Biography

Robert (Bob) A. Kesterson, PhD
Professor of Genetics
Director, Transgenic & Genetically Engineered Models (TGEMs) Facility
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Dr. Kesterson completed his undergraduate studies in Chemistry at Hendrix College, and received a Ph.D. in Cell Biology from Baylor College of Medicine (1993). His postdoctoral training in the field of neuroendocrinology was carried out at The Vollum Institute for Advanced Biomedical Research, Portland, Oregon with Dr. Roger D. Cone, which focused on determining the function of newly cloned melanocortin receptors. Dr. Kesterson was appointed in 1997 to the faculty of Vanderbilt University in the Department of Molecular Physiology and Biophysics and was then recruited to UAB in 2004.

Dr. Kesterson is the Director of the UAB Transgenic & Genetically Engineered Models (TGEMs) Facility and is a member of several UAB Centers including: Comprehensive Cancer Center, Comprehensive Arthritis, Musculoskeletal, Bone and Autoimmunity Center, Hepatorenal Fibrocystic Disease Core Center, Civitan International Research Center, Comprehensive Diabetes Center, and Nutrition Obesity Research Center

The Kesterson laboratory uses genetic mouse models to study numerous physiological and behavioral processes. Originally trained as a bone biologist, Dr. Kesterson was the first to clone the human Vitamin D Receptor (VDR) gene, and to create a series of mouse models using the human osteocalcin gene promoter to map *in vivo* vitamin D responsive enhancer elements. Subsequent postdoctoral training in the melanocortin receptor (GPCRs) field creating knockout mouse models broadened research interests towards molecular mechanisms by which CNS melanocortin receptors regulate feeding, energy balance, thermoregulation, inflammation, and learning and memory. His laboratory uses genetically modified animal models to study numerous physiological and behavioral processes with recent focus on 1) mechanisms by which primary cilium localized on hypothalamic neurons regulate energy balance, and 2) humanized models of Neurofibromatosis Type 1 (NF1) and developing therapeutic interventions.