Phthalates are synthetic plasticizers commonly used in both industrial and consumer products. Despite their ubiquity in domestic environments, many phthalates pose novel health risks due to their potential teratogenicity following developmental exposure. Evidence from previous research suggests that gestational exposure to one such phthalate—Di(2-ethylhexyl) phthalate **(DEHP**)—is associated with altered birth outcomes and offspring neurodevelopmental and behavioral problems in humans. Research conducted in our lab and elsewhere have identified disruptions in zinc homeostasis as one potential mechanism involved in DEHP developmental toxicity . While there is some information regarding the capacity of dietary DEHP to affect the gastrointestinal (**GI**) microbiota, the developing brain, and reproductive system, few studies have examined impact of DEHP exposure on the development of GI tract. Additionally, the capacity for DEHP-mediated alterations in zinc metabolism warrants further study due to zinc’s importance in maintaining intestinal barrier integrity, and facilitating normal cellular differentiation and proliferation during early growth and development. To bridge this gap in knowledge I will use an *in vitro* Caco-2 cell model to directly examine any effects on protein markers related to zinc homeostasis and tight junction integrity.