In this Issue of Adipocyte

Small Chain Fatty Acids from Fermented Fiber

pp 81–8

The short chain fatty acids (SCFAs) propionic acid and butyric acid are generated through the fermentation of dietary fibers by colonic microbiota, and may impact energy metabolism as well as insulin sensitivity. Additionally, studies on obese mice treated with butyric acid have supported the concept that this SCFA may help in the treatment of diet-induced obesity though a decrease in fat content. This research paper by authors Heimann, Nyman, and Degerman explores the effects of these SCFAs on glucose uptake, lipolysis, and de novo lipogenesis, and shows how in rat primary adipocytes they may effect fat mobilization, storage, and glucose uptake as well as improving energy metabolism.

CD24 and in vitro adipogenesis

pp 89–100

Within the body, the cell surface marker CD24 is known to play a critical role in identifying which pre-adipocytes are able to develop into white adipose tissue. Authors Smith, Fairbridge, Pallegar and Christian examine CD24’s role and regulation outside of the body during adipogenesis in this research paper, finding an upregulation of CD24 in the early stages of pre-adipocyte differentiation and a downregulation in mature adipocytes. With evidence supporting the idea that dynamic CD24 upregulation can promote adipogenesis in vitro, their research shows that a prevention of this CD24 increase can reduce mature adipogenic gene expression, and can also lead to fewer lipid-laden adipocytes (Fig. 1).

Adipose Tissue Distribution, Saturated Fat and Fatty Liver

pp 101–12

While visceral adiposity has links to numerous ailments including type-2 diabetes, inflammation, and non-alcoholic fatty liver disease (NAFLD), subcutaneous adiposity does not share these links. In this research paper, authors Gentile et al. look at key differences between the two types of adiposity, with the belief that inherent or diet-derived differences may contribute to the role visceral adiposity may play in NAFLD. Their research shows that although it is clear that high saturated fatty acid diets are associated with liver inflammation, the link does not in fact lie in the impact of fatty acid composition on visceral adipose tissue.

The link between Growth Hormone supplementation and adipocyte lipid metabolism

pp 113–22

The beneficial effects of growth hormone (GH) supplementation on adipose tissue, lipid metabolism and insulin sensitivity are documented, but its molecular mechanism is still unclear. Authors Baląż et al. seek to shed light on these effects through the hypothesis that there exists a causal link between lipid-mobilizing adipokine zinc-α2-glycoprotein and GH on adipose tissue lipid metabolism in this research paper. Their research supports this claim, as they found that a 5-year GH therapy in GH-deficient patients not only improved glucose tolerance and adipose insulin sensitivity, but that the silencing of zinc-α2-glycoprotein actually nullified any effects that GH had on adipocyte lipid metabolism (Fig. 2).

Anti-Inflammatory Macrophage Transplant to trigger Browning

pp 123–8

In this brief report, authors Liu, Lin, Burton, and Wei explore a cell-based therapy for the treatment of high-fat-diet (HFD) -induced insulin resistance. Here, it is explained how the introduction of cultured anti-inflammatory adipose tissue macrophages from macrophage-specific Receptor Interacting Protein 140 null mice (mΔRIP140KD) mice into HFD- fed obese mice was able to start the browning process of their white adipose tissue, thus improving insulin sensitivity and reducing pro-inflammatory responses (Fig. 3).

Citrulline, UCP1 and metabolism within fat

pp 129–34

Citrulline (CIT) enriched diet can result in a reduction of white adipose tissue (WAT) mass. Authors Joffin et al. investigate whether this is due to CIT causing browning of WAT by analyzing uncoupling protein 1 (UCP1) expression and its transcriptional regulators from young high fat diet (HFD) -induced insulin resistance. Here, it is explained how the introduction of cultured anti-inflammatory adipose tissue macrophages from macrophage-specific Receptor Interacting Protein 140 null mice (mΔRIP140KD) mice into HFD- fed obese mice was able to start the browning process of their white adipose tissue, thus improving insulin sensitivity and reducing pro-inflammatory responses (Fig. 3).

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Obesity Risk Predictors in South Indians

pp 135–40
Understanding of the association of polymorphisms in the leptin gene with obesity-related phenotypes has been attempted numerous times, and with contradictory results especially with regards to the Indian population. This brief report by authors Dasgupta et al. seeks to explore these contradictions through the evaluation of leptin gene polymorphism, obesity and leptin levels within a South Indian Population. Their study suggests that obesity and leptin levels can be predicted by the common polymorphisms in the leptin gene, with certain variants being found to be significantly associated with obesity risk.

Tapping in to the Brain’s Thermostat through GLP-1

pp 141–5
The organ responsible for thermogenesis, brown adipose tissue (BAT) has been the focus of numerous studies since its discovery. Glucagon-like peptide-1 (GLP-1) receptors are expressed throughout the brain, and authors López, Dieguez and Nogueiras recently showed that the stimulation of these receptors within the hypothalamus is important in both BAT thermogenesis as well as the browning of white fat. This commentary examines these results as well as how the study of GLP-1 may be used in obesity treatments.

The Balancing Act of RIP140

pp 146–8
In this commentary, authors Liu, Lin, Burton, and Wei discuss the back and forth regulation of brown adipose tissue (BAT) and white adipose tissue (WAT) within our bodies, and the important regulatory role that Receptor Interacting Protein 140 (RIP140) plays in this balance. Levels of macrophage RIP140 appear to elevate in reaction to high-fat diets in order to increase macrophage recruitment to WAT and its inflammatory M1 polarization. Alternately, a lower level of macrophage RIP140 trigger WAT browning as well as fat burning and insulin sensitivity through anti-inflammatory M2 macrophages (Fig.4).
In obesity, macrophage infiltration is known to have numerous detrimental effects, including insulin desensitization, adipogenesis repression, and inflammation. Author Bing sheds light on the possible role of interleukin-1β (IL-1β) in the inhibition of insulin signal transduction, which can lead to impaired insulin sensitivity within adipose tissue. As discussed in this commentary, the blocking of IL-1β can improve insulin signaling in human adipocytes, showing that the targeting of IL-1β may lead to therapeutic advances in the fight against obesity-related insulin resistance.

Perspectives on Erythropoietin

Research has exposed erythropoietin and its receptors role in the regulation of white adipose tissue (WAT) mass, as well as inflammation and energy homeostasis. This commentary by Alnæeli and Noguchi further discusses the effects that erythropoietin can have on obesity-induced WAT inflammation, including possible effects on cytokine responses of macrophages and adipocytes. The authors show that there may also be links between this regulator and glucose metabolism and insulin resistance.

Lipid droplets and Caveolae, a link within cell size fluctuation

Our adipose tissue has evolved to be extremely efficient at storing energy for our continuous consumption. The channeling of nutrients to adipose tissue lipids and the storing of these lipids is also extremely efficient, with neutral lipids packed within lipid droplets. The storage of excess energy can lead to fluctuations of adipose tissue size. Authors Lay, Briand, and Dugail discuss the link between caveolae dynamics and lipid droplet organelles in this commentary, noting that a high caveolae count leads itself to lipid droplet expandability and storage, while forcing lipid droplet shrinkage leads to caveolae disassembly (Fig. 5).