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**The effect of developmental fentanyl exposure on mitochondrial gene expression in the brain and blood in C57Bl/6 mice**

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The potency of the synthetic opioid fentanyl has led to the rapid escalation of use, increased recreational use, and subsequently opioid-related overdoses. The wide spread use of fentanyl has increased the incidence of in utero exposure to the drug, but the long-term effects of this type of exposure are not yet understood. The role of neuronal mitochondria in the context of drug addiction is a new and exciting field, as the metabolic output of mitochondria impact neuronal excitation and signaling. Previous studies demonstrate that cocaine self-administration alters mitochondrial size in nucleus accumbens (NAc) neurons but mitochondria are not well examined with other drugs of abuse. This study examines the expression of mitochondrial related genes in the NAc after perinatal exposure to fentanyl. While understanding the changes that occur in reward processing regions of the brain are fundamental to understanding the motivational changes associated with fentanyl use, this data is not readily available in a clinical population. Therefore, we examined the utility of circulating blood mitochondrial copy number as a proxy for brain gene expression changes and behavior. We found that mitochondrial fission protein 1 (Fis1) mRNA was altered in the NAc by fentanyl exposure and correlated with avoidance behavior in an elevated-plus maze task. Further, there was a trending correlation with blood mitochondrial copy number. These data indicate that developmental fentanyl exposure impacts mitochondrial function in both the brain and body in ways that can impact neuronal signaling and may prime the brain for altered reward-related behavior in adulthood.