

The Graduate Program in Biomedical Sciences  
is proud to announce the



Ph.D. Dissertation Defense of  
**REBECCA E. RUGGIERO-RUFF**

Biomedical Sciences Ph.D. Candidate  
in the Coss Lab

Dr. Djurdjica Coss, Chairperson

**Genetic and Environmental Causes of Obesity and Their Impact on  
Hypothalamic Function and Pituitary Plasticity**

Obesity is a chronic disease that is increasingly becoming a global public health concern. In addition, there are sex differences in obesity mediated pathologies, such that obese men are more at risk for developing metabolic syndrome and cardiovascular disease than obese women. The hypothalamus regulates a wide variety of homeostatic processes, including food intake and energy expenditure, which are tightly regulated in order to maintain proper energy homeostasis. Genetic causes of obesity are largely hypothalamic in origin; however obesity is multifactorial, consisting of both genetic and environmental components that contribute to the steadfast increase in prevalence. In addition, the hypothalamus regulates the pituitary gland, an endocrine gland that is responsible for the synthesis and secretion of hormones that are important in the regulation of basal metabolism, reproduction and stress, all of which are dysregulated in obese patients. This work aims to elucidate genetic and environmental causes in hypothalamic and pituitary dysfunction in obesity. We identified a new genetic target, fragile X messenger ribonucleoprotein 1 (FMR1), in the regulation of energy expenditure in the hypothalamus and etiology of FMR1-linked obesity in mice. When looking at chronic high fat diet as an environmental contributor to obesity, we determined high fat diet-induced changes in pituitary gland plasticity and hormone production that may play a role in altered pituitary gland homeostasis in diet-induced obesity. In a separate study, we investigated sex differences in diet-induced obesity pathogenesis and demonstrated that macrophage-secreted protein, resistin-like molecule alpha (RELM $\alpha$ ), critically protects female mice against diet-induced obesity. Collectively, these studies implicate a new genetic target in hypothalamic regulation of energy expenditure and that diet-induced obesity alters pituitary plasticity and hormone production, which could explain endocrine dysfunction in obesity.

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Wednesday, September 6th, 2023 at 12:00PM (PST)

Multidisciplinary Research Building (MRB), Rm. 1110 (First floor)

Join via Zoom:

<https://ucr-edu-hipaa.zoom.us/j/94303903828?pwd=MUxTdXVXMTZSdHBxOGZ5bDFydG5oQT09>

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