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Impact of Early Life Adversity on Reward Behaviors: A Role for L1 Retrotransposons in the Amygdala

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Adversity experienced early in life can impact reward-related behaviors and increase the risk for the development of psychiatric disorders later in life. We use a predator odor exposure (POE) to model early life adversity. This fear of harm model has previously shown a reduction in juvenile social play behavior. This reduction in social play is accompanied by an increase in copy number in the juvenile amygdala of the retrotransposon, L1. This increase in L1 was inversely correlated with juvenile social play behavior. L1 is an autonomous mobile element that can copy and paste itself elsewhere within the genome. L1 has also been associated with several psychiatric disorders including major depression and substance use disorder. We are now extending our research to investigate whether changes in social behavior extend to adulthood and if these changes are reward general or social specific. We are using social, sucrose, and drug self-administration in adulthood to assess whether POE alters motivated behaviors beyond the adolescent period. To test whether L1 is functionally relevant we are transiently knocking down L1 in the amygdala and assessing the impact on behavior. We are investigating whether L1 in the amygdala is a regulator of rewarding behavior including social behavior and drug self-administration. If our results are consistent with our hypothesis this would be an interesting finding as there is very little research linking L1 genomic changes with rewarding behaviors. Altogether we are developing a nuanced understanding of how early life adversity alters the genome to affect motivation for reward.