reward minus non-reward) was observed (r(49)= -.29, p =.042). However, no association in the regression including covariates (beta_ETSexposure = -.22, t (5, 43) = -1.46, p = .152) was observed.
Conclusions: Overall, there are no indications that ETS exposure affects cue-reactivity and inhibitory control. However, there are some indications that ETS exposure impacts reward processing in never-smoking adolescents.
Supported By: KWF (Dutch Cancer Society)
Keywords: Environmental Tobacco Smoke Exposure, ERPs, EEG, Adolescence, Addiction

F205. Loss of Noradrenergic-Derived Galanin Enhances Opioid Reward and Reinforcement

Stephanie Foster[1], Natale Sciolino[2], Patricia Jensen[2], and David Weinshenker[1]

[1]Emory University, [2]National Institute of Environmental Health Sciences

Background: Galanin is a neuropeptide co-expressed in multiple neurotransmitter systems that modulates opioid-related behaviors in rodents. Here, we sought to investigate whether selective loss of noradrenergic galanin would be sufficient to enhance opioid reward and reinforcement.
Methods: All studies used 3-7 month-old mice lacking galanin in noradrenergic neurons (NE Gal KO), generated by crossing DBHCre and floxed galanin lines, and their wild-type littermates (WT). To study opioid reward, an unbiased 8-day conditioned place preference (CPP) paradigm was conducted with saline and morphine (5 mg/kg) treated groups. Opioid reinforcement was examined via intravenous self-administration studies. Mice were food trained by operant conditioning prior to surgical catheterization of the right jugular vein. After one week of recovery, mice acquired remifentanil self-administration (6.4 μg/kg/infusion) during 1 hour operant sessions on an FR-1 schedule.
Results: NE Gal KO mice formed a statistically significant place preference to 5 mg/kg morphine while WT mice did not (n=8-10 per group, two-way ANOVA with Sidak's post-hoc test, p=.001). For self-administration studies, NE Gal KO mice made significantly more active responses per session than WT over the first 4 days of acquisition (n = 3-5 per group, two-way ANOVA with Tukey's post-hoc test, p<0.05 all time points).
Conclusions: This is the first study to show that loss of galanin in noradrenergic neurons is sufficient to enhance opioid reward and reinforcement. Future studies will evaluate the circuit by which noradrenergic galanin mediates its effects.
Supported By: F31DA044726, R01DA038453
Keywords: Galanin, Opioid, Reward, Reinforcement, Opioid Use Disorder

F206. Neural Mechanisms Guiding Choices for Cannabis and Alternative Rewards in Cannabis Smokers

Gillinder Bedi[1], Xuejun Hao[2], Anna Konova[3], Nicholas Van Dam[4], Paul Glimcher[5], and Margaret Haney[6]

[1]University of Melbourne and Orygen National Centre of Excellence in Youth Mental Health, [2]College of Physicians & Surgeons, Columbia University, [3]Rutgers University, [4]University of Melbourne, [5]New York University, [6]Columbia University Medical Center

Background: Substance misuse is characterised by persistent choices for drugs over other rewards. Neural mechanisms underpinning drug-biased choice in humans are poorly understood. Using an experimental medicine approach, we investigated subjective value (SV) encoding during choices for cannabis and a natural reward (individuals' preferred snacks) in regular cannabis smokers. Effects of cues (cannabis, snack, or neutral) were also assessed.
Methods: Near-daily cannabis smokers (N = 20; 1 female) completed a 6-day, within-subject, inpatient protocol. After sampling the reinforcers (6 cannabis puffs; 6 small snacks), they completed 4 conditions: 1. Neutral cues/cannabis choices; 2. Cannabis cues/cannabis choices; 3. Neutral cues/snack choices; and 4. Snack cues/snack choices. In each, participants were exposed to cues before an fMRI scan during which they chose repeatedly between 0-6 cannabis puffs/snacks and an individualized monetary amount. SV was operationalized as the strength of preference for each choice. Following each scan, two choices were randomly selected for implementation.
Results: There was no effect of cues or interaction between cues and reinforcer type on SV encoding. SVs for cannabis correlated with activation in regions previously shown to encode value for other rewards, including ventromedial Prefrontal Cortex (vmPFC); a similar pattern was not observed during snack choices. Value encoding in vmPFC was greater for cannabis than snack food (Small Volume Correction; p<0.05).
Conclusions: Cannabis smokers had intact value encoding for cannabis but disrupted encoding of non-drug value, consistent with models identifying dysregulated valuation of drug relative to alternative reinforcers as a driver of problematic substance use.
Supported By: NIDA: DA034877; DA044339
Keywords: Neuroeconomics, Cannabis, Subjective Value, Experimental Medicine, Human Behavioral Pharmacology

F207. Multi-Modal Neuroimaging Characterization of Co-Occurring Mild Traumatic Brain Injury and Alcohol Use Disorder

Amy Herrold[1], Neil Jordan[2], Todd Parrish[3], Derin Cobia[4], R. Andrew Chambers[5], Angelle Sander[6], Gwendolyn Kartje[7], Alexandra Aaronson[3], Francis Sesso-Osburn[8], and Theresa Bender Pape[2]

[1]Edward Hines Jr., VA Hospital & Northwestern University, Feinberg School of Medicine, [2]Edward Hines Jr., VA Hospital, Northwestern University, [3]Northwestern University, [4]Brigham Young University, [5]Indiana University, [6]Baylor College of Medicine, [7]Edward Hines, Jr., VA Loyola University, [8]Adler University

Background: Alcohol use disorder (AUD) and mild traumatic brain injury (mTBI) commonly co-occur among Veterans leading to exacerbated alcohol craving. We hypothesize that the co-occurrence of these conditions leads to a exacerbated dysfunction, which could be ameliorated with neuromodulatory treatment.