CONCLUSIONS

1. Induction of obesity resistance in mice by transgenic expression of UCP1 in white fat can be explained by a metabolic switch in adipocytes, due to depression of cellular energy charge, in situ activation of AMPK, down-regulation of adipogenic genes and increase in lipid oxidation.

2. AMPK cascade is involved in the fat depot-specific metabolic responses in various fat depots to starvation. Activation of the cascade occurred in epididymal but not in subcutaneous fat in mice.

3. The activation of AMPK in adipocytes might represent an important mechanism by which body fat stores are regulated and may contribute to regional differences in the metabolic properties of adipose tissue depots.

4. Dietary EPA and DHA reduce development of obesity induced in mice by high fat diets, in part due to counteracting increase in tissue cellularity, particularly in epididymal fat. Low EPA/DHA ratio potentiates the anti-adipogenic effect.

5. Dietary EPA and DHA induce a metabolic shift in white adipose tissue by up-regulating genes for mitochondrial proteins, including their regulatory genes PGC1α and NRF-1, and increase ß-oxidation while depressing lipogenesis, preferentially in the epididymal fat in the abdomen.