

Transferrin stimulates iron absorption, exocytosis, and secretion in cultured intestinal cells

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The cellular mechanism by which basolateral transferrin (Tf) produces an increase in apical-to-basolateral Fe flux in Caco-2 cells was analyzed. After a pulse of ^{59}Fe from the apical medium, three types of basolateral ^{59}Fe efflux were found: a ^{59}Fe efflux that was independent of the presence of Tf in the basolateral medium, a ^{59}Fe efflux in which ^{59}Fe left the cell bound to Tf, and a Tf-dependent ^{59}Fe efflux in which ^{59}Fe came off the cell not bound to Tf. Furthermore, addition of Tf to the basolateral medium doubled the exocytosis rate of Tf and increased the secretion of apolipoprotein A, a basolateral secretion marker. Both apotransferrin and Fe-containing Tf produced similar increases in ^{59}Fe efflux, Tf exocytosis, and apolipoprotein A secretion. The Ca^{2+} channel inhibitor SKF-96365 inhibited both the Tf-mediated increase in transepithelial Fe transport and the secretion of apolipoprotein A. Thus the activation of transepithelial Fe transport by Tf seems to be mediated by Ca^{2+} ent