Role of redox systems on Fe3+ uptake by transformed human intestinal epithelial (Caco-2) cells

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Caco-2 cells were used as a model of human intestinal epithelium to investigate the role of redox systems in transepithelial transport of 59Fe3+. The cells reduced Fe3+ present in the apical medium; the reduction was 50% inhibited by adriamycin and p-chloromercuribenzoate. Addition of [14C]ascorbate to the basolateral medium resulted in accumulation of 14C radioactivity in both cells and apical medium; apical radioactivity increased with time and was probably caused by paracellular flux. The cells provided Fe3+ reduction capacity to the apical incubation medium. Addition of ascorbate to the basolateral medium increased this reduc- tion capacity 2-fold and the cellular uptake of 59Fe3+ 1.8-fold. Adriamycin significantly inhibited both cellular 59Fe uptake and Fe transport into the basolateral side. The results indicate that Caco-2 cells reduce apical Fe3+ by two parallel mechanisms: by a plasma membrane ferrireductase and by the secretion of reductants of either cellular or basolateral