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E-3. The difference of fat metabolism in adipocytes between the ruminant and rodent

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The factors that control fat deposition in adipose tissues are poorly understood. It is known that visceral adipose tissues display a range of biochemical properties that distinguish them from adipose tissues of subcutaneous origin. However, we have little information on gene expression lipid metabolism on inter-species variation in fat deposition. Using a differential display method to compare PCR-amplified gene transcripts in subcutaneous and visceral adipose tissue of cattle, the transcripts identified as having differential expression in the two adipose tissues were cell division cycle 42 homolog, prefoldin 5, decorin, phosphate carrier, 12S ribosomal RNA gene, and kelch repeat and BTB domain containing 2. Compared to cattle, the expression levels of these latter genes were differently expressed in the pig and in mice fed either a control or high-fat diet in order to compare the regulation of fat accumulation in other animal species. This species difference might be due, at least in part, to the different energy sources and digestive systems of these animals. Large amounts of volatile fatty acids are produced as a result of fermentation in the rumen and serve as the principal building blocks for lipid synthesis in cattle. The data presented here extend our understanding of gene expression and lipid metabolism in fat depots and provide further proof that the mechanisms of fat accumulation differ significantly between animal species.

E-4. Nutritional manipulation of adipose tissue development in broiler chickens

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In chickens, lipogenic activity in the liver is much greater than that in adipose tissue and most of fats accumulated in the adipose tissue accounts for by incorporation of triacylglycerols from plasma lipoproteins which are either synthesized in the liver (VLDL) or provided from dietary fats (Chylomicron). In the development of adipose tissue (adipose fat deposition), the rate-limiting step is lipoprotein lipase (LPL)-catalyzed hydrolysis of triacylglycerols in adipose tissues. The crucial role of LPL has been evidenced by our findings that inhibition of LPL activity by anti-LPL monoclonal antibody caused lipemia and decreased adipose fat deposition to half that of control counterparts in chickens. Thus the LPL has been targeted for nutritional modification in order to reduce the fatness of chickens. However, LPL mRNA expression in growing chickens is less responsive to aging and nutritional means compared to mammals, indicating species-specificity in the regulatory mechanism of fat deposition. Therefore, in order to modulate chicken fat deposition, we have explored three viewpoints that are regulation of LPL-catalyzed hydrolysis, VLDL secretion from liver and adipose cell differentiation. In this presentation, we show what extent and how these points work on reducing fat deposition in chickens.