The protein Ceruloplasmin (Ferrooxidase) is a multicopper ferroxidase (MCF) which is necessary for the efflux of iron from cells. Present in the body in two forms, ceruloplasmin 2 and glycosylphosphatidylinositol (GPI), they are also the primary copper carrying protein in blood plasma. Ceruloplasmin 2 is produced by hepatocytes and reticuloendothelial cells in the liver, and GPI by the glia cells in the nervous system (NLM, 2013; Ramos, 2016; Miyajima, 2015).

**Aceruloplasminemia**

The absence of functional ceruloplasmin ferroxidase activity impairs the body’s ability to export iron out of its tissues. The resulting accumulation of iron in the cells result in tissue damage and ultimately health concerns such as aceruloplasminemia, Parkinsonism, and multiple system atrophy (Miyajima, 2003) (Motta, 2009). While not directly affected by mutations in the CP gene, individuals with copper-transport diseases such as Wilson disease and Menkes’ disease may exhibit low levels of Ceruloplasmin (Mercer, 2001).

Aceruloplasminemia is an autosomal recessive disease which occurs when iron accumulation commonly occurs in the pancreas, liver, and brain (Miyajima, 2003) (Motta, 2009). Iron build up in the pancreas impairs the production of insulin, often leading to the development of diabetes. Individuals are also prone to anemia due to a deficiency in red blood cells from an iron shortage in the blood. Other symptoms include non-vision affecting changes to the retina. Prolonged deposition of iron in brain tissue often leads to neurological problems such as ataxia, dystonia, chorea, blepharospasm, and dementia (NLM, 2013; Motta, 2009).

The primary treatment method for aceruloplasminemia is to continuously monitor and manage iron levels in the blood through the use of chelating agents such as Deferasirox (Roberti, 2011).

**Comparative homology map of human ceruloplasmin with protein orthology found in wild bour (Sus scrofa) and Chinese tree shrew (Tupaia chinensis)**

**Fig 2A: Comparative study of upper abdomen MRI shows liver parenchyma with an increased signal in the left image due to iron deposits. On the right, after six months of treatment with Deferasirox, a reduction in iron was observed by the MRI signal attenuation (Roberti, 2011).**

**Fig 2B: Comparative study of brain MRI before (left) and six months after starting treatment with Deferasirox (right) shows no change in the signal of basal ganglia structures (Roberti, 2011).**

References:


