JEFFREY E. PESSIN, Ph.D.

Positions:

Professor, Departments of Medicine and of Molecular Pharmacology
Director, Einstein Diabetes Research Center, Albert Einstein College of Medicine

Research interests:

I have actively supervised a research laboratory since 1983 that has continually focused on the specific mechanisms responsible for insulin signaling at the molecular level, the regulation of glucose uptake and metabolism at the cellular, molecular and integrative systems biology levels. More recently, we have become highly active in the study of the interrelationships among and control mechanisms responsible for insulin sensitivity, intermediary metabolism and energy balance. To accomplish these broad objectives, we take advantage of a variety of experimental systems that include in vitro reconstitution, cell culture, tissue explants, genetic rodent models and human study subjects using biochemical, cell, molecular and integrative physiological approaches. In particular, we have been investigating the role of adipocyte progenitor cells, adipocyte programmed cell death and adipose tissue fibrosis that is induced by high-fat feeding. In these studies, we have used a variety of experimental approaches, including genetically engineered mice, fluorescent activated cell sorting analyses and sorting along with quantitative reverse transcriptase polymerase chain reaction.

Current grant funding:

R01 DK033823 (Pessin) NIH/NIDDK	4/1/15-3/31/19 Regulation of the insulin receptor kinase
R01 AR064420 (Pessin) NIH/NIAMS	3/7/13-2/28/18 Molecular basis for skeletal muscle pathophysiology in Pompe's disease
R01 DK110063 (Pessin) NIH/NIDDK expression	6/15/16-3/31/21 The mediator complex in the coordinate regulation of lipogenic gene
P30 DK020541 (Pessin) NIH/NIDDK	4/1/15-3/31/20 Regional Einstein-Mount Sinai Diabetes Research Center (ES-DRC)is

Recent publications:

1. **ARC is essential for maintaining pancreatic islet structure and β-cell viability during type 2 diabetes.** McKimpson WM, Zheng M, Chua SC, **Pessin JE**, Kitsis RN.
2. Sci Rep. 2017 Aug 1;7(1):7019. doi: 10.1038/s41598-017-07107-w.
3. **Cyclin C regulates adipogenesis by stimulating transcriptional activity of CCAAT/enhancer-binding protein α.**Song Z, Xiaoli AM, Zhang Q, Zhang Y, Yang EST, Wang S, Chang R, Zhang ZD, Yang G, Strich R, **Pessin JE**, Yang F. J Biol Chem. 2017 May 26;292(21):8918-8932. doi: 10.1074/jbc.M117.776229. Epub 2017 Mar 28.
4. **MC4R-dependent suppression of appetite by bone-derived lipocalin 2**. Mosialou I, Shikhel S, Liu JM, Maurizi A, Luo N, He Z, Huang Y, Zong H, Friedman RA, Barasch J, Lanzano P, Deng L, Leibel RL, Rubin M, Nicholas T, Chung W, Zeltser LM, Williams KW, **Pessin JE**, Kousteni S. Nature. 2017 Mar 16;543(7645):385-390. doi: 10.1038/nature21697. Epub 2017 Mar 8. Erratum in: Nature. 2017 Jun 14;546(7658):440.
5. **Transcription Factor EB Controls Metabolic Flexibility during Exercise.** Mansueto G, Armani A, Viscomi C, D'Orsi L, De Cegli R, Polishchuk EV, Lamperti C, Di Meo I, Romanello V, Marchet S, Saha PK, Zong H, Blaauw B, Solagna F, Tezze C, Grumati P, Bonaldo P, **Pessin JE**, Zeviani M, Sandri M, Ballabio A. Cell Metab. 2017 Jan 10;25(1):182-196. doi: 10.1016/j.cmet.2016.11.003. Epub 2016 Dec 20.
6. Martinez-Lopez N, Tarabra E, Sahu S, Garcia-Macia M, Toledo M, Coletto M, Batista A, **Pessin JE**, Schwartz GJ, Kersten S, Singh S. System-wide benefits of twice a day feeding by autophagy. Cell Metab. 2017, in press.