Hepcidin is emerging as an important peptide for brain (patho)physiology.The local molecular regulation of brain hepcidin expression is unfolding, while studies suggest that it bears resemblance with liver hepcidin regulation.The observation of robust production of hepcidin by glial cells compared to neurons during changes in brain homeostasis is in-line with the already established role of glia as supportive, regulatory and protective cells in the brain milieu.This occurs due to the increase in iron import caused by inflammation, which is further exacerbated by FPN downregulation caused by hepcidin-dependent and hepcidin-independent mechanisms.Unfortunately, therapeutic implications of this physiological effect of hepcidin have not been studied in details, despite its potential crucial importance in brain and other pathologies.Intriguingly, hepcidin pretreatment also protects neurons from the deleterious effects of inflammation.Still, pharmacologic manipulation of hepcidin is emerging as e new therapeutic territory in neurodegenerative diseases.Neurons, glial cells, endothelial cells, and other brain cells may express hepcidin, although basal levels of brain hepcidin are low.Hepcidin produced by the brain cells in pathological conditions may limit iron transport through BMVEC, and thereby also limit neuronal iron load.High iron load is detrimental for neuronal function.