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Regulation of iron uptake in primary culture rat hepatocytes: The role of acute phase cytokines

Shakil Ahmad
Georg-August University Goettingen, Germany

Decreased serum- and increased hepatic-iron-uptake is the hallmark of acute-phase response (APR). Iron uptake is controlled by iron transport proteins like transferrin receptors (TfRs) and lipocalin-2 (LCN-2). The current study aimed to understand regulation of iron uptake in primary culture hepatocytes in the presence/absence of acute-phase-mediators. Rat hepatocytes were stimulated with different concentrations of iron alone (0.01 mM, 0.1 mM, 0.5 mM) and acute-phase-cytokines (IL-6, IL-1β, TNFα) in the presence/absence of iron (FeCl3: 0.1 mM). Hepatocytes were harvested at different time points (0h, 6h, 12h, 24h). Total mRNA and proteins were extracted for RT-PCR and Western-blot. A significant iron uptake was detected with 0.1 mM iron administration with a maximum (133.37±4.82 µg/g of protein) at 24 h compared to control and other iron concentrations. This uptake was further enhanced in the presence of AP-cytokines with a maximum iron uptake (481±25.81 µg/g of protein) after concomitant administration of IL-6+ iron to cultured hepatocytes. Concomitantly, gene expression of LCN-2 and ferritin-subunits (FTH, FTL) was up-regulated by iron or/and AP-cytokines with a maximum at 24 h both at mRNA- and protein-level. In contrast, a decreased TfR1 level was detected by IL-6 and iron alone whereas combination of iron and AP-cytokines (mainly IL-6) abrogated the down-regulation of TfR1. An increase of LCN-2 release into the supernatant of cultured hepatocytes was observed after addition of iron/AP-cytokines into the medium. This increase in secretion was further enhanced by combination of IL-6+iron. In conclusion: iron-uptake is tightly controlled by already present iron concentration in the culture. This uptake can be further enhanced by AP-cytokines, mainly by IL-6.

drshakilahmad@live.com