Nuclear Receptor Control of White Adipose Tissue (WAT) pp. XX–XX

White adipose tissue (WAT), one of two types of adipose tissue found in mammals, is critical for energy storage in times when food sources are scarce. Too much WAT, however, evokes an inflammatory response that can lead to metabolic and cardiovascular disorders. This response is, in part, regulated by nuclear receptors found within the adipocyte and infiltrating adipose tissue macrophages. In this review, David Jacobi, Kristopher Stanya, and Chih-Hao Lee focus on these nuclear receptors and how they contribute to both an inflammatory state of WAT, leading to metabolic complications, and to a healthy state of WAT, maintained via homeostatic mechanisms. The authors also discuss recent findings for the role of nuclear receptors in adipose tissue associated macrophage function. With a solid understanding of how nuclear receptors function within adipocytes and macrophages, their modulation may bear out potential therapies to treat WAT inflammatory disorders.

Brown Adipose Tissue, the Other Fat pp. XX–XX

Brown adipose tissue (BAT), the ‘healthier’ cousin of white adipose tissue (WAT), specializes in energy dissipation and thermogenesis. It is found in abundance in hibernating animals and in infants. Until recently, it was thought that any BAT left in an adult had transformed into a state similar to WAT and had little, if any, activity. Recent studies reject this notion. BAT has not only been found in adults but it has also been found to be functionally active. In this review, Kristy Townsend and Yu-Hua Tseng summarize and reflect upon the many new findings surrounding this specialized, mitochondria-laden tissue. The authors also express their viewpoints on possible BAT targeted therapies; for example, increasing BAT may aid in our current battle against obesity and possibly curb the onset of type 2 diabetes, two very welcome outcomes for our ever increasing obese population.

Proteins That Make a Good Adipocyte Go Bad pp. XX–XX

Obesity is on the rise worldwide in a seemingly unabated manner. As expected, the prevalence of metabolic disorders is also on the increase. But, what is it about obesity that contributes to the onset of a metabolic disorder? In this review, David Brockman and Xiaoli Chen detail the roles that adipose tissue play in both healthy and obese states. The authors extol the use of proteomic technology and summarize how proteomic-based studies provide insights to understanding adipocyte biology. Specifically, how protein identification has contributed to the understanding of the pathogenic state of adipose tissue in obesity.

Absence of Notch Signaling Promotes Adipocyte Cross-Talk pp. XX–XX

Adipose tissue is generally known as an inert, often annoying, part of anatomy that stores energy and protects and insulates our bodies. Recently, it has gained another role as a functioning part of our endocrine system. Adipose tissue secretes many hormones that function in a paracrine manner to support its development and regulate the overall metabolism of its resident organism. How the cells within this tissue are able to communicate with each other to promote adipogenesis, angiogenesis and extracellular matrix remodeling is not understood. Urs et al. shed some light on this cellular cross-talk by studying soluble-Jagged1, an inhibitor of the important cell-fate regulating pathway, Notch. The authors find that the inhibition of Notch signaling has an effect on adipogenesis by increasing cell proliferation; however, it also prevents the cells from becoming mature adipocytes. Inhibited Notch also has a positive effect on angiogenesis. Their findings suggest that an initial inhibition of Notch...
signaling is required for successful cross-talk between preadipocytes and endothelial cells to promote adipose tissue growth via hyperplasia and hypertrophy.

**Orexin Receptor-1: Keeping Us Fit and Warm pp. XX–XX**

As you read this sentence, even if you are lying on the couch, you are burning off extra calories. You can thank your brown adipocytes and their thermogenic activity for that. If you are deficient in orexin receptor-1 (OXR1), however, you better invest in a treadmill to burn off those extra calories. OXR1 is one of the two G-protein receptors that the small excitatory neuropeptide (OX) binds and signals through. In this brief report, Dyan Sellayah and Devanjan Sikder attribute a lack of thermogenesis, and its consequential weight gain, to dysfunctional brown adipose tissue (BAT). Specifically, this dysfunction lies in the inability for brown preadipocytes to become functionally mature. This undifferentiated phenotype, along with a predisposition for obesity due to a lack of thermogenic activity, is seen in mice lacking OX, a phenotype also shared with ORX1 knockouts. So, you can thank your OX along with your ORX1 for keeping you warm and trim.

**You Are What Your Mother Eats pp. XX–XX**

As unfair as it may be, our current weight is partly determined by our mother's weight during pregnancy. Counter intuitively, if your mother was underweight during her pregnancy, you are more likely to be overweight and thus subjected to the many obesity-related disorders. In this commentary, Marie-Amélie Lukaszewski, Fabien Delahaye, Didier Vieau and Christophe Breton review their current research suggesting that specific proteins expressed in white adipose tissue (WAT) may have a hand in this maternal under nutrition (MU) fetal programming (Am J Physiol Endocrinol Metab 2011; http://dx.doi.org/10.1152/ajpendo.00011.2011). Studying the offspring from a maternal, food-restricted rat model, the authors found higher levels of leptin and corticosterone, two hormones actively involved in the regulation of WAT, as well as an increase in several other gene transcripts also found in WAT. Together, these increases may be the driving force that predisposes the offspring of an underweight mother to pile on the pounds.

**Want to Lose Weight? Listen to Your Gut Hormones pp. XX–XX**

Applauded increases in life expectancies in the Western world are in danger of being negated by a concurrent decrease in life expectancies from a growing population of obese Westerners. It is no surprise that researchers are determined to develop a safe and effective treatment to retract the recent rise in obesity. Systemic treatments to curb appetite have all but failed due to a plethora of negative side effects. Recently, De Silva et al. described a promising therapy that combined functional magnetic resonance with the administration of anorectic gut hormones, PYY3–36 and GLP-17–36 amide. The resulting brain activation pattern was similar to that seen in a satiated state (Cell Metab 2011; http://dx.doi.org/10.1016/j.cmet.2011.09.010). In this commentary, Salem et al. explain the significance of this study and compare it to other recent studies in this field, while focusing on the brain areas involved in the regulation of appetite.