Iron-induced oxidative damage in colon carcinoma (Caco-2) cells

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Intestinal epithelial cells have an active apical iron uptake system that is involved in the regulated absorption of iron. By the action of this system, intestinal cells acquire increasing amounts of iron with time. Since intracellular reactive iron is a source of free radicals and a possible cause of colon carcinoma, this study analyzed the oxidative damages generated by iron accumulation in Caco-2 cells. Cells cultured with increasing concentrations of iron increased both total intracellular iron and the reactive iron pool, despite an active IRE/IRP system, which regulates intracellular iron levels. Increasing concentrations of iron resulted in increased protein oxidative damage, as shown by the immunoreactivity for 4-hydroxy-2-nonenal-modified proteins, and markedly induced DNA oxidation determined by 8-hydroxy-2?-deoxyguanidine production. Iron also impaired cell viability, resulting in increased cell death after 6 days of culture. In summary, iron accumulation by intestinal Caco-2