August 2022

The Longitudinal Effects of Early Substance Use and ADHD Symptom Development in the ABCD Study

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THE LONGITUDINAL EFFECTS OF EARLY SUBSTANCE USE AND ADHD SYMPTOM DEVELOPMENT IN THE ABCD STUDY

by

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A Dissertation Submitted in

Partial Fulfillment of the

Requirements for the Degree of

Doctor of Philosophy

in Psychology

at

The University of Wisconsin-Milwaukee

August 2022

ABSTRACT

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The University of Wisconsin-Milwaukee, 2022
Under the Supervision of Professor Krista M. Lisdahl

Previous literature has demonstrated a link between Attention-Deficit/Hyperactivity Disorder (ADHD) and substance use. However, few studies have examined how early substance use initiation in late childhood and early adolescents impacts ADHD symptoms overtime. To help investigate these trajectories we utilized the Adolescent Brain Cognitive Development (ABCD) Study, which includes 11,875 children (ages nine and ten at baseline) recruited from schools across 21 different study sites across the United States and followed for ten years. During study visits, participants and their parents completed questionnaires and interviews which were utilized for the current study. Participants were asked about past year substance use and ADHD symptoms, which were obtained through the Child Behavioral Checklist (CBCL) completed by parents. Further, additional demographic information, health factors, and substances use risk factors were collected and incorporated into analyses to account for confounding factors. Multivariate analyses were run to determine differences in ADHD symptomology at baseline by substance use group. Then, multilevel linear mixed effect models were run to examine the impact of early substance use on ADHD symptomology from baseline, one-year, and two-year follow-up. Lastly, follow-up sex analyses were run to determine if different substance use and ADHD patterns emerged between male and female participants. Substance use groupings of caffeine use, alcohol sipping, and total substance use were run separately. At baseline, moderate caffeine use and mild alcohol sipping was associated with higher ADHD symptomology. Total substance use

was not associated with ADHD symptoms at baseline. Longitudinal analyses demonstrated that moderate caffeine use and moderate total substance use was associated with high ADHD symptoms at two-year follow-up. Sex analyses demonstrated that moderate total substance use in male participants and moderate caffeine use in female participants was associated with higher ADHD symptoms at two-year follow-up. These findings suggest that even light and early use can start to impact ADHD symptom trajectories within a normative population. More work is needed to investigate the mechanisms behind the effects of early substance use on ADHD symptoms in late childhood as well as the differences in these patterns by biological sex.

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ACKNOWLEDGEMENTS

I would like to acknowledge the many individuals who invested time into me and my work over the many years leading up to this dissertation. Thank you to my mentor Krista for five years of meetings, draft revisions, and professional advice. Thank you to the BraIN lab for contributing to the collection of this research and the many lab meetings that have been devoted to discussing this research. Further thank you to the graduate students in the BraIN lab who contributed to this work with special thanks to Ryan Sullivan for providing copious amounts of time helping with R code, text revisions, and meetings generating ideas. Thank you to my amazing cohort members, Abel Mathew, Amy Lang, and Carissa Weis who provide emotional support and listened to multiple practice presentations in preparation for my proposal and defense. Thank you to my family members and friends who invested their time and energy into holding me up and listening to me talk about research when I know they would much rather talk about anything else. And finally, thank you to the participants of the ABCD study who volunteered their time to be part of such a momentous study and for which none of this work would be possible without them.

Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder that is marked by a persistent pattern of both inattentive and hyperactive/impulsive symptoms during development (American Psychiatric Association, 2013). While rates of childhood ADHD vary due to methodological characteristics in studies (Polanczyk, Willcutt, Salum, Kieling, & Rohde, 2014), approximately 10% of children and adolescents have received a parent-reported diagnosis of ADHD in their lifetime (Danielson et al., 2018). ADHD is classified as a neurodevelopmental disorder due to evidence that individuals with ADHD phenotypically lag behind their peers in several domains (Sjöwall, Roth, Lindqvist, & Thorell, 2013). While these developmental delays are most commonly seen in children and continue into adolescents (Bussing, Mason, Bell, Porter, & Garvan, 2010), these deficits can continue to persist into young adulthood (Wilens & Spencer, 2010). Some estimates have suggested that about 15% of young adults continue to meet criteria for ADHD diagnosis and around 65% still experience subclinical ADHD symptoms (Faraone, Biederman, & Mick, 2006); although persistent ADHD estimates range anywhere from 5-76% (Caye et al., 2016). This persistent ADHD has been shown to be detrimental in domains of academic (Arnold, Hodgkins, Kahle, Madhoo, & Kewley, 2020; Kuriyan et al., 2013; Voigt et al., 2017), occupational (Fredriksen et al., 2014; Hechtman et al., 2016; Kuriyan et al., 2013), and emotional (Hechtman et al., 2016; Roselló et al., 2020) functioning. For these reasons, much work has gone into examining why some individuals with ADHD "phase" out of these developmental delays as they get older, while others continue to experience significant symptoms that impact functioning.

A large literature has been built linking ADHD and substance use; however, the exact nature of their relationship has been difficult to parse out (Zulauf, Sprich, Safren, & Wilens,

2014). Substance use has been considered as a potential factor relating to persistence of ADHD diagnosis or symptoms during adolescence; indeed, children with ADHD are significantly more likely to develop problems with substance use compared to their non-ADHD peers (Wilens, Biederman, Mick, Faraone, & Spencer, 1997; Wilens et al., 2011). Up to 48% of adults with ADHD have been shown to have comorbid substance use disorder (SUD) (Moffitt et al., 2015). Similarly, 23% of individuals with a SUD meet criteria for comorbid ADHD (van Emmerik-van Oortmerssen et al., 2012). Due to this high rate of comorbidity between the two disorders, more studies have sought to examine the prospective link between childhood ADHD and later substance use onset and trajectory (Lee, Humphreys, Flory, Liu, & Glass, 2011); however, relatively few studies have examined how substance use may impact the trajectories of childhood ADHD symptoms.

Rather, several studies looking at how childhood ADHD impacts future adolescent substance use have shown interesting findings; a meta-analysis by Lee and colleagues (2011) showed that childhood ADHD led to increased lifetime nicotine and cannabis use in adolescent and young adulthood. Further, the same meta-analysis showed that children with ADHD were more than 3 times more likely to report nicotine dependence, 1.7 times more likely to report alcohol abuse/dependence, and 1.5 times more likely to report cannabis abuse/dependence in adolescence and adulthood compared to children without ADHD. Other longitudinal work has highlighted that childhood ADHD was associated with increased trends of alcohol, cannabis, and nicotine use in adolescents compared to their non-ADHD counterparts (Molina et al., 2018; Molina & Pelham Jr, 2003; Sibley et al., 2014). This work also demonstrated that childhood ADHD, when compared to a control group, led to escalated early substance use, particularly alcohol and non-cannabis illicit substance use (Molina et al., 2018). Notably, while these studies

lend evidence that childhood ADHD increased risk for escalated substance use and SUD, no longitudinal studies to date have looked at how early substance use influences persistence of ADHD symptoms into adolescence and young adulthood.

Prospective studies investigating the relationship between ADHD and substance use have largely focused on how childhood ADHD predicts later substance use. While directionality of this relationship is logical due to the typically earlier onset of ADHD compared to substance use, it is also possible that early and persistent use of substances during early adolescence may lead to persistent or increased ADHD symptoms in later adolescence and young adulthood. There is some evidence to support this, as adolescents (13-17 years old) with persistent ADHD are found to have significant more substance use problems (e.g., repetitive drunkenness, alcohol problems, and daily cigarette smoking) than adolescents who no longer meet criteria for ADHD (Molina & Pelham Jr, 2003). Further, in a 10 year follow-up study, young adults with persistent ADHD were found to be 3 times more likely to be diagnosed with an SUD compared to non-persistent ADHD counterparts (Wilens et al., 2011). While these studies indicate some reciprocal relationship between persistent ADHD and substance use, the timing and characterization of these effects are relatively unexplored. Further, this relationship is often explored in adulthood with few studies examining the relationship in adolescents and even fewer in late childhood, a time when substance use initiation is beginning (SAMHSA, 2019).

While these studies largely look at diagnosed ADHD in children, existing literature suggests that even subclinical ADHD symptom trajectories are worthy of studying. Previous research has lent support to considering dimensional ADHD paradigms as children with subclinical ADHD are shown to have cognitive deficits similar to children with ADHD (Christensen & Lundwall, 2018; Salum et al., 2014). These same deficits continue on in adults

who display ADHD symptomology (Brown & Casey, 2016; Mohamed, Börger, Geuze, & van der Meere, 2016). Further, adults with subclinical ADHD symptoms have been shown to experience difficulty related to these ADHD symptoms (Gudjonsson, Sigurdsson, Smari, & Young, 2009). In this way, dimensional ADHD, even in subclinical populations, are important to investigate as they continue to experience problems similar to individuals who meet categorical ADHD criteria. Unfortunately, there is a paucity of research examining these effects as subclinical ADHD, particularly in children, is relatively understudied.

In thinking about causality, it is already understood that early substance use initiation in adolescence mimics cognitive deficits and functional impairments seen in individuals with ADHD (Aytaclar, Tarter, Kirisci, & Lu, 1999; Lisdahl, Gilbart, Wright, & Shollenbarger, 2013; Poudel & Gautam, 2017). Specifically, early substance use initiation has been shown to negatively impact cognition, particularly domains of attention, response inhibition, and working memory (Fontes et al., 2011; Gruber, Dahlgren, Sagar, Gönenc, & Killgore, 2012; Gruber, Sagar, Dahlgren, Racine, & Lukas, 2012; Tapert, Granholm, Leedy, & Brown, 2002; Thoma et al., 2011), domains that are often also associated with ADHD. These changes in cognition are thought to be a result of neurodevelopmental changes that occur with the consumption of psychoactive substances (Squeglia, Jacobus, & Tapert, 2009). Specifically, subtle but significant changes in the hippocampus, prefrontal cortex, and white matter have been observed with the onset of adolescent alcohol and cannabis use (Bava & Tapert, 2010; Lisdahl et al., 2013; Squeglia & Gray, 2016; Squeglia et al., 2009). These changes are thought to be at least partially due to the effects of chronic substance use on the dopamine system, which leads to downregulation and alteration of this system in prefrontal regions of the brain (Volkow & Morales, 2015). It is hypothesized that these alterations, particularly during adolescence, impacts

the developing brain attributing to these neurodevelopmental changes (Thorpe, Hamidullah, Jenkins, & Khokhar, 2020). As aberrant development of these same regions has been implicated in ADHD research (Casey & Jones, 2010; Konrad & Eickhoff, 2010; Krain & Castellanos, 2006), it stands to reason that early substance use may potentiate these brain abnormalities during adolescence that accompany persistent ADHD symptoms.

This aligns with the dynamic developmental behavioral theory that posits that ADHD is associated with altered dopaminergic functioning (Sagvolden, Johansen, Aase, & Russell, 2005). While addressing this aberrant dopaminergic functioning through stimulant medication results in decreases in ADHD behaviors (Volkow, Wang, Fowler, & Ding, 2005; Volkow et al., 2001), it's possible that chronic substance use may exacerbate this dopaminergic dysfunction and result in persistent or worsening ADHD symptoms during development. This hypothesis is further strengthened with the growing literature demonstrating that substance use leads to downregulation of dopaminergic receptors in regions such as the ventral striatum (Thiruchselvam, Malik, & Le Foll, 2017). Despite this potential, no known longitudinal studies have examined how substance use exacerbates ADHD symptoms, rather opting to investigate the longitudinal effects of ADHD on substance use (Lee et al., 2011; Molina et al., 2018; Sibley et al., 2014; Zulauf et al., 2014). Due to the susceptibility of brain development during this time, as highlighted above, it is possible that even early light substance use may present subtle changes to the trajectory of ADHD development. Specifically, previous work has demonstrated that as much as 35% of 8-year-olds and 48% of 10-year-olds have engaged in alcohol sipping (Donovan & Molina, 2008). Developmentally, this early sipping behavior has been implicated for increased concern of future heavy substance use (Jackson, Barnett, Colby, & Rogers, 2015), which mimics

observed patterns of early alcohol and non-cannabis illicit drug use initiation in individuals with ADHD (Molina et al., 2018).

Longitudinal studies investigating ADHD and substance use have largely looked at alcohol, cannabis, nicotine, and other illicit substance, but early and continued caffeine use has largely been ignored. This is noteworthy as adolescents with ADHD are about twice as likely to engage in caffeine use compared to adolescents without ADHD (Walker, Abraham, & Tercyak, 2010). Similar to other substance consumption highlighted above, caffeine use impacts dopamine receptor functioning, primarily in frontal-striatal regions of the brain (Cauli & Morelli, 2005; Pandolfo, Machado, Köfalvi, Takahashi, & Cunha, 2013). This increased dopamine is thought to be the mechanism of action that leads to downstream cognitive benefits such as increased alertness, attention, and executive control (Lara, 2010). Interestingly, caffeine use has been proposed as a potential therapeutic tool for ADHD as a review of the literature suggests that, while caffeine is not as efficacious as psychostimulants, it is more effective than placebo (Leon, 2000). Despite this potential therapeutic effect, the one known longitudinal study that looked at caffeine usage in early adolescents showed that energy drink consumption was associated with increased ADHD symptomology at 16 month follow-up even after controlling for coffee consumption and conduct disorder (Marmorstein, 2016). This suggests that despite the potential therapeutic effects of low doses of caffeine, high caffeinated beverages such as energy drinks may increase ADHD symptoms longitudinally. Due to the considerable overlap of mechanism of action between general substance use and caffeine use, as well as the considerable rate of caffeine consumption in youth (Branum, Rossen, & Schoendorf, 2014), further research into the longstanding effects of caffeine use on ADHD symptomatology should be explored.

When examining ADHD symptoms and substance use trajectories, there are other potential confounding factors that also need to be considered; for example, comorbid Conduct Disorder has also been heavily implicated in the relationship between ADHD and substance use. Investigations into the potential effects of Conduct Disorder on ADHD symptoms and substance use have produced discordant results. Some work have demonstrated that the effects of ADHD on substance use are better accounted for by Conduct Disorder (Flory & Lynam, 2003; Lee et al., 2011); however, other work has demonstrated that ADHD still significantly predicts future substance use, even when controlling for conduct problems (Malone, Van Eck, Flory, & Lamis, 2010; Molina & Pelham Jr, 2003). Despite these conflicting findings, both childhood ADHD and Conduct Disorder are considered important predictors for future substance use (Connor, Steeber, & McBurnett, 2010; Erskine et al., 2016; Wymbs et al., 2014). As such, Conduct Disorder is an important consideration in longitudinal analyses of substance use initiation and ADHD symptoms.

A number of health factors have also been heavily linked with both ADHD symptomology and substance use in children and adolescents. One important factor is sleep; poor quality of sleep in youth has been shown to be related to both greater ADHD symptomology (Hvolby, 2015; Schneider, Lam, & Mahone, 2016) and substance use initiation (Johnson & Breslau, 2001; Wong, Brower, & Zucker, 2009). Further, sports and physical activity has also been investigated as a potential mediator and intervention for ADHD (Benzing, Chang, & Schmidt, 2018; Verret, Guay, Berthiaume, Gardiner, & Béliveau, 2012) and substance use development (Moore & Chudley, 2005; Werch, Moore, DiClemente, Bledsoe, & Jobli, 2005). While the existing literature has examined how these health factors have an influence on ADHD and substance use individually, their strong implication in both disorders underlines the

importance of considering all of these potentially confounding or contributing factors when investigating the developmental trajectories of comorbid ADHD and substance use.

Also of interest are potential sex differences between substance use and ADHD symptomology. Even at young ages, males are more likely than females to engage in caffeine use, alcoholic sipping, and standard substance use (Lisdahl et al., in press). Further, sex differences are found within ADHD symptomology with males expressing more severe ADHD symptomology than their female counterparts (Arnett, Pennington, Willcutt, DeFries, & Olson, 2015). These seemingly mirrored patterns within sex differences may suggest similar underlying mechanisms behind substance use and ADHD symptoms as neurobiological factors have been hypothesized to play a role in impacting dopaminergic processes in the brain (Kuhn, 2015). In this way, sex remains an important factor to investigate when examining these relationships.

In order to best examine these questions, data from the Adolescent Brain Cognitive Development (ABCD ®) is being employed. The ABCD study is an open science project incorporating multiple sites across the country to recruit a diverse group of children that represents the national population (Garavan et al., 2018). In this way, the ABCD study is hoping to address homogeneity in study recruitment and produce results that are more generalizable at the national level. Further, one of the express objectives of the ABCD study is to identify factors in youth that are related to and predictive of developmental trajectories (Jernigan & Brown, 2018). In order to accomplish this, participants are being followed over ten years with in-person evaluations taking place every year. This longitudinal study design allows researchers to track how certain variables may impact the trajectory of behaviors and symptomology. By incorporating this data, we hope to more closely examine how early substance use behaviors may alter ADHD symptom trajectories in youth as they progress through adolescence.

Guided by the above literature review, we aim to examine the relationship between early substance use and ADHD symptom trajectory from baseline to two-year follow-up in youth enrolled in the ABCD Study, while accounting for the influence of substance use risk factors, other health factors, stimulant use medication, and demographic factors. Specifically aims include:

1) **Aim 1:** We will examine whether childhood (nine and ten year old) substance use initiation (any alcohol, cannabis, nicotine or other substance use) and caffeine use is significantly associated with baseline ADHD symptomology after controlling for demographics, stimulant medication, Conduct Disorder diagnosis, sleep quality, physical activity, and environmental SU risk factors (parental history SUD, peer substance use, lenient parental SU rules). **Hypothesis:** Youth with baseline substance use will be associated with higher ADHD symptomology at baseline over and above the controlled variables.
2) **Aim 2:** We will examine if longitudinal substance use patterns predict changes in ADHD symptoms over a two-year follow-up period after controlling for demographic data, stimulant medication, related health factors [Conduct Disorder diagnosis, sleep quality, physical activity], and environmental SU risk factors (parental history SUD, peer SU, lenient parental SU rules)] **Hypotheses:** We hypothesize that higher substance use from baseline to two-year follow-up will contribute to worsening of ADHD symptomology in youth over and above demographics, stimulant medication, health factors and general SU risk factors.
3) **Aim 3:** We will conduct exploratory analyses looking at how longitudinal substance use patterns predict changes in ADHD symptoms differently in males and female participants

over a two-year follow-up period after controlling for demographic data, stimulant medication, related health factors [Conduct Disorder diagnosis, sleep quality, physical activity], and environmental SU risk factors (parental history SUD, peer SU, lenient parental SU rules)]. **Hypotheses:** We predict that significant substance use patterns from baseline to two-year follow-up on ADHD symptomology in youth will be more divergent (i.e., higher) in males compared to their female counterparts.

Method

Procedures

Data from the ABCD study (Jernigan & Brown, 2018; Volkow et al., 2018) will be utilized for this study (https://abcdstudy.org/). Participants enrolled at baseline include 11,875 youth that were recruited from 21 different ABCD Study research sites across the country. Participants were recruited during a two-year baseline enrollment when they were nine to ten-years-old. Recruitment occurred through local school systems utilizing a stratified probability sample of eligible schools within the catchment areas (selected based on gender, race, socioeconomic status, and urbanicity; (Garavan et al., 2018)). Participants included singletons siblings, and twins/triplets. A subset of ABCD recruited an additional 200 pair of twins from four sites with longstanding experience with twin research and twin registries (Iacono et al., 2018). After initial phone screening for (basic exclusion criteria), youth and their parents were invited to be a part of the study. Consent and assessment were obtained from both youth and parents separately and in accordance with centralized Institutional Review Board approval and the Helsinki Declaration. Participants and their parents completed in-person questionnaires, interviews, biological assays, cognitive testing, and structural/functional Magnetic Resonance Imaging (MRI) that collected information on a variety of factors (Casey et al., 2018; Lisdahl et

al., 2018; Luciana et al., 2018; Uban et al., 2018; Zucker et al., 2018). Participants and their parents participated in in-person study sessions every year with an abbreviated battery occurring every other year. For the current study, data from baseline, one-year, and two-year visits will be analyzed (NDA Data Release 3.0) and only measures utilized in the current analyses are described below. Thus, changes in participant's substance use and ADHD symptoms will be assessed from ages nine and ten to ages eleven and thirteen.

Measures

Frequency of Youth Substance Use

Substance use measures were designed to establish baseline substance use in youth before full substance use engagement and without teaching or educating youth about substances (Lisdahl et al., 2018). Substance use questionnaires used a gated approach with youth where participants were asked if they had "heard of" a substance (e.g., alcohol, cannabis, nicotine, caffeine, etc.). If participants endorsed having "heard of" a particular substance, then follow-up questions were asked inquiring about participant's experiences with that substance. If youth had heard of a substance previously, then follow-up questions were asked if they have ever tried said substance. If so, follow-up questions assessing 1) age of first use, 2) age of first regular use (defined at baseline as at least weekly use for 6 months), 3) lifetime total quantity in standard units, 4) lifetime maximum (max) dose in standard units, and 5) last date of use (to measure length of abstinence). If participants endorsed engaging in substance use (beyond just a sip of alcohol), a Timeline Follow Back (TLFB) web-based interview was initiated to capture frequency of participants use over the youth's lifetime (Sobell & Sobell, 1992) (Lisdahl et al., 2018). Substance use measurements were followed up every year. Exact measurements of each substance are detailed down below:

Alcohol. As research has indicated that sipping alcohol by age ten is an important predictor of early-onset drinking (Donovan & Molina, 2011). ABCD adapted questions from Jackson et al. (2015) to create the iSay Sip Inventory (iSip) to assess the number of past alcohol sips, whether or not said sipping was part of a religious ceremony or not, the age on sipping onset, the context win which sipping most frequently occurred, the most frequent type of alcohol sipped, and whether or not sipping occurred with supervision or not. This assessment was given at baseline to the youth who were asked to retrospectively recall as many instances as they can remember. If youth endorsed drinking a full alcoholic beverage, then the TLFB was utilized to measure how many standard alcohol drinks (defined as 1 12-ounce bottle of beer or wine cooler, 1 5-ounce glass of wine, or 1 shot (1.25 ounces) of 80-proof alcohol) were consumed over the past 6-months.

Cannabis & Nicotine. Similar to alcohol use, low level cannabis and nicotine use were also measured. Frequency of the number of puffs/tastes of cannabis, (e-)cigarette, smokeless tobacco, age of first experience, where they obtained the substances, and whether it led to additional uses. Further, endorsement of initial cannabis use led to follow-up questions of participant's subjective experience of feeling "high" and estimated THC potency of product used. Endorsement of cannabis or nicotine use led to initiation of the TFLB which measured occurrences of standard cannabis uses (smoked cannabis assessed in grams; blunts measured in grams; edible cannabis measured in occasions; cannabis concentrates measured in occasions; cannabis-infused alcohol drinks measured in standard drinks; cannabis tinctures measured in ml; and synthetic cannabinoids measured in occasions) and standard nicotine uses (cigarettes assessed in number of cigarettes, e-cigarettes assessed in number of occasions, smokeless tobacco assessed in number of pinches, cigars assessed in number of cigars/cigarillos, hookah

assessed in number of hits, pipes assessed in number of hits, and nicotine replacement assessed in number of doses).

Caffeine. Youth that endorsed having heard of caffeine or caffeinated beverages were asked on average over the past 6-months how many caffeinated beverages they consumed a week. Youth were asked to provide average caffeine consumption across five different caffeinated beverage categories and reported in typical serving size (coffee = 8 oz; espresso = 1 shot; green or black tea = 8 oz; soda = 12 oz; energy drink = 5 oz or 5-h energy drink=2 oz). Total average weekly caffeine consumption was combined to provide a summary variable of total average caffeine consumption in the past 6 months at baseline and past month at one-year and two-year follow-up.

Substance Use Risk Factors

Family History. A modified version of the Family History Assessment Module Screener (FHAM-S) was used to assess the presence of substance use dependence in first and second degree blood relatives (Barch et al., 2018). At baseline, parents were led by an RA in a semi-structured style interview and asked whether any family member had experienced problems with using alcohol or other substances. If yes, parents were asked to designate which family members in relation to the child (e.g., full-sibling, maternal uncle, paternal grandparent, etc.) and what problems they experienced. In this way, family history of problems with adaptive functioning due to alcohol or other substances were obtained. Any endorsement of a problem due to substance use was categorized as a family member with the presence of substance use disorder. Presence or absence of a family history for substance use was calculated for both first degree and second-degree family members separately and were used as dichotomous categorical variables.

Peer Use. Youth were asked up to 9 questions at both baseline, one-year, and two-year follow-up about their subjective experience of peer use on a 5-point likert scale (i.e., "none," "a few," "some," "most," or "all"). Similar to other substance use modules, participants were only asked specific questions if they had previously endorsed "hearing about" a particular substance. Depending on gating question endorsement, participants were asked how many of their friend's drink alcohol, get drunk, have problems with alcohol or other drugs, use other drugs, and sell or give drugs to others. Individual ratings of perceptions of peer substance use will be added together to create a composite score of total peer substance use deviance perceptions. Responses on each peer use question will be compiled into a single composite variable with higher responses indicated greater peer substance use endorsement. Questions that were not administered due to gated questions will be scored as "0" or "none."

Parental Rules. Parents of youth were administered a nine-item inventory about their rules regarding substance use in the home and enforcement of rules. At baseline, one-year, and two-year follow-up visits, parents were asked about rules per specific substance and were asked about alcohol, cannabis, and cigarettes. For each substance, parents were asked if they had family rules for that particular substance with options ranging on a 5-point likert scale ("my child is not allowed to drink/use/smoke," to "my child can drink at home whenever they want") with an additional sixth option if they family does not yet have rules about substance use in their household. If parents responded that they did have rules, they were then asked follow-up questions per a substance if these rules are the same for all family members and if there were penalties for violating family rules around substance use. Parents who reported not yet having rules were not asked these follow-up questions. Responses on these items will be aggregated across all substances and turned into a three-point composite variable indicating if families have

no family rules, have some family rules (whether light or restrictive), or have not established family rules around substance use. As such, this variable will be treated as a categorical variable.

Conduct Disorder Diagnosis. The Kiddie Schedule for Affective Disorder and Schizophrenia (KSADS-5) was given to both parent and child to establish baseline, one-year, and two-year follow-up of existing and developing psychopathology (Barch et al., 2018). Parents were self-administered a computerized version of the KSADS-5 with RA support on lifetime and current psychopathology. Children completed an abbreviated battery on internalizing based symptoms. Diagnostic criteria were based on current Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5). In this way, current Conduct Disorder diagnostic status were obtained and utilized for this study.

ADHD Symptoms. Parents also completed the Child Behavior Checklist (CBCL) on their youth's behavior, which provide measures of emotional and behavioral functioning as well as phenotype scores for DSM-5 internalizing and externalizing disorders. The CBCL was utilized to track ADHD symptom development. Dimensional scores for youth's overall ADHD symptoms were obtained at baseline, one-year, and two-year follow-up. (Barch et al., 2018).

Stimulant Medication. Parents were also asked to bring in all medications their children in the study were taking. In this way, a list of current medications were obtained from all youth at each given time point (Barch et al., 2018). Using this medication list, a dichotomous yes/no variables was coded as yes if participants had taken any of the following medications in the past two weeks: Adderall, Concerta, methylphenidate, Ritalin, Focalin, Strattera, amphetamine, Quillivant, guanfacine, Evekeo, atoxmexetine, lisdexamfetamine, Dexedrine, Adzenys, Metadata, Kapyay, clonidine, Intuniv, Daytrana, Methylin, Zenzedi, Catapres, Aptensio,

Cotempla,QuilliChew, bupropion, Wellbutrin, Norpramin, desipramine, imipramine, Tofranil, nortriptyline, Pamelor, & dextroamphetamine.

Health Factors

Physical Activity. Physical exercise was assessed at both baseline, one-year, and two-year follow-up using a modified version of the Youth Risk Behavior Survey (YRBS). The YRBS asked three items about children's current engagement in physical exercise. Questions asked if youth engaged in at least one hour of cardio activity, strength conditioning, and amount of days in PE on the average week. The number of days children engaged in cardio and strengthening activities were aggregated together and included as a continuous variable. Further, parents were asked about the number of sport and non-sport activities that their youth have participated in at baseline, one-year, and two-year follow-up visits (Gorham, Jernigan, Hudziak, & Barch, 2019). Parents were asked to indicate the number of activities their child engaged in, what the activity was, and how many years they have been engaged in the activity. The number of sport and non-sport-based activities were compiled into one variable and used as a continuous variable to stand for general physical activity children are engaging in. Activities in the survey that require less exercise (i.e., playing a musical instrument, drawing/painting, drama, crafting, competitive games like chess/cards/darts, hobbies like stamp collecting, music, and reading) will not be included in the variable.

Sleep. To assess for sleep functioning in children, parents completed the Sleep Disturbance Scale for Children (SDSC). The SDSC is a 26-item rating scale that calculates six major domains of sleep dysfunction (Barch et al., 2018). Total SDSC scores were calculated by adding all six domains together and higher scores indicating larger sleep dysfunction. SDSC was administered at baseline and at subsequent yearly follow-up visits.

Statistical Analyses

Substance Use Variable Notes

To account for data skew and provide set substance use groupings outlined below, early caffeine use will be grouped into none, mild (>0-3 average drinks a week), and moderate (>3 average drinks a week). Similarly, alcohol sips will be broken into none, mild (1-2 alcoholic sips), and moderate (>3). Finally, the composite variable of other substance use will be grouped into none, mild (1 unit), and moderate (>1 unit). It should be noted that while terms of none, mild, and moderate are used for simplicity, both mild and moderate use are still relatively low-level use in comparison to later adolescence substance use.

Descriptive Analyses

Potential differences in demographics (Race, Ethnicity, Sex, Parental Marriage Status, and Household Income), sleep quality, physical activity, between substance use groupings will be investigated with t-test and chi-square analyses. To determine whether baseline substance use is associated with baseline ADHD symptomology, a series of ANCOVAs will be conducted using caffeine use, alcohol sipping, and full substance use groupings on ADHD symptoms. Covariates will include demographic variables, stimulant medication, health factors, and substance use risk factors.

Longitudinal analyses

A series of multilevel linear mixed effect models will be used to test whether substance use patterns across baseline, one-year, and two-year follow-up will predict changes in ADHD symptoms (CBCL ADHD dimensional symptom scores) across baseline, one-year, and two-year follow-up with time being a second level nested variable and within subject variability across time points, ABCD site, and sibling/twin status included as random effects using an unstructured

covariance matrix. Patterns of substance use will be measured as a time-variant variable and will include three separate analyses (e.g., caffeine use, alcohol sipping, total other substance use groupings) to predict ADHD symptom trajectories across all three time points while accounting for covariates include demographic information, stimulant medication, Conduct Disorder diagnosis, health factors, and environmental SU risk variables. Across all longitudinal models, statistical decisions will be made at α=.05. Further, participants will be removed from analyses if missing data across two or more time points or if missing data shows obvious patterns (i.e., not missing at random).

Sex differences analyses

In order to investigate sex differences between substance use patterns and ADHD symptom trajectories, the same series of multilevel linear effect models will be run separately for female and male participants. In this way, a total of six multilevel linear mixed effect models will be run (three in male participants and three in female participants) in order to test whether substance use patterns predict changes in ADHD symptoms across baseline, one-year, and two-year follow-up with time being a second level nested variable and within subject variability across time points, ABCD site, and sibling/twin status included as random effects using an unstructured covariance matrix. Patterns of substance use will continue to be investigated as a time variant variable.

Results

Demographics

After removing for missing data, the study consisted of 5,259 youth. The sample was balanced by sex (52.3% male), on average was 9 years and 11 months old at baseline (M=119.7 months), primarily White (71.9% White), have a parent with a graduate degree (39.3% post-

graduate degree), live in households that make over $100,000 (46.2% >=$100k), and have parents that are married (73.5% married).

Baseline Descriptive Information

Caffeine Use Groupings

At baseline, 28.8% of participants were non-caffeine users, 57.6% were mild caffeine users, and 13.5% were moderate caffeine users. Baseline caffeine groupings significant differed by sex (χ^2= 23.3, p<.001), race (χ^2= 103.4, p<.001), ethnicity (χ^2= 46.8, p<.001), parental education (χ^2= 297.1, p<.001), household income (χ^2= 194.4, p<.001), parental marriage status (χ^2= 167.4, p<.001), and age (F=14.02, p<.001). See Table 1 for additional descriptive information.

Sipping Groupings

At baseline, 75.5% of participants engaged in no sipping behavior, 11.8% engaged in mild sipping behavior, and 12.7% engaged in moderate sipping behavior. Baseline sipping behavior groupings were significantly different by sex (χ^2= 15.1, p<.001), race (χ^2= 46.0, p<.001), ethnicity (χ^2= 11.7, p=0.003), parental education (χ^2= 84.5, p<.001), household income (χ^2= 107.9, p<.001), household income (χ^2= 27.6, p=.002), and age (F=17.15, p<.001). See Table 2 for additional descriptive information.

Total Substance Use Groupings

At baseline, 99.5% of participants were non-substance users, 0.3% of participants were mild substance users, and 0.2% were moderate substance users. Baseline total substance use group significantly differed by sex (χ^2= 8.53, p=.014). Total substance use did not differ by any other demographic variables. See Table 3 for additional descriptive information.

Baseline Multivariate Analyses

Caffeine Use Groupings

After controlling for covariates, ADHD symptoms significantly differed by caffeine group ($F(2,5229)=6.14$, $p=.002$) with higher caffeine usage groups associated with higher ADHD symptomology. Within the model, sleep dysfunction ($F(1,5229)=1040.40$, $p=<.001$), Conduct Disorder diagnosis ($F=153.0$, $p=<.001$), family drug history status ($F(1,5229=15.03$, $p=<.001$), and days of cardio ($F(1,5229)=4.41$, $p=.04$) was significantly related to ADHD symptoms at baseline.

Sipping Groupings

After controlling for covariate variables, sipping behavior groupings were significantly associated with ADHD symptom ($F(2,5229)=4.61$, $p=0.032$) with mild sipping being associated with the highest ADHD symptoms ($M=53.4$) and no sipping associated with the lowest ($M=52.9$). Within the model, peer attitudes toward substance use was significantly related to ADHD symptoms at baseline ($F(1,5229=19.88$, $p<.001$).

Total Substance Use Groupings

Baseline total substance use was not significantly associated with ADHD symptoms after controlling for covariates of interest. Within the model, peer attitudes toward substance use was significantly related to ADHD symptoms at baseline ($F(1,5229=14.86$, $p<.001$).

Longitudinal Analyses

Caffeine Groupings

Overall, ADHD symptoms marginally decreased at one-year ($\beta=-0.27$, $p=0.054$) but not two-year follow-up ($\beta=-0.31$, $p=0.141$), regardless of group. Moderate caffeine use significantly predicted higher ADHD symptoms at two-year ($\beta=0.46$, $p=0.025$) but not one-year follow-up ($\beta=0.11$, $p=0.425$; see figure 1); mild caffeine use did not significantly predict ADHD symptoms

at follow up (β=-0.03, p=0.853; β=0.32, p=0.111). Several covariates of interest significantly contributed to the model between caffeine use groupings and ADHD symptomology. The significant variables of note included family drug history status (β=0.84, p<.001), sleep dysfunction (β=0.18, p<.001), Conduct Disorder diagnosis (β=2.69, p<.001), peer attitudes toward use (β=0.39, p=0.001), and stimulant medication use (β=2.18, p<.001).

Sipping Groupings

Sipping behavior did not significantly predict ADHD symptomology at one-year (β=-0.29., p=0.234; β=0.08, p=0.717) or two-year follow-up (β=-0.03, p=0.906; β=0.01, p=0.972). Several covariates of interest significantly contributed to the model between sipping behavior groupings and ADHD symptomology. The significant variables of note included family drug history status (β=0.84, p<.001), sleep dysfunction (β=0.18, p<.001), Conduct Disorder diagnosis (β=2.67, p<.001), peer attitudes toward use (β=0.40, p=0.001), and stimulant medication use (β=2.17, p<.001).

Total Substance Use Groupings

Overall, ADHD symptoms significantly decreased at one-year (β=-0.25, p=0.019) but not two-year follow-up (β=0.20, p=0.282) regardless of group. Moderate total substance use significantly predicted higher ADHD symptoms at two-year follow-up (β=3.58, p=0.003) but not one-year follow-up (β=-0.15, p=0.905); mild total substance use did not significantly predict ADHD symptomology at one-year (β=-2.85, p=0.254) or two-year follow-up (β=0.04, p=0.983). Several covariates of interest significantly contributed to the model between total substance use groupings and ADHD symptomology. The significant variables of note included family drug history status (β=0.84, p<.001), sleep dysfunction (β=0.18, p<.001), Conduct Disorder diagnosis

(β=2.69, p<.001), peer attitudes toward use (β=0.39, p=0.001), and stimulant medication use (β=2.17, p<.001).

Sex analyses

Male Participants. Moderate total substance use groupings significantly predicted increased ADHD symptomology at two-year follow-up (β=5.11, p=.001) but not one-year follow-up (β=0.22, p=0.887). No other substance use groupings significantly predicted longitudinal ADHD symptoms in male participants.

Female Participants. Moderate caffeine use predicted significantly higher ADHD symptoms at two-year follow-up (β=0.78, p=0.007) but not one-year follow up in female participants (β=0.34, p=0.072). No other substance use groupings significantly predicted longitudinal ADHD symptoms in female participants.

Discussion

In our study we aimed to investigate the relationship between substance use and ADHD symptomology at baseline and over a two-year period within participants enrolled in the ABCD study as they transition from late childhood into early adolescence. At baseline, ADHD symptoms were significantly linked with baseline caffeine use and sipping behavior, after controlling for demographic, stimulant medication, and associated health and substance use risk factors. Specifically, higher recent caffeine usage and mild sipping behavior was associated with greater ADHD symptoms. Longitudinally, moderate but not mild caffeine use was associated with greater ADHD symptoms at two-year follow-up compared to no caffeine use. Similarly, moderate but not mild total substance use was associated with greater ADHD symptoms at two-year follow up compared to no substance use. Additional sex analyses revealed that total substance use was linked with increased ADHD symptomology in males, while caffeine use was

linked to increased ADHD symptomology in females after controlling for demographic, stimulant medication, health factors, and substance use risk factors.

While statistically significant, differences were observed at baseline between caffeine groupings and ADHD symptoms as higher caffeine usage demonstrated a significant relationship with increased ADHD symptoms. These patterns continued to diverge longitudinally as greater caffeine use led to greater ADHD symptomology over the two-year study period. In this way, moderate caffeine use (defined here as 3 or more standard caffeinated drinks a week) was associated with increased ADHD symptomology over time, supporting the hypothesis that early substance use promotes increased ADHD symptom trajectories. This relationship may be explained by a number of different mechanisms (Garrett & Griffiths, 1997). Neurodevelopmental effects of caffeine on adenosine receptors with downstream behavioral changes through changes in dopaminergic neuromodulation may play a role in these changes in ADHD symptom trajectory (Cauli & Morelli, 2005). Further increased caffeine usage in late childhood may simply be an early indicator of increased genetic risk for impulsivity and sensation seeking. One epidemiological study investigating genetic influence on caffeine use indicated that caffeine usage was not only heritable but explains part of the variance in individual's personalities (Czajkowski et al., 2020). Building off this, previous studies have linked caffeine usage with increased risky behavior in young adult and late adolescent populations (Azagba, Langille, & Asbridge, 2014; Grant & Chamberlain, 2018; Mahoney et al., 2019; Miller, 2008). While these studies suggest the influence of genetic risk in caffeine use and impulsivity, direct studies linking these factors have not been done. Further, despite the prevalence of caffeine use (Lisdahl et al., in press; Miller, Dermen, & Lucke, 2018), caffeine research in late childhood and earlier adolescence is relatively understudied; indicating a need for more research to better understand

this relationship. Also of note, despite evidence of sleep being a mediator between caffeine use and ADHD symptoms (Cusick, Langberg, Breaux, Green, & Becker, 2020), moderate caffeine findings were found while accounting for sleep dysfunction. This further implies the complexity of this relationship and urges for greater investigation into the neuropsychological and genetic underpinnings of caffeine use and ADHD symptoms.

Despite there being no significant differences at baseline between total substance use groupings and ADHD symptoms, moderate total substance use (defined as 3 or more standard substance uses) was associated with increased ADHD symptoms over two years. It is worth noting, that moderate- but not mild- substance use contributed to ADHD symptomology over time, suggesting a dose-dependent relationship. As mentioned earlier, past studies have demonstrated a link between adolescent substance use and ADHD diagnosis (Lee et al., 2011; Molina et al., 2018; Molina & Pelham Jr, 2003; Sibley et al., 2014; Wilens et al., 2011); while the directionality of these findings have been difficult to parse out, our findings suggest that early substances use behavior may extend upon or even increase ADHD symptoms overtime. In this way, very early substance use is already contributing to the relationship between substance use and ADHD symptom severity, even after accounting for other comorbid health factors, Conduct Disorder, and current stimulant medication usage. While substance use research has traditionally examined the roles of substance use starting in adolescence (Chassin et al., 2004; Lisdahl et al., 2013; Squeglia et al., 2009; Thorpe et al., 2020), our findings suggest that even earlier, low-level substance use initiation should be considered as it is playing a role in these neurodevelopmental trajectories. Indeed, one study that examined childhood ADHD and early substance use trajectories demonstrated that while ADHD status alone did not significantly impact alcohol, cannabis use, or other illicit drug use trajectories, early substance use in individuals with ADHD

did lead to increased substance use trajectories (Molina et al., 2018). Our findings extend this literature by considering that early substance use not only lead to more severe substance use trajectories, but also appears to contribute to increased ADHD symptom trajectories even within subclinical samples.

There are a few different mechanisms that may underlie this relationship between moderate substance use and ADHD symptom severity from childhood to pre/early adolescence. Animal models have indicated that exposure to substances during this sensitive period may lead to changes in neurotransmission (Hoegberg, Lomazzo, Lee, & Perry, 2015; Shram, Funk, Li, & Lê, 2007; Zhang, Feng, & Chergui, 2015) including dopaminergic systems (Pascual, Boix, Felipo, & Guerri, 2009). Greater and repeated exposure to substances during this sensitive period may lead to possible changes in dopamine signaling (Thorpe et al., 2020; Volkow & Morales, 2015). These findings are further underlined by human studies showing that earlier age of onset related to abnormal structural brain morphometry (Lisdahl et al., 2013), particularly in individual with ADHD (Lisdahl et al., 2016). It has long been understood that substance use severity is associated with altered dopaminergic systems (Esposito-Smythers, Spirito, Rizzo, McGeary, & Knopik, 2009; Leyton & Vezina, 2013; Noble, 1996). Additionally, recent positron emission tomography (PET) study findings have shown that chronic substance use is linked with reduced receptor availability and pre-synaptic hypo-functioning within dopamine systems (Thiruchselvam et al., 2017). Further, links between neurodevelopmental impulsivity and substance use through dysregulation of these dopaminergic pathways have been discussed (Leyton & Vezina, 2014). These findings all lend support to the dynamic developmental theory which posits that altered dopaminergic systems are a core mechanism behind ADHD development (Sagvolden et al., 2005), and our findings open up the possibility that even low-

level early substance use initiation may influence this development. Indeed, while low level use is rarely if ever studied, emerging evidence is beginning to suggest that even instances of one or two exposures to substances like cannabis can lead to structural and cognitive effects (Orr et al., 2019). Further in line with the dynamic developmental theory of ADHD is the idea that ADHD symptom expression is dependent on environmental factors and the time at which they are produced (Sagvolden et al., 2005). While low level use did not seem to significantly impact trajectories one year from baseline, it did impact trajectories two years out indicating increased dopamine sensitization as participants age into early adolescence (Thorpe et al., 2020). However, it must be noted that dopamine levels were not measured in this study, therefore; this mechanism would need to be confirmed in future studies. Further, while much of this research has largely centered on alcohol, nicotine, cannabis, and other illicit substance use, dopamine receptor involvement has similarly been linked with caffeine usage and thus it is hypothesized that similar mechanisms may be at play (Garrett & Griffiths, 1997).

Another possible hypothesis for the mechanism between early substance use and increased substance use is genetic. Strong shared genetic variance between ADHD symptoms and substance use has been demonstrated within adults (Derks, Vink, Willemsen, Van Den Brink, & Boomsma, 2014). Further, retrospective studies have linked genetic risk for externalizing behaviors on alcohol consumption starting in early adolescents and reaching a peak around mid-adolescence (Kendler, Gardner, & Dick, 2011). While these studies were demonstrated in older populations than our cohort, these early substance use patterns and increased ADHD symptomology may indicate early signs of this shared genetic variance. Further, strong links between genetics and shared environmental factors (such as peer deviance, availability of alcohol) of substance use and ADHD could also explain genetic by environmental

interactions that would further contribute to this relationship (Geels, Vink, Van Beijsterveldt, Bartels, & Boomsma, 2013; Kendler et al., 2011; Laucht, Hohm, Esser, Schmidt, & Becker, 2007). However, as genetic information was not included in this study, we can only hypothesize on its potential influence. Future work should utilize genetic samples of children and adolescents to further investigate genetic contributions to substance use and ADHD symptomology.

Despite being linked at baseline, sipping behaviors did not impact ADHD trajectories from childhood to pre/early adolescence. This relationship implies that while early alcohol sipping is linked with ADHD symptoms at baseline, changes in these sipping behaviors do not impact overall ADHD symptom trajectories into early adolescence. The fact that sipping behavior was not related to ADHD symptoms overtime may suggest that low-level sipping does not alter systems linked with ADHD severity, such as dopaminergic functioning (Swanson et al., 2007). Further, moderate sipping behavior as defined as 3 or more sips, may be too low to capture any subtle long-term effects on attentional processes. Another possible explanation is that low-level sipping is more normative behavior; indeed, 22.5% of youth in the overall ABCD sample engaged in some level of sipping already at baseline (Lisdahl et al., in press). Therefore, examining the impact of consuming full alcoholic drink may be more appropriate in examining these longitudinal trajectories.

While the relationships between substance use and ADHD symptoms were significant over and above other factors included in the model, health and diagnostic factors such as positive family drug status, sleep dysfunction, peer attitudes toward substance use, stimulant medication use, and Conduct Disorder diagnosis were also shown to be linked with substance use and ADHD symptoms. Due to the existing body of research on these factors (Benzing et al., 2018; Flory & Lynam, 2003; Hvolby, 2015; Lee et al., 2011; Malone et al., 2010; Molina & Pelham Jr,

2003; Moore & Chudley, 2005; Wong et al., 2009) and significant findings within our models, future work should continue to examine their roles in the relationships between substance use and ADHD symptomology as they all significantly contributed to increased ADHD symptoms across all substance use groupings.

Also of interest was the relationship of biological sex on substance use and ADHD symptoms. Interestingly when separated by sex, substance use findings remained only for the male participants while the caffeine use findings were specific to female participants. Considering moderate total substance use was predominately male, male participants driving the relationship between moderate total substance use and ADHD symptomology is not that surprising. It appears that even at early age, male rather than female children are more likely to engage in slightly more substance use initiation (Lisdahl et al., in press). Although this use is still small, as highlighted above, even one or two uses can lead to significant changes in brain morphometry and cognition (Orr et al., 2019) and may provide an explanation for the increased rates of ADHD symptoms in males compared to females (Arnett et al., 2015). Interestingly, caffeine findings only remained significant within the female participants compared to male participants. While more male participants engaged in moderate caffeine use across all time points (baseline=57.6%, one-year=57.9%, and two-year=56.9%), the significant relationship between caffeine and ADHD symptoms within female participants suggests a separate causality than the mechanism driving substance use and ADHD symptoms in males. One possible explanation is the role of neurobiological factors, such as the effects of estradiol on dopaminergic processes, that may drive these sex differences (Kuhn, 2015). Further, another explanation could lie in the type of symptom presentation. Female rather than male children are more likely to display inattentive ADHD symptoms (American Psychiatric Association, 2013).

As caffeine is associated with increased sustained attention (Smith, 2002), it's possible that female participants with higher ADHD symptoms may be seeking out higher levels of caffeine to "self-medicate." Previous literature has suggested benefits of caffeine as a treatment for ADHD (Leon, 2000); however, this review largely draws from male samples. More research is necessary to start to parse out sex differences in caffeine use and ADHD symptomology.

Overall, our findings point to a need for clinicians and researchers to put more focus on early caffeine and substance use in children. Study results suggest small but significant differences in ADHD symptoms within children who engage in caffeine and substance use are already being demonstrated by late childhood. This emphasizes a need to start focusing on early use, even if early use appears small relative to later use in adolescence and adulthood. At its earliest, most research investigates substance use starting in adolescence (Casey & Jones, 2010; Lisdahl et al., 2013; Squeglia & Gray, 2016). Similarly, clinical assessment and intervention work largely focuses on adolescent substance use rather than their younger peers (Hogue, Henderson, Becker, & Knight, 2018; Kulak & Griswold, 2019; Thurstone, Hull, Timmerman, & Emrick, 2017). Greater importance needs to be made on validating assessment and intervention of early substance use even before adolescence. Further, emphasis on early prevention could help to mitigate early childhood substance usage. Currently, there are no known federal guidelines for child caffeine usage in the United States despite the potential deleterious effects. Continued work into preventative measures of childhood caffeine and substance use are needed.

While this research provides a novel perspective on substance use and ADHD symptom relationships, there are several limitations to the study. While the study highlights the benefit of examining dimensional ADHD symptom trajectories, the overall sample was largely subclinical and therefore does not truly reflect ADHD research. Future investigations should examine

ADHD symptom trajectories within ADHD diagnosed populations to see if similar patterns emerge. Further, our study examined overall ADHD symptoms but does not capture the difference in ADHD symptom subtype (i.e., hyperactive and inattentive symptoms (American Psychiatric Association, 2013)). As previous work has demonstrated different patterns of substance use between ADHD subtypes (Molina & Pelham Jr, 2003), future work should also examine these relationship between differences in subtype symptoms. Additionally, our study examined these relationships within a linear direction, only examining substance use on ADHD symptoms. Due to the complexity of the relationship between ADHD and substance, bidirectional models such as structural equation model are needed to better understand these variables. Finally, while we speculated on the mechanisms behind our findings, our study falls short of actually measuring the factors that may underpin this relationship. Future work should examine dopaminergic levels as well as genetic factors that may better explain the connection between substance use and ADHD symptoms.

At baseline, ADHD symptoms significantly differed by caffeine use and alcoholic sipping with higher substance use related to higher ADHD symptoms. Longitudinally, moderate caffeine use (defined as 3 of more standard caffeinated drinks a week) and moderate total substance use (defined as more than 1 full standard substance usage) were related to increased ADHD symptoms two years after baseline over and above demographics, stimulant medication, health factors, and other substance use risk factors. These findings suggest that even light and early use can start to impact ADHD symptom trajectories within a normative population. Further, analyses divided by sex suggest that standard substance use was specific to male children while caffeine use findings were specific to female children suggesting sex differences. More work is needed to investigate the effects of early substance use on ADHD symptomology

to further elucidate the impact early use has in adolescent trajectories as well as differences in these patterns by biological sex.

Figure 1.
Longitudinal Caffeine Use on ADHD Symptoms

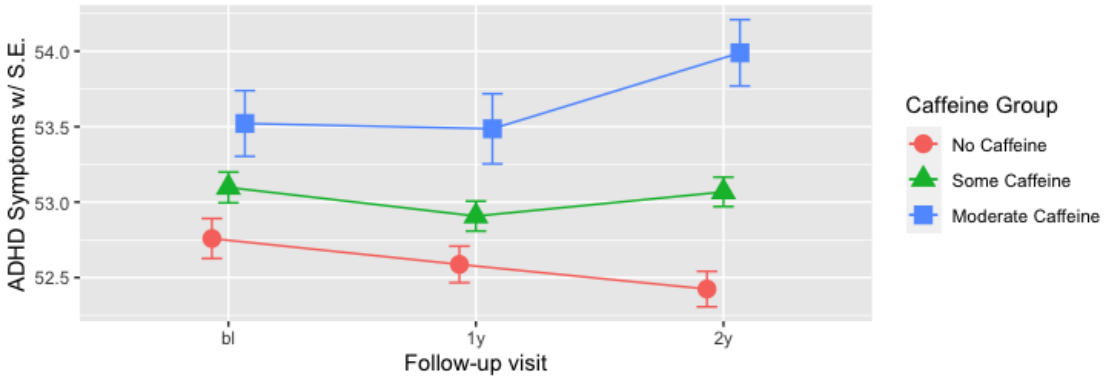

Figure 1. Linear graphs depicting longitudinal trajectories across all three time points and separated by caffeine group status. Bl=baseline visit, 1y=1 year visit, 2y=2 year visit. Error bars represent standard error.

Figure 2.
Total Substance Use on ADHD Symptoms

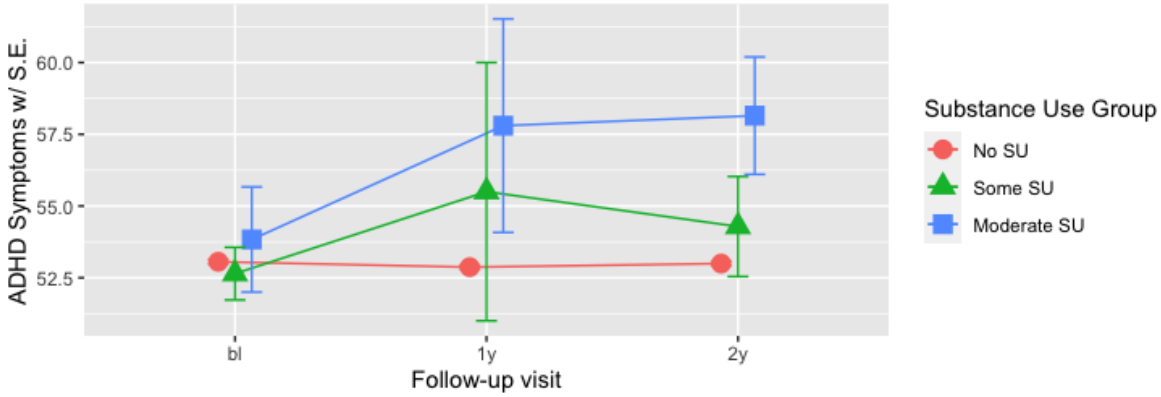

Figure 2. Linear graphs depicting longitudinal trajectories across all three time points and separated by substance use group status. SU=Substance use, bl=baseline visit, 1y=1 year visit, 2y=2 year visit. Error bars represent standard error.

Table 1.
Baseline Caffeine Groupings

	No Caffeine Use (N=1516)	Mild Caffeine Use (N=3031)	Moderate Caffeine Use (N=712)
***Age (Months M,SD)**	118.9 (7.4)	120.0 (7.5)	120.2 (7.3)
***Race (% White)**	77.8%	70.5%	65.0%
(% Black)	6.1%	10.7%	18.9%
(% Asian)	2.9%	2.3%	1.1%
(% Other/Mixed)	13.1%	6.5%	14.9%
***Ethnicity (% Not Hispanic)**	89.6%	81.9%	82.6%
***Sex (% Male)**	47.6%	53.5%	57.6%
***Parental Education (% post-grad)**	49.7%	37.9%	22.8%
***Household Income (% >=100K)**	55.7%	45.2%	30.8%
***Parental Marriage Status (% Married)**	83.2%	71.6%	60.7%
***Sleep Dysfunction (M,SD)**	35.8 (7.2)	36.3 (8.0)	37.6 (8.6)
***Conduct Disorder Diagnosis (% with Diagnosis)**	1.9%	2.1%	3.7%
***Family Alcohol History (% Positive History)**	10.6%	15.1%	19.5%
Family Drug History (% Positive History)	31.5%	32.1%	31.0%
***Household Rules (% Rules are No)**	78.6%	80.1%	80.3%
***Peer Substance Use (% Peers Use)**	3.3%	4.4%	8.6%
***Days Strength Training (Days M, SD)**	1.8 (2.0)	1.9 (1.9)	2.2 (2.2)
***Days Cardio Activity (Days M, SD)**	3.8 (2.3)	3.6 (2.3)	3.6 (2.3)
***Sports Engagement (# of Sports M,SD)**	1.8 (1.5)	1.7 (1.5)	1.4 (1.5)
***Stimulant Medications (% on stimulant medications)**	4.2%	5.1%	7.0%

Notes. *Denotes significant differences in group via t-test or chi-square testing with statistical decision made at α=.05. M=mean, SD=Standard Deviation.

Table 2.
Baseline Sipping Groupings

	No Sipping (N=3971)	Mild Sipping (N=622)	Moderate Sipping (N=666)
***Age (Months M,SD)**	119.5 (7.4)	120.2 (7.6)	120.6 (7.3)
***Race (% White)**	70.3%	74.9%	78.4%
(% Black)	12.1%	7.1%	4.7%
(% Asian)	2.2%	2.4%	3.0%
(% Other/Mixed)	15.4%	15.6%	14.0%
***Ethnicity (% Not Hispanic)**	87.2%	85.5%	88.3%
***Sex (% Male)**	53.5%	53.1%	59.2%
***Parental Education (% post-grad)**	38.3%	46.3%	49.0%
***Household Income (% >=100K)**	44.6%	54.7%	60.5%
***Parental Marriage Status (% Married)**	75.8%	73.0%	80.5%
Sleep Dysfunction (M,SD)	36.3 (8.1)	36.4 (7.6)	36.3 (7.0)
Conduct Disorder Diagnosis (% with Diagnosis)	2.3%	2.6%	2.4%
Family Alcohol History (% Positive History)	15.1%	13.8%	15.2%
Family Drug History (% Positive History)	33.9%	30.2%	29.7%
***Household Rules (% Rules are No)**	85.4%	76.7%	71.9%
***Peer Substance Use (% Peers Use)**	4.0%	5.6%	8.3%
***Days Strength Training (Days M, SD)**	1.9 (2.0)	1.7 (2.0)	1.8 (2.0)
Days Cardio Activity (Days M, SD)	3.6 (2.3)	1.7 (2.3)	3.8 (2.2)
***Sports Engagement (# of Sports M,SD)**	1.6 (1.5)	1.8 (1.4)	1.9 (1.4)
Stimulant Medications (% on stimulant medications)	5.2%	5.3%	5.6%

Notes. *Denotes significant differences in group via t-test or chi-square testing with statistical decision made at α=.05. M=mean, SD=Standard Deviation.

Table 3.
Total Substance Use Groupings

	No Substance Use (N=5233)	Mild Substance Use (N=14)	Moderate Substance Use (N=12)
Age (Months M,SD)	119.7 (7.4)	120.9 (8.0)	120.5 (7.1)
Race (% White)	71.9%	78.6%	66.7%
(% Black)	10.5%	7.1%	8.3%
(% Asian)	2.4%	0.0%	0.0%
(% Other/Mixed)	15.2%	14.3%	25.0%
Ethnicity (% Not Hispanic)	84.2%	85.7%	75.0%
***Sex (% Male)**	52.2%	78.6%	83.3%
Parental Education (% post-grad)	39.3%	35.7%	25.0%
Household Income (% >=100K)	46.3%	57.1%	25.0%
Parental Marriage Status (% Married)	73.5%	78.6%	50.0%
Sleep Dysfunction (M,SD)	36.3 (7.9)	37.6 (4.9)	34.4 (8.0)
Conduct Disorder Diagnosis (% with Diagnosis)	2.3%	0%	0%
Family Alcohol History (% Positive History)	14.4%	14.3%	33.3%
Family Drug History (% Positive History)	31.7%	42.9%	50.0%
Household Rules (% Rules are No)	79.8%	78.6%	66.7%
***Peer Substance Use (% Peers Use)**	4.6%	0.0%	25.0%
Days Strength Training (Days M, SD)	1.9 (2.0)	1.9 (2.5)	3.0 (2.4)
Days Cardio Activity (Days M, SD)	3.7 (2.3)	3.8 (2.4)	4.8 (2.3)
Sports Engagement (# of Sports M,SD)	1.7 (1.5)	1.6 (1.2)	1.2 (0.9)
Stimulant Medications (% on stimulant medications)	5.1%	14.3%	16.7%

Notes. *Denotes significant differences in group via t-test or chi-square testing with statistical decision made at α=.05. M=mean, SD=Standard Deviation.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*: American Psychiatric Pub.

Arnett, A. B., Pennington, B. F., Willcutt, E. G., DeFries, J. C., & Olson, R. K. (2015). Sex differences in ADHD symptom severity. *Journal of Child Psychology and Psychiatry, 56*(6), 632-639.

Arnold, L. E., Hodgkins, P., Kahle, J., Madhoo, M., & Kewley, G. (2020). Long-term outcomes of ADHD: academic achievement and performance. *Journal of attention disorders, 24*(1), 73-85.

Aytaclar, S., Tarter, R. E., Kirisci, L., & Lu, S. (1999). Association between hyperactivity and executive cognitive functioning in childhood and substance use in early adolescence. *Journal of the American Academy of Child & Adolescent Psychiatry, 38*(2), 172-178.

Azagba, S., Langille, D., & Asbridge, M. (2014). An emerging adolescent health risk: Caffeinated energy drink consumption patterns among high school students. *Preventive Medicine, 62*, 54-59.

Barch, D. M., Albaugh, M. D., Avenevoli, S., Chang, L., Clark, D. B., Glantz, M. D., . . . Yurgelun-Todd, D. (2018). Demographic, physical and mental health assessments in the adolescent brain and cognitive development study: Rationale and description. *Developmental cognitive neuroscience, 32*, 55-66.

Bava, S., & Tapert, S. F. (2010). Adolescent brain development and the risk for alcohol and other drug problems. *Neuropsychology review, 20*(4), 398-413.

Benzing, V., Chang, Y.-K., & Schmidt, M. (2018). Acute physical activity enhances executive functions in children with ADHD. *Scientific reports, 8*(1), 1-10.

Branum, A. M., Rossen, L. M., & Schoendorf, K. C. (2014). Trends in caffeine intake among US children and adolescents. *Pediatrics, 133*(3), 386-393.

Brown, A. J., & Casey, B. M. (2016). Subclinical ADHD-symptoms are associated with executive-functioning and externalizing problems in college students without ADHD-diagnoses. *Journal of Educational and Developmental Psychology, 6*(1), 204-220.

Bussing, R., Mason, D. M., Bell, L., Porter, P., & Garvan, C. (2010). Adolescent outcomes of childhood attention-deficit/hyperactivity disorder in a diverse community sample. *Journal of the American Academy of Child & Adolescent Psychiatry, 49*(6), 595-605.

Casey, B., Cannonier, T., Conley, M. I., Cohen, A. O., Barch, D. M., Heitzeg, M. M., . . . Garavan, H. (2018). The adolescent brain cognitive development (ABCD) study: imaging acquisition across 21 sites. *Developmental cognitive neuroscience, 32*, 43-54.

Casey, B., & Jones, R. M. (2010). Neurobiology of the adolescent brain and behavior: implications for substance use disorders. *Journal of the American Academy of Child & Adolescent Psychiatry, 49*(12), 1189-1201.

Cauli, O., & Morelli, M. (2005). Caffeine and the dopaminergic system. *Behavioural pharmacology, 16*(2), 63-77.

Caye, A., Swanson, J., Thapar, A., Sibley, M., Arseneault, L., Hechtman, L., . . . Rohde, L. A. (2016). Life span studies of ADHD—conceptual challenges and predictors of persistence and outcome. *Current psychiatry reports, 18*(12), 111.

Chassin, L., Hussong, A., Barrera Jr, M., Molina, B. S., Trim, R., & Ritter, J. (2004). Adolescent substance use.

Christensen, K. E., & Lundwall, R. A. (2018). Errors on a computer task and subclinical symptoms of attention-deficit/hyperactivity disorder (ADHD). *Scandinavian journal of psychology, 59*(5), 511-517.

Connor, D. F., Steeber, J., & McBurnett, K. (2010). A review of attention-deficit/hyperactivity disorder complicated by symptoms of oppositional defiant disorder or conduct disorder. *Journal of Developmental & Behavioral Pediatrics, 31*(5), 427-440.

Cusick, C. N., Langberg, J. M., Breaux, R., Green, C. D., & Becker, S. P. (2020). Caffeine use and associations with sleep in adolescents with and without ADHD. *Journal of pediatric psychology, 45*(6), 643-653.

Czajkowski, N., Kendler, K. S., Torvik, F. A., Ystrom, E., Rosenström, T., Gillespie, N. A., & Reichborn-Kjennerud, T. (2020). Caffeine consumption, toxicity, tolerance and withdrawal; shared genetic influences with normative personality and personality disorder traits. *Experimental and Clinical Psychopharmacology*.

Danielson, M. L., Bitsko, R. H., Ghandour, R. M., Holbrook, J. R., Kogan, M. D., & Blumberg, S. J. (2018). Prevalence of parent-reported ADHD diagnosis and associated treatment among US children and adolescents, 2016. *Journal of Clinical Child & Adolescent Psychology, 47*(2), 199-212.

Derks, E., Vink, J., Willemsen, G., Van Den Brink, W., & Boomsma, D. (2014). Genetic and environmental influences on the relationship between adult ADHD symptoms and self-reported problem drinking in 6024 Dutch twins. *Psychological medicine, 44*(12), 2673.

Donovan, J. E., & Molina, B. S. (2008). Children's introduction to alcohol use: sips and tastes. *Alcoholism: Clinical and Experimental Research, 32*(1), 108-119.

Donovan, J. E., & Molina, B. S. (2011). Childhood risk factors for early-onset drinking. *Journal of studies on alcohol and drugs, 72*(5), 741-751.

Erskine, H. E., Norman, R. E., Ferrari, A. J., Chan, G. C., Copeland, W. E., Whiteford, H. A., & Scott, J. G. (2016). Long-term outcomes of attention-deficit/hyperactivity disorder and conduct disorder: a systematic review and meta-analysis. *Journal of the American Academy of Child & Adolescent Psychiatry, 55*(10), 841-850.

Esposito-Smythers, C., Spirito, A., Rizzo, C., McGeary, J. E., & Knopik, V. S. (2009). Associations of the DRD2 TaqIA polymorphism with impulsivity and substance use: preliminary results from a clinical sample of adolescents. *Pharmacology Biochemistry and Behavior, 93*(3), 306-312.

Faraone, S. V., Biederman, J., & Mick, E. (2006). The age-dependent decline of attention deficit hyperactivity disorder: a meta-analysis of follow-up studies. *Psychological medicine, 36*(2), 159-165.

Flory, K., & Lynam, D. R. (2003). The relation between attention deficit hyperactivity disorder and substance abuse: what role does conduct disorder play? *Clinical child and family psychology review, 6*(1), 1-16.

Fontes, M. A., Bolla, K. I., Cunha, P. J., Almeida, P. P., Jungerman, F., Laranjeira, R. R., . . . Lacerda, A. L. (2011). Cannabis use before age 15 and subsequent executive functioning. *The British Journal of Psychiatry, 198*(6), 442-447.

Fredriksen, M., Dahl, A. A., Martinsen, E. W., Klungsoyr, O., Faraone, S. V., & Peleikis, D. E. (2014). Childhood and persistent ADHD symptoms associated with educational failure and long-term occupational disability in adult ADHD. *ADHD Attention Deficit and Hyperactivity Disorders, 6*(2), 87-99.

Garavan, H., Bartsch, H., Conway, K., Decastro, A., Goldstein, R., Heeringa, S., . . . Zahs, D. (2018). Recruiting the ABCD sample: design considerations and procedures. *Developmental cognitive neuroscience, 32,* 16-22.

Garrett, B. E., & Griffiths, R. R. (1997). The role of dopamine in the behavioral effects of caffeine in animals and humans. *Pharmacology Biochemistry and Behavior, 57*(3), 533-541.

Geels, L. M., Vink, J. M., Van Beijsterveldt, C. E., Bartels, M., & Boomsma, D. I. (2013). Developmental prediction model for early alcohol initiation in Dutch adolescents. *Journal of studies on alcohol and drugs, 74*(1), 59-70.

Gorham, L. S., Jernigan, T., Hudziak, J., & Barch, D. M. (2019). Involvement in sports, hippocampal volume, and depressive symptoms in children. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 4*(5), 484-492.

Grant, J. E., & Chamberlain, S. R. (2018). Caffeine's influence on gambling behavior and other types of impulsivity. *Addictive Behaviors, 76*, 156-160.

Gruber, S. A., Dahlgren, M. K., Sagar, K. A., Gönenc, A., & Killgore, W. D. (2012). Age of onset of marijuana use impacts inhibitory processing. *Neuroscience letters, 511*(2), 89-94.

Gruber, S. A., Sagar, K. A., Dahlgren, M. K., Racine, M., & Lukas, S. E. (2012). Age of onset of marijuana use and executive function. *Psychology of Addictive Behaviors, 26*(3), 496.

Gudjonsson, G. H., Sigurdsson, J. F., Smari, J., & Young, S. (2009). The relationship between satisfaction with life, ADHD symptoms, and associated problems among university students. *Journal of attention disorders, 12*(6), 507-515.

Hechtman, L., Swanson, J. M., Sibley, M. H., Stehli, A., Owens, E. B., Mitchell, J. T., . . . Jensen, P. S. (2016). Functional adult outcomes 16 years after childhood diagnosis of attention-deficit/hyperactivity disorder: MTA results. *Journal of the American Academy of Child & Adolescent Psychiatry, 55*(11), 945-952. e942.

Hoegberg, B. G., Lomazzo, E., Lee, N. H., & Perry, D. C. (2015). Regulation of α4β2α5 nicotinic acetylcholinergic receptors in rat cerebral cortex in early and late adolescence: sex differences in response to chronic nicotine. *Neuropharmacology, 99*, 347-355.

Hogue, A., Henderson, C. E., Becker, S. J., & Knight, D. K. (2018). Evidence base on outpatient behavioral treatments for adolescent substance use, 2014–2017: Outcomes, treatment delivery, and promising horizons. *Journal of Clinical Child & Adolescent Psychology, 47*(4), 499-526.

Hvolby, A. (2015). Associations of sleep disturbance with ADHD: implications for treatment. *ADHD Attention Deficit and Hyperactivity Disorders, 7*(1), 1-18.

Iacono, W. G., Heath, A. C., Hewitt, J. K., Neale, M. C., Banich, M. T., Luciana, M. M., . . . Bjork, J. M. (2018). The utility of twins in developmental cognitive neuroscience research: How twins strengthen the ABCD research design. *Developmental cognitive neuroscience, 32*, 30-42.

Jackson, K. M., Barnett, N. P., Colby, S. M., & Rogers, M. L. (2015). The prospective association between sipping alcohol by the sixth grade and later substance use. *Journal of studies on alcohol and drugs, 76*(2), 212-221.

Jernigan, T., & Brown, S. (2018). ABCD Consortium Coordinators. Introduction. *Developmental cognitive neuroscience, 32*, 1-3.

Johnson, E. O., & Breslau, N. (2001). Sleep problems and substance use in adolescence. *Drug and alcohol dependence, 64*(1), 1-7.

Kendler, K., Gardner, C., & Dick, D. (2011). Predicting alcohol consumption in adolescence from alcohol-specific and general externalizing genetic risk factors, key environmental exposures and their interaction. *Psychological medicine, 41*(7), 1507.

Konrad, K., & Eickhoff, S. B. (2010). Is the ADHD brain wired differently? A review on structural and functional connectivity in attention deficit hyperactivity disorder. *Human brain mapping, 31*(6), 904-916.

Krain, A. L., & Castellanos, F. X. (2006). Brain development and ADHD. *Clinical psychology review, 26*(4), 433-444.

Kuhn, C. (2015). Emergence of sex differences in the development of substance use and abuse during adolescence. *Pharmacology & therapeutics, 153*, 55-78.

Kulak, J. A., & Griswold, K. S. (2019). Adolescent substance use and misuse: Recognition and management. *American family physician, 99*(11), 689-696.

Kuriyan, A. B., Pelham, W. E., Molina, B. S., Waschbusch, D. A., Gnagy, E. M., Sibley, M. H., . . . Yu, J. (2013). Young adult educational and vocational outcomes of children diagnosed with ADHD. *Journal of abnormal child psychology, 41*(1), 27-41.

Lara, D. R. (2010). Caffeine, mental health, and psychiatric disorders. *Journal of Alzheimer's disease, 20*(s1), S239-S248.

Laucht, M., Hohm, E., Esser, G., Schmidt, M. H., & Becker, K. (2007). Association between ADHD and smoking in adolescence: shared genetic, environmental and psychopathological factors. *Journal of neural transmission, 114*(8), 1097.

Lee, S. S., Humphreys, K. L., Flory, K., Liu, R., & Glass, K. (2011). Prospective association of childhood attention-deficit/hyperactivity disorder (ADHD) and substance use and abuse/dependence: a meta-analytic review. *Clinical psychology review, 31*(3), 328-341.

Leon, M. R. (2000). Effects of caffeine on cognitive, psychomotor, and affective performance of children with attention-deficit/hyperactivity disorder. *Journal of attention disorders, 4*(1), 27-47.

Leyton, M., & Vezina, P. (2013). Striatal ups and downs: their roles in vulnerability to addictions in humans. *Neuroscience & Biobehavioral Reviews, 37*(9), 1999-2014.

Leyton, M., & Vezina, P. (2014). Dopamine ups and downs in vulnerability to addictions: a neurodevelopmental model. *Trends in pharmacological sciences, 35*(6), 268-276.

Lisdahl, K. M., Gilbart, E. R., Wright, N. E., & Shollenbarger, S. (2013). Dare to delay? The impacts of adolescent alcohol and marijuana use onset on cognition, brain structure, and function. *Frontiers in psychiatry, 4*, 53.

Lisdahl, K. M., Sher, K. J., Conway, K. P., Gonzalez, R., Ewing, S. W. F., Nixon, S. J., . . . Heitzeg, M. (2018). Adolescent brain cognitive development (ABCD) study: overview of substance use assessment methods. *Developmental cognitive neuroscience, 32*, 80-96.

Lisdahl, K. M., Tamm, L., Epstein, J. N., Jernigan, T., Molina, B. S., Hinshaw, S. P., . . . Bjork, J. M. (2016). The impact of ADHD persistence, recent cannabis use, and age of regular cannabis use onset on subcortical volume and cortical thickness in young adults. *Drug and alcohol dependence, 161*, 135-146.

Lisdahl, K. M., Tapert, S., Sher, K. J., Gonzalez, R., Nixon, S. J., Ewing, S. W. F., . . . Heitzeg, M. (in press). Substance Use Patterns in 9-10 Year Olds: Baseline Findings from the Adolescent Brain Cognitive Development (ABCD) Study. *Drug and alcohol dependence*.

Luciana, M., Bjork, J., Nagel, B., Barch, D., Gonzalez, R., Nixon, S., & Banich, M. (2018). Adolescent neurocognitive development and impacts of substance use: Overview of the adolescent brain cognitive development (ABCD) baseline neurocognition battery. *Developmental cognitive neuroscience*, *32*, 67-79.

Mahoney, C. R., Giles, G. E., Williams, C. S., Brunye, T. T., Taylor, H. A., Kanarek, R. B., . . . Lieberman, H. R. (2019). Relationships between use of dietary supplements, caffeine and sensation seeking among college students. *Journal of American College Health, 67*(7), 688-697.

Malone, P. S., Van Eck, K., Flory, K., & Lamis, D. A. (2010). A mixture-model approach to linking ADHD to adolescent onset of illicit drug use. *Developmental psychology*, *46*(6), 1543.

Marmorstein, N. R. (2016). Energy drink and coffee consumption and psychopathology symptoms among early adolescents: cross-sectional and longitudinal associations. *Journal of caffeine research, 6*(2), 64-72.

Miller, K. E. (2008). Wired: energy drinks, jock identity, masculine norms, and risk taking. *Journal of American College Health*, *56*(5), 481-490.

Miller, K. E., Dermen, K. H., & Lucke, J. F. (2018). Caffeinated energy drink use by US adolescents aged 13–17: A national profile. *Psychology of Addictive Behaviors*, *32*(6), 647.

Moffitt, T. E., Houts, R., Asherson, P., Belsky, D. W., Corcoran, D. L., Hammerle, M., . . . Polanczyk, G. V. (2015). Is adult ADHD a childhood-onset neurodevelopmental disorder? Evidence from a four-decade longitudinal cohort study. *American Journal of Psychiatry*, *172*(10), 967-977.

Mohamed, S. M., Börger, N. A., Geuze, R. H., & van der Meere, J. J. (2016). Post-error adjustments and ADHD symptoms in adults: the effect of laterality and state regulation. *Brain and cognition, 108*, 11-19.

Molina, B. S., Howard, A. L., Swanson, J. M., Stehli, A., Mitchell, J. T., Kennedy, T. M., . . . Vitiello, B. (2018). Substance use through adolescence into early adulthood after childhood-diagnosed ADHD: findings from the MTA longitudinal study. *Journal of Child Psychology and Psychiatry, 59*(6), 692-702.

Molina, B. S., & Pelham Jr, W. E. (2003). Childhood predictors of adolescent substance use in a longitudinal study of children with ADHD. *Journal of abnormal psychology, 112*(3), 497.

Moore, M. J., & Chudley, E. (2005). Sport and physical activity participation and substance use among adolescents. *Journal of Adolescent Health, 36*(6), 486-493.

Noble, E. (1996). Alcoholism and the dopaminergic system: a review. *Addiction biology, 1*(4), 333-348.

Orr, C., Spechler, P., Cao, Z., Albaugh, M., Chaarani, B., Mackey, S., . . . Bokde, A. L. (2019). Grey matter volume differences associated with extremely low levels of cannabis use in adolescence. *Journal of Neuroscience, 39*(10), 1817-1827.

Pandolfo, P., Machado, N. J., Köfalvi, A., Takahashi, R. N., & Cunha, R. A. (2013). Caffeine regulates frontocorticostriatal dopamine transporter density and improves attention and cognitive deficits in an animal model of attention deficit hyperactivity disorder. *European Neuropsychopharmacology*, *23*(4), 317-328.

Pascual, M., Boix, J., Felipo, V., & Guerri, C. (2009). Repeated alcohol administration during adolescence causes changes in the mesolimbic dopaminergic and glutamatergic systems and promotes alcohol intake in the adult rat. *Journal of neurochemistry, 108*(4), 920-931.

Polanczyk, G. V., Willcutt, E. G., Salum, G. A., Kieling, C., & Rohde, L. A. (2014). ADHD prevalence estimates across three decades: an updated systematic review and meta-regression analysis. *International journal of epidemiology, 43*(2), 434-442.

Poudel, A., & Gautam, S. (2017). Age of onset of substance use and psychosocial problems among individuals with substance use disorders. *BMC psychiatry, 17*(1), 10.

Roselló, B., Berenguer, C., Baixauli, I., Mira, Á., Martinez-Raga, J., & Miranda, A. (2020). Empirical examination of executive functioning, ADHD associated behaviors, and functional impairments in adults with persistent ADHD, remittent ADHD, and without ADHD. *BMC psychiatry, 20*, 1-12.

Sagvolden, T., Johansen, E. B., Aase, H., & Russell, V. A. (2005). A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *Behavioral and Brain Sciences, 28*(3), 397-418.

Salum, G., Sonuga-Barke, E., Sergeant, J., Vandekerckhove, J., Gadelha, A., Moriyama, T., . . . Rohde, L. (2014). Mechanisms underpinning inattention and hyperactivity: neurocognitive support for ADHD dimensionality. *Psychological medicine, 44*(15), 3189-3201.

SAMHSA. (2019). *Key substance use and mental health indicators in the United States: Results from the 2018 National Survey on Drug Use and Health*. Retrieved from https://www.samhsa.gov/data/:

Schneider, H. E., Lam, J. C., & Mahone, E. M. (2016). Sleep disturbance and neuropsychological function in young children with ADHD. *Child Neuropsychology, 22*(4), 493-506.

Shram, M. J., Funk, D., Li, Z., & Lê, A. (2007). Acute nicotine enhances c-fos mRNA expression differentially in reward-related substrates of adolescent and adult rat brain. *Neuroscience letters, 418*(3), 286-291.

Sibley, M. H., Pelham Jr, W. E., Molina, B. S., Coxe, S., Kipp, H., Gnagy, E. M., . . . Lahey, B. B. (2014). The role of early childhood ADHD and subsequent CD in the initiation and escalation of adolescent cigarette, alcohol, and marijuana use. *Journal of abnormal psychology, 123*(2), 362.

Sjöwall, D., Roth, L., Lindqvist, S., & Thorell, L. B. (2013). Multiple deficits in ADHD: executive dysfunction, delay aversion, reaction time variability, and emotional deficits. *Journal of Child Psychology and Psychiatry, 54*(6), 619-627.

Smith, A. (2002). Effects of caffeine on human behavior. *Food and chemical toxicology, 40*(9), 1243-1255.

Sobell, L. C., & Sobell, M. B. (1992). Timeline follow-back *Measuring alcohol consumption* (pp. 41-72): Springer.

Squeglia, L. M., & Gray, K. M. (2016). Alcohol and drug use and the developing brain. *Current psychiatry reports, 18*(5), 46.

Squeglia, L. M., Jacobus, J., & Tapert, S. F. (2009). The influence of substance use on adolescent brain development. *Clinical EEG and neuroscience, 40*(1), 31-38.

Swanson, J. M., Kinsbourne, M., Nigg, J., Lanphear, B., Stefanatos, G. A., Volkow, N., . . . Wadhwa, P. D. (2007). Etiologic subtypes of attention-deficit/hyperactivity disorder: brain imaging, molecular genetic and environmental factors and the dopamine hypothesis. *Neuropsychology review, 17*(1), 39-59.

Tapert, S. F., Granholm, E., Leedy, N. G., & Brown, S. A. (2002). Substance use and withdrawal: neuropsychological functioning over 8 years in youth. *Journal of the International Neuropsychological Society: JINS, 8*(7), 873.

Thiruchselvam, T., Malik, S., & Le Foll, B. (2017). A review of positron emission tomography studies exploring the dopaminergic system in substance use with a focus on tobacco as a co-variate. *The American journal of drug and alcohol abuse, 43*(2), 197-214.

Thoma, R. J., Monnig, M. A., Lysne, P. A., Ruhl, D. A., Pommy, J. A., Bogenschutz, M., . . . Yeo, R. A. (2011). Adolescent substance abuse: the effects of alcohol and marijuana on neuropsychological performance. *Alcoholism: Clinical and Experimental Research, 35*(1), 39-46.

Thorpe, H. H., Hamidullah, S., Jenkins, B. W., & Khokhar, J. Y. (2020). Adolescent neurodevelopment and substance use: receptor expression and behavioral consequences. *Pharmacology & therapeutics, 206*, 107431.

Thurstone, C., Hull, M., Timmerman, J., & Emrick, C. (2017). Development of a motivational interviewing/acceptance and commitment therapy model for adolescent substance use treatment. *Journal of contextual behavioral science, 6*(4), 375-379.

Uban, K. A., Horton, M. K., Jacobus, J., Heyser, C., Thompson, W. K., Tapert, S. F., . . . Sowell, E. R. (2018). Biospecimens and the ABCD study: Rationale, methods of collection, measurement and early data. *Developmental cognitive neuroscience, 32*, 97-106.

van Emmerik-van Oortmerssen, K., van de Glind, G., van den Brink, W., Smit, F., Crunelle, C. L., Swets, M., & Schoevers, R. A. (2012). Prevalence of attention-deficit hyperactivity disorder in substance use disorder patients: a meta-analysis and meta-regression analysis. *Drug and alcohol dependence, 122*(1-2), 11-19.

Verret, C., Guay, M.-C., Berthiaume, C., Gardiner, P., & Béliveau, L. (2012). A physical activity program improves behavior and cognitive functions in children with ADHD: an exploratory study. *Journal of attention disorders, 16*(1), 71-80.

Voigt, R. G., Katusic, S. K., Colligan, R. C., Killian, J. M., Weaver, A. L., & Barbaresi, W. J. (2017). Academic achievement in adults with a history of childhood attention-deficit/hyperactivity disorder: a population-based prospective study. *Journal of developmental and behavioral pediatrics: JDBP, 38*(1), 1.

Volkow, N. D., Koob, G. F., Croyle, R. T., Bianchi, D. W., Gordon, J. A., Koroshetz, W. J., . . . Conway, K. (2018). The conception of the ABCD study: From substance use to a broad NIH collaboration. *Developmental cognitive neuroscience, 32*, 4-7.

Volkow, N. D., & Morales, M. (2015). The brain on drugs: from reward to addiction. *Cell, 162*(4), 712-725.

Volkow, N. D., Wang, G.-J., Fowler, J. S., & Ding, Y.-S. (2005). Imaging the effects of methylphenidate on brain dopamine: new model on its therapeutic actions for attention-deficit/hyperactivity disorder. *Biological psychiatry, 57*(11), 1410-1415.

Volkow, N. D., Wang, G.-J., Fowler, J. S., Logan, J., Gerasimov, M., Maynard, L., . . . Franceschi, D. (2001). Therapeutic doses of oral methylphenidate significantly increase extracellular dopamine in the human brain. *Journal of Neuroscience, 21*(2), RC121-RC121.

Walker, L. R., Abraham, A. A., & Tercyak, K. P. (2010). Adolescent caffeine use, ADHD, and cigarette smoking. *Children's Health Care, 39*(1), 73-90.

Werch, C. C., Moore, M. J., DiClemente, C. C., Bledsoe, R., & Jobli, E. (2005). A multihealth behavior intervention integrating physical activity and substance use prevention for adolescents. *Prevention Science, 6*(3), 213.

Wilens, T. E., Biederman, J., Mick, E., Faraone, S. V., & Spencer, T. (1997). Attention deficit hyperactivity disorder (ADHD) is associated with early onset substance use disorders. *The Journal of nervous and mental disease, 185*(8), 475-482.

Wilens, T. E., Martelon, M., Joshi, G., Bateman, C., Fried, R., Petty, C., & Biederman, J. (2011). Does ADHD predict substance-use disorders? A 10-year follow-up study of young adults with ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry, 50*(6), 543-553.

Wilens, T. E., & Spencer, T. J. (2010). Understanding attention-deficit/hyperactivity disorder from childhood to adulthood. *Postgraduate medicine, 122*(5), 97-109.

Wong, M. M., Brower, K. J., & Zucker, R. A. (2009). Childhood sleep problems, early onset of substance use and behavioral problems in adolescence. *Sleep medicine, 10*(7), 787-796.

Wymbs, B. T., McCarty, C. A., Mason, W. A., King, K. M., Baer, J. S., Vander Stoep, A., & McCauley, E. (2014). Early adolescent substance use as a risk factor for developing conduct disorder and depression symptoms. *Journal of studies on alcohol and drugs, 75*(2), 279-289.

Zhang, X., Feng, Z.-J., & Chergui, K. (2015). Induction of cannabinoid-and N-methyl-D-aspartate receptor-mediated long-term depression in the nucleus accumbens and dorsolateral striatum is region and age dependent. *International Journal of Neuropsychopharmacology, 18*(4).

Zucker, R. A., Gonzalez, R., Ewing, S. W. F., Paulus, M. P., Arroyo, J., Fuligni, A., . . . Wills, T. (2018). Assessment of culture and environment in the Adolescent Brain and Cognitive Development Study: Rationale, description of measures, and early data. *Developmental cognitive neuroscience, 32*, 107-120.

Zulauf, C. A., Sprich, S. E., Safren, S. A., & Wilens, T. E. (2014). The complicated relationship between attention deficit/hyperactivity disorder and substance use disorders. *Current psychiatry reports, 16*(3), 436.

Curriculum Vitae

EDUCATION

2016-2022	**Doctor of Philosophy in Psychology** **Clinical Psychology Track** **Emphasis in Neuropsychology** **University of Wisconsin-Milwaukee** Dissertation: *The longitudinal effects of early substance use on ADHD symptom development in the ABCD study* Defense Date: June 2021 Advisor: Krista Lisdahl, Ph.D.
2016-2019	**Masters of Science in Psychology** **Major: Psychology** Degree: MS, May 2019 Thesis: *BOLD responses to inhibition in cannabis using adolescent and emerging adults* Advisor: Krista Lisdahl, Ph.D.
2010-2013	**Bachelors of Science with Honors** **University of Iowa** Iowa City, IA Major: Psychology Minor: Human Relations

AWARDS AND FUNDING

Fall 2020 – Spring 2021	UWM Distinguished Dissertator Fellowship
Spring 2020	Association of Graduate Students in Neuropsychology Travel Award
Fall 2019	UWM Graduate Student Excellence Fellowship
Spring 2019	CPDD Travel Award for Early Career Investigators
Fall 2018	Academy of Psychological Clinical Science Flexibility Fund Award
Fall 2018	UWM Distinguished Graduate Student Fellowship Nominee
Summer 2018	Health Psychology Graduate Students Travel Award
Summer 2018	University of Wisconsin-Milwaukee Department of Psychology Summer Graduate Research Fellowship
Summer 2017	Health Psychology Graduate Students Travel Award
Fall 2016 – Spring 2018	University of Wisconsin-Milwaukee Chancellor's Award
Spring 2013	Graduated with Honors
Fall 2010 – Spring 2013	University of Iowa Dean's List
Fall 2010 – Spring 2013	University of Iowa Honors Program

PUBLICATIONS

Sullivan, R. M., Maple, K. E., **Wallace, A. L.,** Thomas, A. M., & Lisdahl, K. M. (Accepted). Examining inhibitory affective processing within the rostral anterior cingulate cortex among abstinent cannabis-using adolescents and young adults. *Frontiers in Psychiatry, Addictive Disorders*. doi:10.3389/fpsyt.2022.851118

Wade, N.E., McCabe, C.J., **Wallace, A.L.**, Gonzalez, M. R., Hoh, E., Infante, M.A., Haist, F., & Hernandez, M., (Accepted). Clouding up cognition: Secondhand cannabis and tobacco exposure related to cognitive functioning in youth. B*iological Psychiatry: Global Open Science*.

Harris, J.C., **Wallace, A.L.**, Thomas, A.M., Wirtz, H.G., Kaiver, C.M., & Lisdahl, K.M. (2022). Disrupted resting state attentional network connectivity in adolescent and young adult cannabis users following two-weeks of monitored abstinence. *Brain Sciences. 12*(2):287.

Sullivan, R.M., **Wallace, A.L**., Arechiga, J.R., Dommer, L., & Lisdahl, K.M. (2021). Examination of withdrawal, affective, and sleep inventories after sustained abstinence from cannabis in young adults. *Cannabis and Cannabinoids Research.*

Stinson, E.A., Sullivan, R.M., Peteet, B.J., Tapert, S.F., Baker, F.C., Breslin, F.J., Dick, A.S., Gonzalez, M.R., Guillaume, M., Marshall, A.T., McCabe, C.J., Pelham III, W.E., Rinsveld, A.V., Sheth, C.S., Sowell, E.R., Wade, N.E., **Wallace, A.L.**, & Lisdahl, K.M. (2021). Longitudinal Impact of Childhood Adversity on Early Adolescent Mental Health During the COVID-19 Pandemic in the ABCD Study® Cohort: Does Race or Ethnicity Moderate Findings? *Biological Psychiatry: Global Open Science, , 1*(4), 324–335.

Lisdahl, K.M., Tapert, S., Sher, K.J., Gonzalez, R., Nixon, S.J., Ewing, S.W.F., Conway, K.P., **Wallace, A.**, Sullivan, R., Hatcher, K., Kaiver, C. Thompson, W., Reuter, C., Gruber, S., Bartsch, H., Jacobus, J., Bagot, K., Foxe, J., Haist, F., Kaufman, A., LeBlanc, K., Lessov-Schlaggar, C., Lopez, M., Madden, P., Frieze, Sc., {ABCD Consortium Investigators}.... Heitzeg, M., (2021). Substance use patterns in 9-10 year olds: Baseline findings from the Adolescent Brain Cognitive Development (ABCD) Study. *Drug and Alcohol Dependence, 227,* 108946.

Austin, J.E., Lang, A.C., Nacker, A.M., **Wallace, A.L**., Schwebel, D.C., Brown, B.B., & Davies. W.H. (2021). Adolescent experiences with self-asphyxial behaviors and problematic drinking in emerging adulthood. *Global Pediatric Health, 8,* 2333794X211037985.

Sullivan, R.M., **Wallace, A.L.**, Wade, N.E., & Lisdahl, K.M. (2021). Cannabis use & brain volume in adolescent and young adult cannabis users: Does sex or aerobic fitness moderate effects? *Journal of the International Neuropsychological Society, 27*(6), 607-620.

Ritchay, M.M., Huggins, A.A., **Wallace, A.L.**, & Lisdahl, K.M. (2021). Resting State Functional Connectivity in the Default Mode Network: Relationships Between Cannabis Use, Gender, and Cognition in Adolescents and Young Adults. *NeuroImage: Clinical,30,* 102664.

Wade, N.E., Palmer, C.E., Gonzalez, M.R., **Wallace, A.L.**, Infante, M.A., Tapert, S.F., Jacobus, J., Bagot, K.S., (2021). Risk factors associated with curiosity about alcohol use in the ABCD cohort. *Alcohol. 92*, 11-19.

Wade, N.E., **Wallace, A.L.**, Sullivan, R.M., Lisdahl, K. M. (2020). Association Between Brain Morphometry and Aerobic Fitness Level and Sex in Healthy Emerging Adults. *PLOS One. 15*(12): e0242738.

Wade, N.E., Kaiver, C.M., **Wallace, A.L.**, Hatcher, K.F., Swartz, A.M., & Lisdahl, K.M. (2020). Objective Aerobic Fitness Level and Neuropsychological Functioning in Healthy Adolescents and Emerging Adults: Unique Sex Effects. *Psychology of Sport & Exercise, 51,* 101794.

Wallace, A.L., Barr, A.T., Maple, K.E., & Lisdahl, K.M. (2020). BOLD responses to inhibition in cannabis using adolescents and emerging adults after two weeks of monitored cannabis abstinence. *Psychopharmacology, 237*(11), 3259-3268.

Wallace, A.L., Wade, N.E., Lisdahl, K.M. (2020). Impact of two-weeks of monitored abstinence on cognition in adolescent and young adult cannabis users. *Journal of the International Neuropsychological Society: JINS, 26*(8), 776-784.

Sullivan, R.M., **Wallace, A.L.**, Wade, N.E., Swartz, A.M., & Lisdahl, K.M. (2020). Assessing the role of cannabis use and cortical surface structure in adolescents and young adults: Exploring gender and aerobic fitness as potential moderators. *Brain Sciences, 10*(2), 117.

Vaidya, J.G., Elmore, A.L., **Wallace, A.L.**, Langbehn, D.R., Kramer, J.R., Kuperman, S., & O'Leary, D.S. (2019). Association between age and familial risk for alcoholism on functional connectivity in adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry, 58*(7), 692-701.

Wade, N.E., **Wallace, A. L**., Swartz, A.M., & Lisdahl, K.M. (2019). Aerobic fitness level moderates the association between cannabis use and executive functioning and psychomotor speed in adolescents and young adults. *Journal of the International Neuropsychological Society, 25(2),* 134-145.

Wallace, A.L., Wade, N.E., Hatcher, K.F., & Lisdahl, K.M. (2019). Effects of cannabis use and subclinical ADHD symptomology on attention based tasks in adolescents and young adults. *Archives of Clinical Neuropsychology, 34*(5), 700–705.

Wallace, A., Ullsperger, J.M., & Nikolas, M.A. (2016). Do personality traits explain the association between childhood attention-deficit hyperactivity disorder symptoms and substance use and problems in young adults? *Personality and Individual Differences, 92*, 22-28.

(Under Review)

Wallace, A.L., Lehman, S.M., Sullivan, R.M., Heitzeg, M.M., Tapert, S.F., & Lisdahl, K.M. (Under Review). Effects of caffeine use and ADHD status on cognitive function in youth enrolled in the ABCD study.

Wallace, A.L., Kaiver, C.M., Sullivan, R.M., Swartz, A.M., Cho, C.C., & Lisdahl, K.M. (Under Review). Stand-biased desks impact on cognition in elementary students.

Wallace A.L., Hanson, B.F., Sullivan, R.M., Wade, N.E., Stinson, E.A. & Lisdahl, K.M. (In Preparation). The Effects of Sports Activity on Cognition in Nine- and Ten-Year-Old Children.

Kaiver, C.M., **Wallace, A.L.**, Kangiser, M.M., Mulligan, D., Messman, G.M., & Lisdahl, K.M. (Under Review). Binge drinking impacts prefrontal and parietal gyrification in young adults.

Jennette, K.J., **Wallace, A.L.,** Lisdahl, K.M., (Under Review). The Association of Aerobic Fitness with Resting State Functional Connectivity and Verbal Learning and Memory in Healthy Young Adults.

(In Preparation)

Wallace, A.L., Hatcher, K.F., Wade, N.E., Lehman, S.M., & Lisdahl, K.M. (In Preparation). Gender moderates the impact of aerobic fitness on mood and executive dysfunction.

Wallace, A.L., & Lisdahl, K.M., (In Preparation). The longitudinal effects of early substance use on ADHD symptom development in the ABCD study.

Wallace, A.L., Stinson, E.A., Leclaire, K.N., Sullivan, R.M., Harris, J.C., & Lisdahl, K.M. (In Preparation). White matter integrity in abstinent cannabis-using adolescents and young adults: Associations with age of regular use onset.

Sullivan, R.M., **Wallace, A.L**., Weis, C.N., Larson, C.L., & Lisdahl, K.M. (In Preparation). Appetitive functional responding and task-dependent functional connectivity among cannabis-using adolescent and young adults.

Sullivan, R. M., Maple, K. E., **Wallace, A. L.**, Barr, A. T., & Lisdahl, K. M. (In Preparation). Examining inhibitory affective processing within the rostral anterior cingulate cortex among cannabis-using adolescents and young adults.

Sullivan, R. M., Lisdahl, K. M., **Wallace, A. L.**, Hahn, S., Garavan, H., Allgaier, N., {ABCD Substance Use Workgroup Investigators}, & Heitzeg, M. M. (In Preparation). Alcohol sipping in youth: Multifactor associations revealed using machine-learning.

PRESENTATIONS

(Oral Presentations)

Wallace, A.L., Sullivan, R.M., Stinson, E.A., Wade, N.E. & Lisdahl, K.M. (2021, February). *Sports Engagement Predicts Cognition in Nine and Ten Year Old Children in the ABCD Study.* Presented at the virtual 49th annual scientific meeting of the International Neuropsychological Society, San Diego, California.

Wallace, A.L., Lehman, S.M., Sullivan, R.M., Lisdahl, K.M. (2020, February). *The Independent and Interactive Relationships Between ADHD Diagnosis and Caffeine Use on Cognitive Functioning in Youth.* Presented at the 48th annual scientific meeting of the International Neuropsychological Society, Denver, Colorado.

Wallace, A.L., Maple, K.E., Lisdahl, K.M. (2019, June) *Does Gender Moderate BOLD Responses to Inhibition in Cannabis Using Adolescents and Emerging Adults.* Presented at The College on Problems of Drug Dependence 81st Annual Conference, San Antonio, Texas.

Lisdahl, K.M, **Wallace, A.L.**, Sullivan, R.M., Reuter, C., Thompson, W.K., Heitzeg, M.M. (2019, June) *Substance Use Patterns & Neurocognitive Predictors of Sipping and Caffeine Use: Year Two Findings from the ABCD Study.* Presented at The College on Problems of Drug Dependence 81st Annual Conference, San Antonio, Texas.

Wallace, A.L., Jennette, K.J., Lisdahl, K.M. (2018, June). *Impact of Three-Weeks of Sustained Abstinence on Cognition in Young Adult Cannabis Users*. Presented at the College on Problems of Drug Dependence 80th Annual Conference, San Diego, California.

Wade, N.E., **Wallace, A.L**., Lisdahl, K.M. (2018, February) *Aerobic Fitness and Marijuana Use Interact to Predict Neuropsychological Functioning in Emerging Adults.* Presented at the International Neuropsychological Society 46th Annual Conference, Washington, D.C.

(Poster Presentations)

Wallace, A. L., Sullivan, R. M., Stinson, E. A., Kaiver, C. M. & Lisdahl, K. M. (Accepted). *The Effects of Substance Use on ADHD Symptomology in Late Childhood.* Poster submitted to the 50th annual scientific meeting of the International Neuropsychological Society (INS).

Leclaire, K.N., Harris, J.C., **Wallace, A.L.,** Esson, M., & Lisdahl, K.M. (Accepted). *The Association Between Aerobic Fitness Level, Sex, and Netork Connectivity in the Defaul Mode Network in Healthy Emerging Adult.* Poster submitted to the 50th annual scientific meeting of the International Neuropsychological Society (INS).

Sullivan, R. M., Kaiver, C. M., Stinson, E. A., **Wallace, A. L.,** Sauber, G., Hillard, C. J., & Lisdahl, K. M. (Accepted). *Associations of Pre-Adolescent Circulating Endocannabinoid Concentrations with Cognitive and Behavioral Executive Functioning Measures: Preliminary Data from the ABCD Study.* Poster submitted to the 50th annual scientific meeting of the International Neuropsychological Society (INS).

Kaiver, C. M., Sullivan, R. M., Stinson, E. A., **Wallace, A. L.,** Hillard, C. J., & Lisdahl, K. M. (Accepted). *Associations Between Circulating Endocannabinoid (eCB) Levels and Mathematics Functioning in Preadolescent Youth: Pilot Data from the ABCD eCB Substudy.* Poster submitted to the 50th annual scientific meeting of the International Neuropsychological Society (INS).

Stinson, E. A., Kaiver, C. M., Sullivan, R. M., Jarvis, J. E., **Wallace, A. L.,** Sauber, G., Hillard, C. J., & Lisdahl, K. M. (Submitted). *Investigation of the Relationship Between Circulating Endocannabinoid Levels and Episodic Memory in Preadolescents from the ABCD® Study Cohort.* Poster submitted to the 50th annual scientific meeting of the International Neuropsychological Society (INS).

Wallace, A.L., Sullivan, R.M., Lisdahl, K.M., (2021, June). *White Matter Integrity in Cannabis Using Adolescents and Young Adults and Relations to Age of Regular Use Onset.* Poster presentation at The College on Problems of Drug Dependence 83rd Annual Conference, Virtual Conference.

Sullivan, R.M., **Wallace, A.L**., Lisdahl, K.M. (2021, June). *Functional Response Inhibition to Appetitive Face Stimuli among Adolescent and Young Adult Alcohol and Cannabis Use.* Poster presentation at The College on Problems of Drug Dependence 83rd Annual Conference, Virtual Conference.

Harris, J.C., **Wallace, A.L**., Barr, A.T., Lisdahl, K.M. (2021, June). *Behavioral Correlates of Attentional Network Differences in Cannabis Using Adolescents and Young Adults.* Poster presentation at The College on Problems of Drug Dependence 83rd Annual Conference, Virtual Conference.

Wallace, A.L., Sullivan, R.M., Wade, N.E., Lehman, S.M., & Lisdahl, K.M. (2020, June). *Aerobic Fitness as a Potential Moderator on the Relationship between Binge Drinking and Cognition in Adolescent and Young Adults*. Poster presentation at the 43rd annual Research Society on Alcoholism (RSA) Conference, New Orleans, LA. (Conference canceled)

Wade, N.E., Palmer, C., **Wallace, A.L.**, Infante, A.A.M, Gonzalez, M., Tapert, S.F., Jacobus, J., & Bagot, K. (2020, June). *Risk Factors Associated with Curiosity About Alcohol Use in the ABCD Cohort.* Poster presentation at the 43rd annual Research Society on Alcoholism (RSA) Conference, New Orleans, LA. (Conference canceled)

Sullivan, R.M., Jarvis, J.E., **Wallace, A.L.**, Wade, N.E., & Lisdahl, K.M. (2020, June). *Alcohol-Related Familial and Environmental Factors and its Association with Early-Sipping Behavior in Youth: Examination of the ABCD Cohort*. Poster presentation at the 43rd annual Research Society on Alcoholism (RSA) Conference, New Orleans, LA. (Conference canceled)

Sullivan, R.M., **Wallace, A.L.**, Wade, N.E., & Lisdahl, K.M. (2020, February). *Examination of Gender and Aerobic Fitness on the Impact of Cannabis Use on Brain Structure in Young Adults: Correlated with Neuropsychological Performance*. Poster presentation at the 48th annual scientific meeting of the International Neuropsychological Society, Denver, Colorado.

Kaiver, C.M., **Wallace, A.L.**, Lehman, S.M., Wirtz, H.G., Lisdahl, K.M. (2020, February) *Effects of Sedentary Behavior and Adipose Tissue on Cognition in Adolescents and Young Adults*. Poster presentation at the 48th Annual International Neuropsychology Society the 48th annual scientific meeting of the International Neuropsychological Society, Denver, Colorado.

Sullivan, R.M., **Wallace, A.L.**, Lisdahl, K.M. (2019, June) *Gender Differences in the Impact of Aerobic Fitness and Cannabis Use on Brain Structure in Adolescents and Young Adults.* Poster presentation at The College on Problems of Drug Dependence 81st Annual Conference, San Antonio, Texas

Wirtz, H., **Wallace, A.L**., Kaiver, C.M., Lisdahl, K.M. (2019, April) *The Relationship between Adipose Tissue and Executive Functioning in Adolescents and Young Adults.* Poster presentation at 11th Annual UWM Undergraduate Research Symposium, Milwaukee, Wisconsin.

Arechiga, J.R., **Wallace, A.L**., Lisdahl, K.M. (2019, April) *Effects of 3-weeks of Abstinence from Cannabis on Depression and Anxiety.* Poster presentation at 11th Annual UWM Undergraduate Research Symposium, Milwaukee, Wisconsin.

Esson, M., **Wallace, A.L.**, Kaiver, C.M., Lisdahl, K.M. (2019, April) *Effects of Body Fat on Sleep.* Poster presentation at 11th Annual UWM Undergraduate Research Symposium, Milwaukee, Wisconsin.

Kaiver, C.M., **Wallace, A.L.**, Kangiser, M.M., Mulligan, D., Messman, G.M., Lisdahl, K.M. (2019, February) *Binge Drinking Impacts Prefrontal and Parietal Gyrification in Adolescents and Young Adults*. Poster presentation at The International Neuropsychology Society 47th Annual Conference. New York City, NY

Maple, K.E., Thomas, A.M., **Wallace, A.L**., Lisdahl, K.M. (2019, February) *Blunted Rostral Anterior Cingulate Activation during an Emotional Inhibitory Processing Task in Abstinent Cannabis Users*. Poster presentation at The International Neuropsychology Society 47th Annual Conference. New York City, NY

Lehman, S.M, Jennette, K.J., **Wallace, A.L.**, & Lisdahl, K.M. (2018, April) Effects of Early Onset Marijuana Use on Executive Functioning Compared to Late Onset Marijuana Use. Poster presented at the National Conference on Undergraduate Research. University of Central Oklahoma.

Wallace, A.L., Wright, N.E., Gilbart, E.R., and Lisdahl, K.M. (2017, June). *ADHD symptoms and Marijuana Use Predicts Inattention Performance.* Poster presented at the College on Problems of Drug Dependence 79th Annual Conference, Montreal, Canada.

O'Leary, D.S., Vaidya, J.G., **Wallace, A.L**., Nguyen, R.H., Fuhrmeister, L.A. (2015, March). *The Effects of Concurrent Binge Drinking and Marijuana Use on Adolescent Brain Development*. Poster presented at the National Institutes of Health Collaborative Research on Addiction grantee meeting, Rockville, MD.

RESEARCH EXPERIENCE

August 2021-Present **Intern Research Assistant, St. Jude Children's Research Hospital**
Supervisor: Lisa Jacola, Ph.D. ABPP

1) Conduct research at St. Jude Children's Research Hospital on the Early Childhood Clinic database, a database consisting of children from birth to three who were seen for neuropsychological services due to underlying cancer diagnosis, as part of the Jacola lab.

Duties: Conduct data cleaning of existing database. Formulate research question and aims from the data provided in the database, conduct literature review, run analyses, and write manuscript under the supervision of Lisa Jacola and fellow Early Childhood Clinic faculty.

January 2017-July 2021 **Graduate Research Assistant, University of Wisconsin-Milwaukee, Adolescent Brain Cognitive Development Study (https://abcdstudy.org)**
Supervisor: Krista Lisdahl, Ph.D.

1) Serve as Graduate Research Assistant and work directly with youth and parents participating in the ABCD Study, a ten year, NIH-funded, prospective longitudinal study examining multiple factors that affect adolescent brain, cognitive, emotional and physical development in a diverse, national sample across 21 research sites.

Duties: Recruit participants and families from the community, attend community engagement events; conduct 7-hour youth (ages 9-16) research study sessions that include neuropsychological battery (measuring working processing speed, working memory, verbal fluency, executive functioning, and attention), psychiatric and mental health (e.g., K-SADS), substance use, culture/environment, and physical health questionnaires and interviews; collect biological markers; assist with HCP multi-band functional (task based, resting scan) and structural MRI scans; conduct physical and mental health interviews (e.g., K-SADS) and questionnaires with parents. Implement contingency and behavioral management techniques with youth. Assessing and triaging for suicide risk with youth and parents, supervise staff and RAs on suicide, homicide and abuse risk assessments. Attend and contribute content for weekly RA meetings. Attend bi-weekly meetings with PI and weekly laboratory meetings. Supervise undergraduate students in completing mentored research projects. Responsible for conducting research sessions under observation during annual ABCD data harmonization site visits. Conduct data analysis, manuscript preparation, and local and national dissemination of findings.

Funding Source: U01 DAO41025 (PI Lisdahl); 2U01-DA041025 (PI Lisdahl, Larson)

January 2017-July 2021 **Graduate Research Assistant, University of Wisconsin-Milwaukee, Imaging Data in Emerging Adults with Addiction (https://abcdstudy.org)**
Supervisor: Krista Lisdahl, Ph.D.

1) Serve as Graduate Research Assistant in managing the Imaging Data in Emerging Adults with Addiction (IDEAA) database, a database created by a multi-site consortium comprised of investigators using common, overlapping neuroimaging and behavioral measures in over 600 cannabis using and healthy control adolescents and emerging adults.

Duties: Help manage and clean data across all sites and provide necessary data as requested by members of the consortium and aid with data analysis.

Funding Source: 3R01 DAO030354-03S1 (PI Lisdahl)

August 2016-May 2020 **Graduate Research Assistant, University of Wisconsin-Milwaukee, Brain Imaging and Neuropsychology Lab**
Supervisor: Krista Lisdahl, Ph.D.

1) Served as a Graduate Research Assistant and assisted in running a three-week study of the effects of marijuana use on adolescent and emerging adult brain development as well as measuring potential influences such as alcohol use, life stress, and exercise.

Duties: Conducted 3 hour research sessions with adolescents and young adults (ages 16-26). Study sessions included neurocognitive batteries (measuring working memory, attention, and processing speed), assessing substance use, administering questionnaires evaluating emotional and mental health, collecting biological markers, assist with functional (task based, resting scan) and structural MRI scans. Continuing to conduct data analyses for presentation and manuscript preparation.

Funding Source: **R01 DA030354**, NIDA; PI: Lisdahl, K.M.

September 2016-May 2017 **Research Assistant, University of Wisconsin-Milwaukee, Kinesiology and Psychology Department**
Supervisor: Ann Swartz, Ph.D.
Neuropsychology Supervisor: Krista Lisdah, Ph.D.

1) Integration of Standing Desks in Elementary Schools to Reduce Sedentary Behavior and Improve Neuropsychological Functioning

Duties: Assisted in a year long study determining if standing desks impact grade school aged children with cognitive outcomes as well as other physical activity levels. Administered subtests

of the NIH toolbox measuring executive functioning, attention, and learning at baseline, 4 months, and 8 months.

Funding source: **SAFCO Products Company**

May 2014-July 2016 **Research Assistant, University of Iowa, Department of Psychiatry**
Supervisor: Jatin Vaidya, Ph.D./ Dan O'Leary, Ph.D.

Duties: Completed intellectual, neuropsychological testing, and behavioral imaging tasks with adolescent and young adult participants. Organizing and running preliminary analyses on behavioral data. Preliminary analyses using AFNI on Diffusion Tensor Imaging scans, resting state scans, etc.

November 2011- July 2016 **Research Assistant, University of Iowa, Iowa ADHD and Development Lab**
Principal Investigator: Molly A. Nikolas, Ph.D.

Duties: Completed intellectual, academic, and neuropsychological testing with child, adolescent, and young adult participants with ADHD. Routinely scored, entered, checked, and cleaned data for various projects in the lab. Further, I utilized data collected in the young adult study as part of my honor's thesis project.

May 2013-August 2014 **Research Coordinator, Iowa City Veteran Affairs Hospital**
Supervisor: Dr. Terry Wahls, M.D.

Duties: My role as research coordinator required me to oversee recruitment of participants for studies on nutritional interventions and quality of life with individuals diagnosed with multiple sclerosis as well as to complete neuropsychological testing procedures with participants. Additionally, I updated and maintained IRB protocols and cleaned and prepared data for analyses.

Software/Coding Proficiencies: Linux, SPSS, AFNI, FreeSurfer, MPlus, Matlab, R

CLINICAL EXPERIENCE

August 2021-Present **Clinical Internship, University of Tennessee Health Science Consortium, St. Jude Neuropsychology Track**
Supervisor: Brian Potter, Psy.D., ABPP, Jennifer Longoria, Ph.D., ABPP

Duties: Complete neuropsychological evaluations of children, adolescent, and young adults who have previously or currently undergoing treatment for pediatric cancer, sickle cell disease, and

other pediatric disease within oncology and hematology. Evaluations consist of conducting interview, scoring and interpreting neuropsychological testing, providing diagnoses and potential etiology/prognosis, and providing feedback, recommendations and referrals with clients. Evaluations often occur with multidisciplinary teams, including weekly rounds for patients with tumors and early childhood clinic. Participated in weekly didactics in clinical neuropsychology, pediatric psychology, and general psychology.

December 2021-Present **Clinical Internship, University of Tennessee Health Science Consortium, St. Jude Acute Neurological Injury**
Supervisor: Darcy Raches, Ph.D.

Duties: Complete neuropsychological evaluations of children and adolescents who are undergoing active oncology treatment in inpatient and outpatient care, including posterior fossa syndrome. Evaluations consist of medical chart review, brief interviews with parents, and both outpatient and inpatient neuropsychological tools to determine extend of functioning to inform ongoing care. Evaluations often occur with multidisciplinary teams that meet biweekly and include rehabilitation services. Also conduct cognitive rehabilitation services with children undergoing active care to help build and continue to evaluate cognitive functioning.

December 2021-Present **Clinical Internship, University of Tennessee Health Science Consortium, Semmes Murphey**
Supervisor: Brandon Baughman, Ph.D., ABPP

Duties: Complete neuropsychological evaluations of adolescents and adults across a wide range of medical referrals, including traumatic brain injuries, epilepsy, genetic disorders, and externalizing developmental disorders. Duties include conducting interviews, administering and interpreting neuropsychological testing, providing diagnoses and potential etiology/prognosis, and providing feedback, recommendations, and referrals with clients.

August 2021-December 2021 **Clinical Internship, University of Tennessee Health Science Consortium, Kindred Place**
Supervisor: Catherine Collins, Ph.D.

Duties: Provide intervention and evaluations for children and adults who have survived domestic and sexual abuse as well as court ordered therapeutic intervention anger management for perpetuators of domestic violence and assault. Also conducted state mandated co-parenting courses for newly divorced parents with children to help center child development and needs.

May 2020-May 2021 **Adult Neuropsychology Practicum Externship, Department of Neurology, Division of Neuropsychology, Froedtert Hospital and Medical College of Wisconsin**
Supervisor: Julie Bobholz, Ph.D., ABPP, Sara Swanson, Ph.D., ABPP, Alissa Butts, Ph.D., ABPP, Laura Umfleet, Psy.D., ABPP, Julie Janecek, Ph.D., ABPP, Sara Pillay, Ph.D.

Duties: Conduct comprehensive neuropsychological evaluations of adults (from young adults to geriatric populations) including conducting interviews, administering and interpreting neuropsychological testing, providing diagnoses and potential etiology/prognosis, and providing feedback, recommendations and referrals with clients. Worked as part of a multidisciplinary team in the context of cancer patients as well as surgical cases (e.g., tumor board, epilepsy board). Training included working with diverse populations and neuropsychological disorders (including developmental concerns, epilepsy, neuro-oncology, memory concerns, genetic referrals, and traumatic brain injury). Participated in weekly externship and post-doctoral case conferences and didactics in clinical neuropsychology, journal club, and fact-findings.

May 2019-June 2020 **Pediatric Neuropsychology Practicum Externship, Department of Neurology, Division of Neuropsychology, Children's Hospital of Wisconsin and Medical College of Wisconsin**
Supervisor: Amy Heffelfinger, Ph.D., MPE, ABPP, Jennifer Koop, Ph.D., ABPP, Michelle Loman, Ph.D., and Joseph Amaral, Ph.D.

Duties: Conduct comprehensive neuropsychological evaluations of children and adolescent including conducting interviews, scoring and interpreting neuropsychological testing, providing diagnoses and potential etiology/prognosis, and providing feedback, recommendations and referrals with clients. Worked as part of a multidisciplinary team in the context of cancer patients as well as surgical cases (e.g., tumor board, epilepsy board). Training included working with diverse populations and neuropsychological disorders (including developmental concerns, Autism Spectrum Disorder (ASD), epilepsy, neuro-oncology, genetic referrals, and traumatic brain injury). Participated in weekly externship and post-doctoral case conferences and didactics in clinical neuropsychology, journal club, and fact-findings. Observed and scored along to empirically-supported ASD assessments (e.g., ADOS), pediatric development assessment (e.g., Mullen), and pediatric play observations.

Summer 2020 **Graduate Student Trainee, Department Psychology Clinic, University of Wisconsin-Milwaukee** *(Supplemental Clinical Experience)*
Supervisor: Stacey Nye, Ph.D.

Duties: Conducted intakes with adult clients to determine diagnostic impressions and fit for the departmental clinic. Intakes were completed virtually through videoconferencing and in accordance with telebehavioral health guidelines.

August 2018-May 2021 **Graduate Student Trainee, Child Neuropsychology Clinic, University of Wisconsin-Milwaukee** *(Supplemental Clinical Experience)*
Supervisor: Bonita Klein-Tasman, Ph.D.

Duties: Conduct psychoeducational evaluations of children and adolescents under the supervision of a licensed psychologist. Administered, scored, and interpreted a variety of cognitive and academic assessments. Completed psychoeducational reports and conducted feedback sessions with clients and their families.

August 2018-May 2021 **Practicum in Therapy, Treatment of Eating Disorders, University of Wisconsin-Milwaukee**
Supervisor: Stacey Nye, Ph.D. and Robyn Ridley, Ph.D.

Duties: Conduct empirically supported interventions to treat eating disorders in adolescents and young adult utilizing a Cognitive-Behavioral Therapy (CBT) theoretical perspective. This included setting treatment goals, evaluating progress on goals, and working with a multidisciplinary team (e.g., nutritionist, primary physician) to help facilitate treatment goals. I have conducted this work in-person and through telebehavioral health appointments via videoconferencing.

August 2018-May 2021 **Practicum in Therapy, General CBT Treatment, University of Wisconsin-Milwaukee**
Supervisor: Robyn Ridley, Ph.D.

Duties: Conduct empirically supported interventions to treat anxiety and depression in young adult clients with eating disorders, anxiety, and depression utilizing a CBT theoretical perspective. This work included setting treatment goals and evaluating client progress toward those goals.

August 2017-May 2018 **Practicum in Assessment, University of Wisconsin-Milwaukee**
Supervisor: Bonita Klein-Tasman, Ph.D. and Kristin Smith, Ph.D.

Duties: Conducted child and adult psychoeducational evaluations under the supervision of higher-level graduate students and licensed psychologists within both a departmental clinic and school setting. Administered, scored, and interpreted a variety of cognitive and academic assessments. Completed psychoeducational reports and conducted feedback sessions with clients and their families. Worked with a multidisciplinary school Individualized Education Plan (IEP) team to help devise IEP's based on child's specific strengths and weaknesses.

August 2016-May 2018 **Graduate Student Trainee, Vertical Teams, University of Wisconsin-Milwaukee**
Supervisor: Stacey Nye, Ph.D. and Robin Ridley, Ph.D.

Duties: Observed therapy conducted by students during their therapy practicum while being supervised by licensed psychologists. Learned through observation and supervision core CBT principles and therapeutic tools. Conducted pyschodiagnostic assessments to help aid case conceptualization and intervention.

August 2016-May 2017 **First Year Clinical Psychology Practicum, University of Wisconsin-Milwaukee**

Duties: Practiced conducting semi-structured interviews with the MINI 6.0 and SCID-5 and administering and scoring a variety of cognitive tests for children and adults. Work on building

foundational therapeutic skills and other core clinical skills such as writing integrated reports based on interview and testing data.

CLINICAL SUPERVISION

June 2018-May 2020 **Second Year Practicum Assessment Supervisor, University of Wisconsin-Milwaukee**

Duties: Supervised and directly live observed second-year doctoral students in their assessment practicum while they conducted intakes, cognitive assessments, and provided feedback on assessment results and recommendations. Had monthly supervision meetings with second-year students to discuss cases and conduct trainings on behavioral management, test assessment, and other clinical responsibilities. Met weekly with Bonita Klein-Tasman, Ph.D. and Kristin Smith, Ph.D. to receive supervision over peer supervision.

Fall 2019 **Student Therapy Peer Supervisor, University of Wisconsin-Milwaukee**

Duties: Supervised third-year doctoral students in their therapy practicum on CBT via peer-to-peer group and individual supervision as well as observation of video. Met for both group and individual peer supervision once a week. Received supervision of peer supervision once a month with Robyn Ridley, Ph.D.

TEACHING EXPERIENCE

Teaching Assistant

Fall 2018-Spring 2020 Psych 821-822 Graduate Assessment Practicum, University of Wisconsin-Milwaukee
Instructor: Bonita Klein-Tasman, Ph.D. and Kristin Smith, Ph.D.

Spring 2017-Spring 2018 Psych 325 Research Methods, University of Wisconsin-Milwaukee
Instructor: Susan Lima, Ph.D. and Marcellus Merritt, Ph.D.

Fall 2016 Psych 205 Personality, University of Wisconsin-Milwaukee
Instructor: Marcellus Merritt, Ph.D.

Guest Lecturer

Spring 2021 Psy 323 Health Psychology. *Alcohol and Other Drugs*.
Instructor: Amy Lang, M.S.

Fall 2020 Psych 912 Developmental Psychopathology (Graduate Level). *Substance Use Disorders.*
Instructor: Christine Larson, Ph.D.

Spring 2020 Psy 323 Health Psychology. *Alcohol and Other Drugs*.
Instructor: Amy Lang, M.S.

Fall 2019 Psych 912 Developmental Psychopathology (Graduate Level). *Substance Use Disorders.*
Instructor: Christine Larson, Ph.D.

Fall 2019	Psych 802 First-Year Practicum (Graduate Level). *Clinical Use of the WIAT-III*. Instructor: Kristin Smith, Ph.D.
Fall 2018	Psych 454 Psychopharmacology and Addiction. *Opioids.* Instructor: Krista Lisdahl, Ph.D.
Fall 2018	Psych 193 First-Year Seminar: Research in Clinical Health Psychology. *ABCD Study* Instructor: Hobart Davies, Ph.D.
Fall 2016	Psych 433 Neuropsychology. *Child Cognitive Development*. Instructor: Natasha Wright, M.S.

PROFESSIONAL AFFILIATIONS

Fall 2019-Current	Society of Clinical Child and Adolescent Psychology
Fall 2019-Current	American Psychological Association
Fall 2018-Current	Academy of Psychological Clinical Science
Spring 2018-Current	Division 40 – Society for Clinical Neuropsychology
Fall 2018- May 2021	Associated Graduate Students in Psychology (Treasurer)
Fall 2016- May 2021	Associated Graduate Students in Neuropsychology
Fall 2016-May 2021	Health Graduate Students in Psychology (Treasurer)

AD-HOC REVIEWER

Spring 2021	*Journal of the International Neuropsychological Society*
Spring 2020	*Cognitive, Affective, and Behavioral Neuroscience*
Fall 2019	*Neuropsychology.*
Spring 2019	*Psychiatric Research: Neuroimaging*.

CLINICAL AND PROFESSIONAL TRAINING

Summer 2021	Cannabis Testing 101: Introductory analysis of cannabis-derived samples (12 hours) Instructor: Jack Henion, Ph.D.
Summer 2020	Longitudinal Data Analysis Using R (16 hours) Instructor: Stephen Vaisey, Ph.D.
Spring 2020	Telepsychology Best Practices 101; APA (8 hours) Instructor: Marlene Maheu, Ph.D.
Fall 2019	Machine Learning Training at University of Vermont (16 hours) Instructors: Hugh Garavan, Ph.D., Sage Hahn
Spring 2019	R Statistics Workshop (16 hours) Instructor: Andrew Miles, Ph.D.
Spring 2019	AFNI Bootcamp (40 hours) Instructors: Robert Cox, Ph.D., Rick Reynolds, Ph.D., Paul Taylor, Ph.D., University of Wisconsin-Milwaukee

Fall 2018	Functional Neuroimaging Workshop (16 hours) Instructor: Andrew Jahn, Ph.D., University of Wisconsin-Milwaukee
Spring 2018	MGH FreeSurfer Workshop (32 hours) Martinos Center for Biomedical Imaging, Boston, MA
Summer 2017	Clinical Assessment and Treatment of Eating Disorders Seminar Instructor: Stacey Nye, Ph.D., FAED, University of Wisconsin - Milwaukee
Fall 2016	Introduction to "R" – Learning by Example Seminar Instructor: David Armstrong, Ph.D., University of Wisconsin - Milwaukee
Fall 2016	Responsible Conduct of Research Course Instructor: Kari Whittenberger-Keith, Ph.D., University of Wisconsin Milwaukee
Spring 2016	Group ICA of fMRI Toolbox (GIFT) Workshop (16 hours) Instructor: Vince Calhoun, Ph.D., University of Iowa
Fall 2013	Sensitivity to Obesity Training
Spring 2013	VA Human Subjects Protection and Good Clinical Practices
Spring 2013	VA Privacy and Information Security Awareness and Rules of Behavior training
Spring 2013	VHA Privacy HIPAA training
Fall 2011	CITI Training (with continued training as appropriate)
Fall 2011 & 2012	Diversity Training with the National Coalition Building Institute

SERVICE

Spring 2022	Psychological Safety Policy Group for St. Jude Psychosocial Department
Summer 2020-Summer 2021	Member of the Clinical Work Group of the Clinical Program Diversity Committee at UWM
Summer 2019- Summer 2021	Formed and led Summer Neuropsychology Didactics for UWM Clinical Psychology Students
Spring 2019	Session chair for THC Treatment at The College on Problems of Drug Dependence 81st Annual Conference
Fall 2018	Organized UWM's 13th Annual Association of Graduate Student's in Psychology Symposium
Fall 2017	Organized UWM's 12th Annual Association of Graduate Student's in Psychology Symposium