Recap of adiposity signals and hypothalamic pathways.

- Leptin inhibits NPY/AgRP neurons. This decreases inhibition to the catabolic pathway and decreases excitation to the anabolic pathway. The net result is an increase in catabolic pathway activity relative to the anabolic pathway.
- Leptin stimulates POMC otherwise known as α-MSH/CART neurons. This increases excitation of the catabolic pathway, and it increases inhibition of the anabolic pathway. The result again is a net increase in activity of the catabolic pathway relative to the anabolic pathway.
- The net increase in catabolic activity means that leptin normally serves to decrease caloric consumption and increase energy expenditure.

Effects of leptin on hypothalamic neurocircuitry

Leptin deficiency causes increased excitatory inputs to the NPY/AgRP neurons, thereby increasing inhibition to the catabolic pathway and increasing excitation of the anabolic pathway. Similarly, lack of leptin caused decreased excitatory inputs to POMC neurons, causing decreased excitation of the catabolic pathway, and decreased inhibition of the anabolic pathway. Leptin repletion can reverse these synaptic changes.