In this issue of *Adipocyte*

Elaine Ellerton  
Landes Bioscience; Austin, TX USA

**When Food is Scarce, Cells Dine on Themselves**  
pp.75–9

Highly conserved mechanisms are in place to drive food-seeking behavior in organisms. When resources are scarce, similarly conserved mechanisms drive behavior at the cellular level. Depletion of nutrients drives cells to undergo autophagocytosis, a process that, in a sense, “eats” its own redundant cellular components. In this review, Rajat Singh discusses hypothalamic autophagy and its resulting effect on food intake and energy balance. Interestingly, eradicating hypothalamic autophagy reduces body fat, in rodents at least. Further exploration in this area may contribute to new therapies to help control obesity and insulin resistance in humans.

**Metabolic Health Depends on Adipocyte Size**  
pp. 80–8

White adipose tissue (WAT) is a dynamic entity, expanding and shrinking in order to keep up with the body’s metabolic needs. In fact, metabolic state can be ascertained by the size of individual adipocytes. This regulation of lipid storage and release is a complex process and too much or too little can lead to metabolic disorders. In this review, Jo, Shreif and Periwal explain how computational modeling of adipocyte size distributions, along with measuring metabolic states, can shed some light on how the dynamic characteristics of WAT can lead to obesity related metabolic disorders.

**Fat or Thin Results in Fibrosis**  
pp. 89–95

It is common knowledge that obesity contributes to cardiovascular disease, diabetes and cancer, but less known is that a loss of fat tissue, in the form of abnormal degeneration (lipodystrophy), can also contribute to these metabolic disorders. This phenomenon points to the importance of a balance between too much or too little adiposity. Interestingly, an excess or deficit of adipose tissue shares a common factor, an increase in collagen deposition, or fibrosis. Adipocytes are greatly influenced by their surrounding extracellular matrix (ECM) and because collagen is one of the largest members of this ECM, it is important to understand the molecular mechanisms through which this matrix regulates adipocyte function. In this review, Tae-Hwa Chun takes an in depth look at rodent models to help elucidate some of these mechanisms.

**Galectins to Treat Obesity**  
pp. 96–100

Taking in more calories while expending less creates a build-up of fat, which then puts strain on adipocytes, or fat cells. In their normal state, adipocytes help regulate energy storage and expenditure, but lipid-burdened adipocytes contribute to many metabolic disorders, including type 2 diabetes. Keeping lipid levels low in these cells is key to maintaining healthy energy homeostasis. In recent findings, Yang et al. located galectin-12, a member of the galectin family of animal lectins, to lipid droplets and observed its role as a regulator of lipid degradation. The authors focus on galectin, specifically galectin-12, and explore how these proteins may shed light on potential therapies for treating obesity-related metabolic disorders.

**Linking Uncoupling Protein Genes to Metabolic Syndrome**  
pp. 101–7

Westernized countries are not alone in the ongoing obesity epidemic, as India has now joined the ranks. In fact, Asian Indians are more susceptible to acquiring type 2 diabetes, cardiovascular disease and insulin resistance, metabolic disorders that accompany obesity, compared with their Westernized brethren. In this research paper, Mahadik et al. investigate this phenomenon by looking at uncoupling protein (UCP) genes as they are mapped to the same regions that are linked to obesity and hyperinsulinemia. The authors not only found a reduction in UCP2, a mitochondrial membrane transporter expressed in white adipose tissue, in obese and diabetic patients, but also found differences in expression between sexes, fat tissue type and associations with a number of parameters of the metabolic syndrome.

**Adenosine Receptor Mediated Metabolism**  
pp. 108–11

As the incidence of diabetes continues to rise in many countries in an unabated fashion, identifying treatments to control or eradicate diabetes is a hot topic. Using adenosine A1 receptor (A1AR) deficient mice, Robert Faulhaber-Walter describes findings that these mice not only gained fat, but also became insulin resistant.
They also found evidence for A1AR’s involvement in the metabolic regulation of adipose as well as in insulin-controlled glucose homeostasis and insulin sensitivity. Possible mechanisms for adenosine mediated central regulation are also suggested in this commentary.

**Another Member of the IL-6 Family Steps up to the Plate in the Fight against Obesity**

pp. 112–5

Another potential avenue toward obesity fighting therapies lies within the interleukin-6 family of cytokines. In the past, these cytokines have had positive results on weight loss and insulin sensitivity in humans and rodents. One member of this family, cardiotropin-1 (CT-1), has recently been revealed as a master regulator of energy metabolism. In this commentary, Moreno-Aliaga et al., the authors of this study, relay their findings on CT-1 and place them in context with other obesity and insulin resistance related studies.

**BAT and the Orexin Connection**

pp. 116–20

Orexin is a neuropeptide that is involved in a wide range of behaviors, from sleep to metabolism. Not surprisingly, a loss of orexin producing neurons also has a wide range of effects, including an impaired metabolism and risk of obesity. Interestingly, thermogenic properties (thermoregulatory and metabolic homeostasis) of brown adipose tissue (BAT) closely follow changes in orexin regulation. This connection lead Shaun Morrison’s group to explore the orexergic pathways that are involved in BAT control. In this commentary, Morrison et al. summarizes their findings, providing valuable insight into the neural control of BAT function.