Introduction:

Endocrine signals are signals from distant cells, which are from the endocrine cells located in the endocrine gland. The endocrine signals produce a slower response, but have long lasting effects. The endocrine signal released a hormone called insulin. The insulin hormone released by the endocrine signal helps to control the level of nutrients in the blood and also controls the uptake of glucose, fatty acid, and amino acid into the liver, tissues, and muscles. Insulin also helps in the storage of nutrients in the form of glycogen, lipids, and protein and failure to control the level of nutrients can lead to diabetes. (OpenStax College,n.d.)
The insulin hormone becomes responsible in transportation of the nutrients through the blood stream over a large distance between the endocrine cells and their target cells. Because of the large distance to cover, the hormones become diluted and present in low concentrations when acting on their target cells. (Hooper, 2019)

The prototypical metabolic effect of insulin is the stimulation of glucose transport in adipose tissue and skeletal and cardiac muscle. Glucose disposal into muscle is the major component of insulin action that prevents postprandial hyperglycemia. This is accomplished through the translocation by exocytosis of the insulin-sensitive glucose transporter GLUT4 from intracellular vesicles to the plasma membrane. (Hooper, 2019)

Calcium (Ca2+) is essential for numerous physiological functions, which include intracellular signalling processes, neuronal excitability, muscle contraction and bone formation. Through the coordination of the intestinal absorption, calcium homeostasis is maintained. Ca2+ makes its way into the endocrine system through TRPV5, Ca2+ bound to calbindin-D28K diffuses to the basolateral side, where it is extruded into the blood compartment through NCX1 and to a lesser extent PMCA1b. In the urinary compartment, both klotho and tissue kallikrein (TK) increase the apical abundance of TRPV5. In the blood compartment, PTH, 1,25(OH)2D3 and estrogen increase the transcription and protein expression of the luminal Ca2+ channels, calbindins, and the extrusion systems. (KEGG,n.d.)
Three other pathways involve:
Ko04080 Neuroactive ligand-receptor interaction
Ko04978 Parathyroid hormone synthesis, secretion and action
Ko04961 Endocrine and other factor-regulated calcium reabsorption
Disease:
H00680 Primary failure of tooth eruption
H00700 Centronuclear myopathy
H00784 Localized autosomal recessive hypotrichosis
H00998 Alternating hemiplegia of childhood
H01143 Vitamin D-dependent rickets
H01193 Familial tumoral calcinosis
H01593 Osteoporosis
H02026 Familial hypocalciuric hypercalcemia