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Research Summary:

The research of the Blaner laboratory has contributed significantly to knowledge regarding the role of retinoids in both white and brown adipocyte biology. Our laboratory was the first to establish that both white and brown adipocytes store retinoid (retinol and retinyl ester) and may account for as much as 15-20% of the total retinoid stored in the body; that white and brown adipocytes synthesize and secrete retinol-binding protein 4 (RBP4); that lipoprotein lipase (LPL) associated with adipocytes contributes to the uptake of chylomicron retinyl ester by adipocytes; that hormone sensitive lipase (HSL) contributes to hydrolysis of retinyl ester stored within adipocytes and the mobilization of retinol from adipocytes; that adipocytes are enriched in CRBPIII whereas adipose tissue stromal-vascular cells are enriched in CRBPI; and that neither lecithin:retinol acyltransferase (LRAT) nor diacylglycerol acyltransferase 1 (DGAT1) (a physiologically significant acyl-CoA:retinol acyltransferase (ARAT)) contributes to retinyl ester formation within adipocytes.

Recent research activity in the Blaner laboratory has focused on adipocyte-derived RBP4 and its role in metabolic disease development. Although the laboratory has a longstanding research interest in RBP4 and has published extensively on RBP4, our past research on RBP4 was aimed primarily at understanding its role in retinoid transport and metabolism. We are now focusing on RBP4 and its role in metabolic disease development. Since the publications in 2005 by Kahn and colleagues showing linkages between elevated circulating RBP4 levels, obesity and insulin resistance, there is a large and growing literature relating blood RBP4 levels with the development of insulin resistance, non-alcoholic fatty liver disease, and cardiovascular disease. However, this literature relating RBP4 and metabolic disease, including both human and animal studies, is controversial and still inconclusive. Thus, to better understand possible relationships between adipocyte-derived RBP4 and metabolic disease, we have generated and are studying a number of genetic mouse models that express RBP4 specifically in adipocytes. Indeed, these transgenic mice show impaired responses to a glucose challenge and an increased propensity for developing fatty liver. The goal of our future research will be to establish the molecular basis for these observations.

Selected Publications: (2014-2015)

Wongsiriroj, N., Jiang, H., Plantedosi, R., Yang, K.J.Z., Kluwe, J.


More about: William S. Blaner

Complete List of Published Work in MyBibliography
