NEW UNDERSTANDING OF DIETARY RETINOID ABSORPTION,
POSTPRANDIAL PROCESSING AND ACTIONS.

William S. Blaner¹², Nuttaporn Wongsiriroj³, Sheila M. O'Byrne², Krzysztof Palczewski³, and Jenny M. Libien³ Departments of Medicine¹ and Pathology¹ and the Institute of Human Nutrition², Columbia University, New York, NY USA and the Department of Pharmacology³, Case Western Reserve University, Cleveland, OH USA

Retinoids are taken up from the diet and packaged as retinyl esters in chylomicrons. Approximately 66-75% of dietary retinoid is cleared and stored by the liver. The remainder is stored in extrahepatic tissues, with adipose tissue being the most important tissue site of retinoid storage after the liver. Study of lecithinretinol acyltransferase (LRAT)-deficient mice indicates that LRAT is responsible for synthesizing approximately 90% of the retinyl ester packaged into chylomicrons. The remainder is synthesized by an acyl-CoA-dependent enzyme that we hypothesize is diacylglycerol acyltransferase 1 (DGAT1). Interestingly, LRAT accounts for nearly all of retinyl ester synthesis in the liver and other tissues in the body aside from adipose tissue where retinyl esters are present at markedly elevated levels in LRAT-deficient mice. Moreover, the level of cellular retinol-binding protein, type III (CRBPIII), which is involved in the uptake and processing of retinoid in adipocytes, is markedly dysregulated in LRAT-deficient mice. Recently published work by Kahn and colleagues indicates that plasma retinol-binding protein (RBP) that is synthesized and secreted by adipocytes acts as a signal that brings about diminished insulin responsiveness in muscle. This finding suggests that retinoid storage and processing by adipocytes may be importantly linked to the development of diabetes. Our recent work has focused on gaining better understanding of the molecular mechanisms responsible for the storage and processing of retinoids in adipocytes and on elucidating linkages between these and the development of obesity and diabetes. (Supported by NIH grants DK061310 and DK068437)
WILLIAM STEPHEN BLANER
Departments of Medicine and the Institute of Human Nutrition,
Columbia University,
New York, NY USA

Professor of Nutritional Medicine (in Medicine and the Institute of Human Nutrition) (University Tenure), Columbia University

Born: November 12, 1950. Birthplace: Johnstown, Pennsylvania

Academic Training and Traineeship
University of Maryland, College Park, Maryland, B.S. in Biochemistry, 1972.
University of Tennessee, Knoxville, Tennessee, M.S. in Biochemistry, 1975.
University of Tennessee, Knoxville, Tennessee, Ph.D. in Biochemistry, 1979. Thesis Title: Two Dehydrogenases: Sucinic Semialdehyde Dehydrogenase and Retinol Dehydrogenase; Professor Jorge E. Churchich, Sponsor.

Professional Organizations and Societies.
American Society for Biochemistry and Molecular Biology (ASBMB)
American Society of Nutritional Sciences (ASNS)
American Chemical Society
Harvey Society

Honors and Activities.
Ruth L. Pike Lecture and Award, Distinguished Young Investigator, Pennsylvania State University, April 1991
Committee Member, Food and Drug Administration, Food Safety Committee, Thirty Month Review of the Safety of Olestra, June, 1998.
Advisory Committee Member, National Cancer Institute, Consensus Panel Reviewing the Clinical Applications of 4-Hydroxyphenyletriminamide, February 1999.
Advisory Committee Member, Society of Investigative Dermatology, Consensus Panel Reviewing the Safety and Clinical Application of Isotretinoin, June 1999.
Organizing Committee Member, 1997 European Retinoid Research Group Meeting, Nice, France, September 1997.
Peer Review Team for the 5-Year External Evaluation of the Department of Nutritional Sciences, Texas A&M University, College Station, TX, March 2000.
Co-Chairman, 2002 FASEB Summer Conference on Retinoids.
Chairman, 2004 FASEB Summer Conference on Retinoids.