DEPARTMENT OF
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## Tuesday, October 1, 2019
3:00 p.m., Room 1415 BPS

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*Sponsored by Pam Fraker*

# "New insights into Growth Hormone and STAT5 dependent regulation of adipose tissue mass and metabolic health "

STATs (Signal Transducers and Activators of Transcription) are a family of transcription factors whose activity is largely controlled by hormone-induced tyrosine phosphorylation. STAT5A and STAT5B are highly induced during murine and human adipocyte differentiation. We and others have identified STAT5 target genes related to lipid metabolism and/or insulin resistance.

Decades of research have demonstrated that growth hormone (GH) induces has both lipolytic and diabetogenic activities. Although substantial advances have been made, the mechanism(s) by which GH regulates these processes are still not completely understood. Also, many of the relevant animal models generated thus far have had limited metabolic phenotyping analyses.

To investigate the physiological roles of STAT5 in adipose tissue, we generated mice lacking both STAT5 genes in adiponectin-expressing cells (**STAT5$^{AKO}$**). As expected, both STAT5$^{AKO}$ male and female mice have a prominent adiposity phenotype on chow diets. Notably, other features of these mice were not predicted and have provided potentially paradigm-shifting data related to lipolysis and GH actions on adipocytes**.** We observed several unpredicted phenotypic observations in the STAT5$^{AKO}$ mice including: 1) the adiposity phenotype is lost with high-fat feeding such that both floxed and STAT5$^{AKO}$ mice have equivalent fat mass; 2) the adiposity phenotype is not accompanied by altered lipolytic responses; 3) exogenous GH reduces fat mass similarly in STAT5$^{AKO}$ and floxed control mice; and 4) sex-specific differences in adipose tissue mass/maintenance are observed in STAT5$^{AKO}$ mice during weight loss. These highly novel observations suggest there are fundamental gaps and misunderstandings regarding the roles of adipocyte STAT5 and the ability of these proteins to systemically affect metabolic health.