mammalian cells (indeed probably all cells) have plasma membrane receptors for transferrin, and the transferrin receptor has recently been purified and characterised [1]. It is an integral membrane protein of MW 180,000 composed of two disulphide linked subunits which are each capable of binding one transferrin molecule (MW 80,000 bearing one or two iron atoms). Subsequent to binding to its receptor, the transferrin-receptor complex is internalised by endocytosis and its iron is released by protonation of the bicarbonate (or carbonate) anion bound to the iron atom [2]. The apotransferrin molecule is recycled to the plasma membrane and released for reutilisation. In some cell types an additional mechanism intervenes in which the transferrin molecule is digested in lysosomes subsequent to iron release.

*Accumulation of iron in ferritin.* The intracellular iron storage protein is ferritin which provides a bio-available, soluble and non-toxic form of iron essentially as hydrolysed ferric oxyhydroxide enclosed within a globular protein shell (apoferritin) of MW 480,000. The amino acid sequences of human and horse apoferritins have been determined [3, 4] and the X-ray structure of horse spleen protein is well advanced [5]. The mechanism of iron deposition involves catalysis by the protein of the oxidation of Fe II bound to adjacent subunits, which may involve peroxo-bridged and  $\mu$ -oxo intermediates prior to hydrolysis. Subsequent events may occur at the polymer surface or be catalysed by the protein and our current understanding of the incorporation of iron in ferritin will be reviewed.

*Intracellular Iron Transport for Haem Synthesis.* The ferriochelate of the mitochondrial matrix catalyses the final step in haem biosynthesis, namely the incorporation of Fe II into protoporphyrin IX. Recent studies suggest [6, 7] that ferritin may well be the source of ferrous iron for haem synthesis via reduction of ferritin iron by reduced flavins generated by electrons from the respiratory chain, and implying a rather specific interaction of ferritin with the mitochondria. Recent results in this area will be presented and the role of ferritin in intracellular iron transport will be reviewed.

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## S9

### Vanadium Absorption by Plants: the Uptake of Vanadium by Excised Barley Roots (*Hordeum Vulgare* c.v. *Maris Mink*)

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The absorption of vanadium by plants has received only limited attention [1]. However it presents an