Acyl-CoA thioesterases (Acots) are enzymes whose reactions are well defined, but their biological consequences are not. This mini-review focuses on one specific type of Acot that is enriched in brown adipose tissue, thioesterase superfamily member 1, Them1. The author, David Cohen, reviews a recent study demonstrating that mice lacking Them1 not only had an increase in energy expenditure, but also were resistant to diet-induced obesity and corresponding metabolic disorders. He places these findings in context with what is known about this gene family.

**OPA1 for Healthy Mitochondria pp. 7–11**

Mitochondria are very dynamic organelles that are central to cellular energy metabolism. For mitochondrial to function properly, a high amount of quality control is required to regulate the proper assembly and turnover of mitochondrial proteins. When this regulation is lacking, disease ensues. Quirós et al. give evidence for the function of OMA1 as a mitochondrial quality control protease in this mini-review and further expand on the role of the OPA1-OMA1 system in mitochondrial biology and metabolic homeostasis (Fig. 1).

**Natural Killer T Cells Add Insult to Injury for the Obese pp. 12–6**

Inflammation is a common thread between obesity and its associated medical problems. Cells from both the adaptive and innate immune system appear to regulate this inflammation. In this mini-review, Wu and Van Kaer look at the role of natural killer T (NKT) cells in the inflammation process. The authors tie together evidence from NKT cell-deficient animals, adoptive transfer of NKT cells, and iNKT cell agonists and discuss their results. Targeting NKT cells may be yet another line of attack in the fight against obesity related disorders.

**The Effect of Palmitoylated Proteins in Adipocytes pp. 17–27**

Protein trafficking and sorting is a requirement for successful Gluat4 membrane translocation, adipokine signaling and lipid production: all important functions in a healthy adipocyte. Protein palmitoylation is involved in this trafficking and sorting, but to what extent? Here, Ren, Ulupi and Du undertake proteomic analysis of palmitoylated proteins in adipose tissue and 3T3-L1 adipocytes. The group identified over 800 putative palmitoylated proteins, including those involved in membrane translocation of Gluat4. They also found some specific alterations of palmitoylation in the obese state, suggesting a novel role for protein palmitoylation in adipocytes and the pathogenesis of obesity (Fig. 2).

**Valsartan’s Fight Against Inflammation pp. 28–32**

Obesity often leads to chronic inflammation in adipose tissue that contributes to insulin resistance. Protection against
these two detrimental effects would be a windfall to health services overburdened by those suffering from obesity-related metabolic disorders. Fortunately, a series of angiotensin II receptor blockers (ARBs) has shown some benefit in improving insulin resistance in vitro. Iwashita et al. further studied the effects of valsartan, a widely used ARB, to find that it also suppresses macrophage inflammation. In this brief report, they take this one step further and look at the effects of valsartan in vivo. The authors found that administration of valsartan to mice infused with lipopolysaccharides were able to keep the expression of adipose and liver inflammatory cytokines in check. Valsartan could prove to be an effective way of not only combating insulin resistance, but also other metabolic disorders arising from chronic inflammation.

**Acetylation Affects Adipocyte Differentiation pp. 33–40**

What happens to a protein post-translationally can alter our metabolism. Acetylation is one way to modify proteins, however, little is known about how this process dynamically affects metabolism during adipocyte differentiation. Xu, Ande and Mishra take on this issue in this brief report, concluding that protein acetylation follows many different temporal patterns that lead to differentially expressed proteins all coming together to play important roles in adipocyte differentiation (Fig. 3).

**Silencing Fetal Adipokine Expression pp. 41–6**

Gestational diabetes mellitus (GDM) has a relatively high prevalence, affecting 2–10%, even 20% in some populations. This not only leads to an increased risk for type 2 diabetes in the mother, but also leads to an increased risk for metabolic disorders in the offspring. The group focuses on two adipokines that are altered epigenetically after exposure and how this affects fetal metabolic programming.

**Blocking Cannabinoid Receptors for Weight Loss pp. 47–9**

Obesity has a strong association with insulin resistance, especially if there is an abundance of visceral fat. This may be due to an increase in adipose-derived free fatty acids and adipokines that raise glucose production by their direct delivery to the liver. Interestingly, blocking receptors of the endocannabinoid system, specifically the CB1 receptor, can decrease the amount of visceral fat. In this commentary, Stella Kim looks at a recent study that uses a canine model to explore the role of continual CB1 antagonism and how this affects fat deposition and insulin sensitivity.

**To Be or Not to Be—An Osteoblast or an Adipocyte pp. 50–4**

In bone marrow, a dilemma exists of whether to become an osteoblast or an adipocyte as both cell types share a common progenitor cell. The influences that decide the progenitor cell’s fate are inversely regulated via crosstalk from different transcription factors. In this commentary, Baek and Baek look at the role of two specific factors, myeloid elf-1 factor (MEF) and distal-less homeobox 5 (Dix5), and discuss how their relationship affects cell type differentiation.

**Genomic Variation and Metabolism pp. 55–7**

It’s not always the amount of calories consumed vs. the amount of calories burned that dictate whether someone will become obese and develop metabolic syndrome; genetics can also play a large part. But what role does genomics and copy number variation play in the etiology of metabolic syndrome and obesity? Lacaria, Gu and Lupski explore their findings on this topic in this commentary. They further discuss the relevance of these findings on future metabolic studies.