Research Abstract
Juliann Jugan

The persistent nature of the insecticide DDT and its metabolite, Dichlorodiphenyldichloroethylene (DDE), leads to chronic interaction between these toxicants and the adipose tissue they accumulate within. DDE has been detected ubiquitously in human samples and is consistently associated with increased risk of obesity. Studies investigating the obesogenic effect of DDT in mice have shown that this pollutant impairs non-shivering thermogenesis in brown adipose tissue, decreasing energy expenditure and increasing adiposity. Variants in the gene FTO have also been strongly associated with impaired thermogenesis and increased risk of obesity. Preliminary data in human blood and mouse brown adipose has shown differential methylation of FTO as a result of exposure to DDT and DDE. The effect of DDE exposure on the epigenetic regulation of FTO has not been documented. I plan to investigate the epigenetic effects of DDT and DDE exposure on FTO in the context of thermogenic regulation in an in vitro model of brown adipose and through secondary analysis of human genomic and methylomic data. I speculate that hypermethylation of FTO as a result of DDT and DDE exposure prevents repressor binding to downstream negative thermogenic regulators, suppressing thermogenesis and leading to increased obesity risk.