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Does Neighborhood Socioeconomic Status Moderate the Association Between ADHD and Overweight/Obesity?

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Abstract

This study aimed to examine whether neighborhood-level socioeconomic status (NSES) moderated the Attention-Deficit/Hyperactivity Disorder (ADHD)-overweight/obesity association. Participants (N=568; mean (SD) age = 20.74 (3.20) years; 70.8% female) were part-time or full-time students attending the City College of New York. They were recruited through an online platform and participated in the study in exchange for course credit. Eligible participants were required to self-report their height, weight, from which body mass index (BMI) was calculated. Participants were categorized as Low BMI (Underweight or Typical BMI) or High BMI (Overweight or Obese BMI). Participants self-reported current ADHD symptoms using the ADHD-EF Index of the Barkley Deficits in Executive Functioning Scale. They were then categorized as Low or High ADHD, with threshold set at scores 1 or more SD above the mean. Multiple indices of deprivation were coded from US Census data for each participant's zip code, from which a factor analysis generated two NSES factors. A chi square analysis indicated that individuals with High ADHD were no more likely to be in the High BMI group than individuals with Low ADHD. To test the interaction of ADHD and NSES on BMI status, a binary logistic regression was conducted. Neither ADHD nor NSES were found to be significant predictors of BMI status, and there was no significant interaction. This study confronts the difficulty of attempting to understand physical and mental health outcomes by measuring socioeconomic status on a community level. We also discuss the importance of socioeconomic status in psychological research.

Keywords: *Attention-deficit/hyperactivity disorder; obesity; neighborhood socioeconomic status; college students.*

Does Neighborhood Socioeconomic Status Moderate the Association between ADHD and Overweight/Obesity?

What is ADHD?

Attention deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by developmentally inappropriate inattention and hyperactivity/impulsivity that interferes with day-to-day functioning, and which may continue throughout the lifespan (American Psychiatric Association, APA, 2013). Inattention is characterized by the individual often having difficulty focusing on a task. The Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5, APA, 2013) lists nine symptoms of inattention which include: making careless mistakes, difficulty sustaining attention during tasks or play activities, forgetfulness, distractibility, losing things, not listening when spoken to directly, disorganization, failure to finish tasks, and avoiding tasks that require mental effort. Hyperactivity is manifested by excessive fidgeting, movement, or extreme restlessness. Impulsivity is seen through the individual engaging in hasty actions (e.g. social intrusiveness) or making decisions without thinking or considering consequences, having difficulty waiting, interrupting others, and blurting out answers (APA 2013). Hyperactivity/Impulsivity is also marked by nine specific symptoms, including: often fidgeting with hands or feet and squirming in seat, often leaves seat in situations when remaining seated is expected, running/climbing, difficulty playing quietly, acting as if ‘on the go’ or driven by a motor, excessive talkativeness, unable to wait one’s turn, difficulty slowing down, blurting out answers, and often interrupting or intruding on others. To make a diagnosis of ADHD these symptoms must be persistent, occur in more than one setting, and multiple symptoms must be present before the age of 12 (APA, 2013).

There are currently three primary subtypes of ADHD, predominantly inattentive presentation, predominantly hyperactive-impulsive presentation, and combined presentation. For children,

predominately inattentive presentation is met if the symptoms present over the previous 6 months are mostly from the inattentive domain, and there are relatively fewer hyperactive/impulsive symptoms. That is, at least 6 of the 9 inattentive symptoms are present, but five or fewer of the 9 hyperactive/impulsive symptoms (for adults, the threshold is five symptoms) (APA, 2013). For children, predominately hyperactive-impulsive presentation is met if for the past 6 months, six or more of the 9 symptoms of impulsivity/hyperactivity are present, but five or fewer symptoms of inattention (again, for adults the symptom threshold is five) (APA, 2013). Last, a combined presentation will be met for children if 6 or more symptoms of inattention and 6 or more hyperactive/impulsive symptoms are present, for the past 6 months; for adults with five or more symptoms must be met in both domains (APA, 2013).

ADHD manifests differently across stages of development. Childhood ADHD may be marked by poor school performance, immature behavior compared to peers, and difficulty controlling emotions (Barry, Lyman, and Klinger, 2002; Fleming et. al., 2017; Murray-Close et. al., 2010). Teens with ADHD will often have the same difficulties as in childhood, but hyperactive symptoms may become more subtle due to improved ability to regulate overt hyperactivity. Adolescent ADHD may be marked by poor academic performance and difficulty with establishing and maintaining peer relationships (Murray-Close et. al. 2010; Scholtens, Rydell, & Yang-Wallentin, 2013). In adulthood, individuals with ADHD symptoms may exhibit difficulties at work and in their personal life (Spencer, Biederman, & Mick, 2007). ADHD across the lifespan will be discussed in more detail below.

It is estimated that in the U.S. 9.4% of children aged 2-17 have been diagnosed with ADHD, with boys being more likely to receive the diagnosis than girls (Danielson et.al., 2018). For American adults aged 18 to 44 years, 4.4% are estimated to have a current ADHD diagnosis, with the prevalence being higher for males than females (Kessler et. al., 2006). These prevalence rates show

that ADHD persists into adulthood. However, adult prevalence rates do not distinguish between continuous childhood ADHD onset and the occurrence of adult ADHD onset (Kessler et. al., 2006).

A birth cohort study conducted by Caye et. al. (2016) suggests that adult ADHD is not simply a continuation of childhood ADHD. The authors concluded this after analyzing cohort survey data for ADHD during childhood (11 years of age; C-ADHD) and young adulthood (18 to 19 years of age; YA-ADHD). An analysis of results showed that C-ADHD was present in 8.9% of the sample. Of these, 15.3% had ADHD symptoms continue into young adulthood. At the 18 to 19 years of age assessment, 492 individuals met YA-ADHD criteria. Of these, only 12.2.% had C-ADHD. Even when excluding individuals with comorbidities in young adulthood this difference persisted. More so, the YA-ADHD group, showed a preponderance of females and a greater presentation of the inattentive subtype. The authors hypothesize that these findings suggest adult onset ADHD may be a separate syndrome.

Agnew-Blais et. al. (2016) produced similar results to Caye and colleagues (2016) but provided alternative explanations for such results. Agnew-Blais (2016) utilized a longitudinal study design to assess ADHD symptoms in a population-based cohort at ages 5, 7, 10, and 12 (childhood) and 18 years (young adulthood). The researchers found that 70% of individuals with adult ADHD did not meet diagnostic criteria in childhood. Additionally, the results showed that those with late onset were less likely to be male, had fewer behavioral problems during childhood, and less cognitive impairment than did individuals with a persistent diagnosis. The authors suggested that symptoms of ADHD in childhood were suppressed by protective factors such as a supportive family environment. Another explanation is that late-onset individuals have a different disorder presenting with similar symptoms to that of ADHD. Last, like Caye et. al. (2016), Agnew-Blais and colleagues proposed that late-onset ADHD is a distinct disorder since the late-onset group displayed important differences from the persistent group, including fewer neuropsychological deficits during childhood, an equal prevalence rate across gender, and lower heritability. In opposition to Caye et. al. (2016) and Agnew-

Blais et. al. (2016), a longitudinal population-based study (Sibley et. al., 2018) did not find support for adult-onset ADHD. Rather, the authors' evidence suggested that late-onset ADHD in the sample was better explained by heavy substance use or another comorbid mental disorder.

Regardless of age of onset, it is possible for ADHD to affect individuals throughout their lives and in multiple domains of functioning. For instance, during childhood, ADHD symptoms may impair parent-child relationships. Daley, Sonuga-Barke, and Thompson (2003) found that mothers of ADHD children displayed an overall less positive relationship, less warmth, greater critical comments and fewer positive comments during parent-child interactions than did mothers of non-ADHD control children. A study of sibling pairs with and without ADHD also showed that siblings with ADHD experienced less maternal warmth when compared to their non-ADHD siblings (Cartwright et. al., 2011). Negative parent-child relationships may then affect a child's social development and peer relations (Deault, 2010). A longitudinal analysis revealed that children with ADHD exhibited aggressive and antisocial behaviors, weaker social skills, and poorer ability to gauge their social and behavioral awareness than did a control group, resulting in greater peer rejection (Murray-Close et. al. 2010). Aggressive and externalizing behaviors may additionally lead to an increased incidence of unintentional injuries such as burns, head injuries, and bone fractures (Rowe, Maughan, & Goodman, 2004; Shilon, Pollak, Aran, Shaked, & Gross-Tsur, 2012). Barry, Lyman, and Klinger (2002) also find that children diagnosed with ADHD tend to be more academically impaired than a non-ADHD comparison group. This was evident by academic underachievement in the areas of basic skills, reading, writing, and mathematics, greater occurrence of comorbid diagnosis of a learning disability, and frequent placement in special education classes.

Impairment during childhood tends to create similar negative outcomes for affected adolescents. In the domain of parent-child relations, the relationship may worsen due to intensified levels of disruptive and defiant behaviors during the adolescent years (Modesto-Lowe, Chaplin, Godsay, & Soovajian, 2014). Weaker social skill and peer rejection observed in childhood persists

through adolescence, with aggression having a direct effect on peer rejection (Murray-Close et. al. 2010). Likewise, academic under-achievement continues into adolescence (Scholtens, Rydell, & Yang-Wallentin, 2013), with ADHD associated with diminished odds of graduating high school (Bussing, Mason, Bell, Porter, & Garvan, 2010). Bussing et al. (2010) also found that individuals with childhood ADHD had a threefold chance of juvenile justice involvement compared to non-ADHD controls. In contrast, one study showed that maternal psychosocial adjustment and parenting skills along with higher academic performance during childhood seem to be associated with positive academic and behavioral outcomes during adolescence for those with childhood ADHD (Latimer, et. al., 2003).

ADHD-related impairment is also observed in adulthood. For example, adults with current ADHD experienced greater substance misuse (Murphy & Barkley, 1996a), reported having significantly more marriages, somatic complaints, interpersonal issues, hostility, job changes, and speeding violations than controls. They were also more likely to have dropped out of college and been fired from a job. Further, it is noted that adults with ADHD have lower socioeconomic status, fewer years of education, and lower rates of professional employment than controls (Spencer, Biederman, & Mick, 2007).

ADHD is a debilitating disorder throughout multiple stages of life. However, proper treatment can be effective in reducing symptoms and negative outcomes. Primarily, ADHD in children is treated with behavioral therapy (including parent behavioral training) and medication. According to the CDC (2019), it is recommended that diagnosed children under the age of six utilize parent behavior therapy training as the first line of treatment. Parent behavioral training involves using behavioral principles, such as rewarding wanted behaviors and withdrawing reinforcement for unwanted behaviors to increase the frequency with which the child engages in the desired behavior. Parents are equipped with necessary skills and strategies to help regulate the child's behavior and to foster a strong relationship with the child. Children above the age of 6 are recommended to seek

therapy that teaches the person to monitor and regulate their own behavior, emotions, and actions. Additionally, children at this age may benefit from a combination of therapy and medication. Though there is no cure for ADHD, stimulant medication is efficacious and has a positive effect on symptoms (but may not normalize behaviors) for about 80% of individuals. They work by increasing the amount of dopamine and norepinephrine in the brain which in turn increases alertness, attention, and energy and effectively decreases symptoms of ADHD (CDC, 2019; NIH, 2019). A review of the literature suggests that although stimulants are helpful in improving ADHD symptoms, non-pharmacological therapy benefits academic and organizational skills in adolescents (Chan, Fogler, & Hammerness, 2016).

Recommendations for treatment of adult ADHD are like those for children above the age of 6. A literature review on treatment for adults with ADHD proposes that long acting stimulants and psychosocial treatment, especially target-specific psychoeducation and cognitive behavioral therapy, are effective treatment approaches for problems faced by adults with ADHD, such as poor time management and difficulties maintaining relationships (Kolar, et. al., 2008). Notably, one double-blind, placebo-controlled, randomized study found that adults with ADHD showed a significant reduction in ADHD symptoms, with a 76% response rate to stimulant medication (Spencer at. al., 2005). These results demonstrate the robust effectiveness of stimulant medication treatment for adult ADHD.

Yet, drug treatment of ADHD in children and adults is not without criticism. More specifically, the potential side effects of long-term stimulant use are unclear (Meijer, Faber, Den Ban, & Tobi, 2009). In children, mild side effects such as sleep problems, loss of appetite, and reduction in growth trajectories have been reported. While more severe, but rare, an increased risk of suicidal ideation in children and adolescents has been associated with the non-stimulant drug, Strattera (Meijer, Faber, Den Ban, & Tobi, 2009). The greatest critique of pharmacotherapy for the treatment of ADHD in children, adolescents, and adults is the potential for cardiovascular effects such as

increased blood pressure and heart rate. This concern has led the Food and Drug Administration to recommend a warning label describing the cardiovascular risk of drugs used to treat ADHD (Nissen, 2006). Even so, a large-scale population-based study conducted by Habel et. al. (2011) reported no evidence for an increased risk of cardiovascular events associated with ADHD medication use in adults. More so, there was little support of risk associated with any specific medication or duration of use.

The mechanisms underlying ADHD are unclear, though, family and twin studies, indicate that genetics have a strong part in the etiology of ADHD (Cantwell, 1975; Kuntsi, Rijsdijk, Ronald, Asherson, and Plomin, 2005). A literature review of the earliest studies analyzing genetic components of ADHD suggests that family members of children with ADHD also showed symptoms of hyperactivity in childhood (Cantwell, 1975). This evidence proposes that the disorder is familial, being passed from generation to generation. The reviewed studies however do not provide proof of the genetic markers associated with ADHD.

Twin studies also provide evidence for a genetic factor in the development of ADHD. For instance, Kuntsi, Rijsdijk, Ronald, Asherson, and Plomin (2005) conducted a large-scale longitudinal twin study recruiting 3,541monozygotic (MZ) or dizygotic (DZ) twin pairs. Beginning at two years of age until about eight years of age, the researchers asked parents to rate the behavior of each twin on three scales measuring hyperactivity (at 2, 3, 4, 7, and 8 years of age) impulsivity (at 8 years of age), and inattention (at 4, 7, and 8 years of age). The logic behind utilizing MZ and DZ twins for this study weighs heavily on the fact that MZ twins share all of their genetic material while DZ twins share 50% of their genes. Therefore, if some measured behavior shares greater similarity in MZ twins than in DZ twins, this supports a genetic contribution to that behavior. When analyzing ADHD symptom scores at 8 years of age the results showed that MZ twins shared a greater correlation in their parent-reported symptom score than did DZ twins. The researchers concluded that such results

indicated substantial genetic contribution to ADHD symptoms. Though more compelling, this association study lacked evidence for the specific biomarkers associated with ADHD.

As technology advances, contemporary research studies are able to support the genetic factors of ADHD by in-depth genetic analysis. Specifically, a genome-wide association study allows researchers to scan human DNA for small genetic variances that occur more frequently in people with a certain disorder than people without the disorder (Genetics Home Reference – NIH, 2019). Fisher et. al. (2002) utilized this approach to examine loci influencing ADHD in affected sibling pairs. The investigators determined that four gene regions, 5p12, 10q26, 12q23, and 16p13, that may have a moderate effect on ADHD susceptibility. However, this loci-trait association did not meet a threshold of statistical significance. Nonetheless, the researchers state that a single major gene most likely does not influence the heredity of ADHD, but rather multiple loci. Conversely, Smalley et. al. (2002) reported a strong statistical association for ADHD symptoms and markers on certain areas of chromosome 16. Yet, a literature review of genetic linkage and association studies suggests that, though the evidence for a genetic etiology of ADHD is substantial, the relationship is complex, and the current research lacks conclusive reliability (Faraone & Mick, 2010).

Still, studies using a candidate gene approach suggest a strong association between ADHD and certain transporter genes, genes supporting the mechanisms of transportation of neurotransmitters between neuronal synapses (Gizer, Ficks, & Waldman, 2009; Khan & Faraone, 2006). Candidate gene association studies allow for researchers to analyze the genetic variation of a single relevant gene that is previously associated with the disorder of interest (Alghamdi & Padmanabhan, 2014; Modena, Doroudchi, Patel, & Sathish, 2019). For instance, a literature review assessing the validity of dopaminergic pathways in the susceptibility to ADHD found that the dopamine transporter gene (DAT1) may have multiple allele variations in specific gene regions that contribute to the genetic risk underlying ADHD (Gizer, Ficks, & Waldman, 2009). More so, the serotonin transporter gene (5HTT), including specified gene regions, have had substantial attention

as a strong candidate for ADHD (Gizer, Ficks, & Waldman, 2009). Specifically, the long variant of the gene has shown significant association with ADHD (Gizer, Ficks, & Waldman, 2009; Kent et. al., 2002; Khan & Faraone, 2006). Even with evidence of gene transporters, ADHD is considered a polygenic disorder with many genes most likely contributing to its phenotypic presentation (Khan & Faraone, 2006).

Other research suggests that environmental and lifestyle factors also contribute to the risk of developing ADHD. Exposure to tobacco smoke is a risk factor associated with ADHD symptoms (Motlagh et. al., 2010). Braun, Kahn, Froehlich, Auinger, and Lanphear (2006) for example, found that prenatal exposure to nicotine was significantly associated with parental reports of ADHD and stimulant medication use between ages 4 and 15 years. Furthermore, Freitag et. al. (2012) suggest that smoking during pregnancy is specifically associated with hyperactive-impulsive symptoms of ADHD. Additionally, parental mental health (Wustner et. al., 2019; Van Dyk et. al., 2015), birth complications (Van Dyk et. al., 2015), socioeconomic disadvantage (Russell, Ford, Williams, Russell, & Russell, 2016), exposure to certain environmental chemicals via breastmilk (Lenters et. al., 2019), and exposure to high levels of fluoridated water (Malin & Till, 2015) are implicated as risk factors associated with ADHD and ADHD symptoms. Although risk factors for ADHD are quite ambiguous, these risk factors mostly occur during pre-natal or early development which is consistent with the fact that ADHD is a neurodevelopmental disorder (Banerjee, Middleton, & Faraone, 2007). However, it is evident that post-natal factors, such as family dysfunction, significantly contribute to the severity of ADHD symptoms (Galéra et. al. 2011).

Numerous studies suggest that these genetic and environmental risks may modify the developing brain, which in turn cause deficits in neuropsychological functioning throughout the lifespan, though the directionality of this relationship is not well understood (Faraone & Biederman, 1998; Rajendran et. al., 2013; Seidman, 2006). For instance, Rajendran et. al. (2013) conducted a longitudinal study assessing the neuropsychological functioning of high and low risk preschool-aged

children prospectively over a 3- to 4-year period. Neuropsychological functioning was measured in five domains, which included, attention/executive functioning, language, visuospatial, sensorimotor, and memory. Greater neuropsychological functioning in preschool was associated with less severe inattention/hyperactivity impulsivity one year later. However, after the age of 4-5 years, greater ADHD severity appeared to impede development of neuropsychological functioning, suggesting evidence of a reciprocal relation between neuropsychological functioning and behavior.

An association between neuropsychological functioning and ADHD behaviors is also seen in adolescence and adulthood (Holst & Thorell, 2017; Seidman, 2006). Robinson and Tripp (2013) measured the neuropsychological function of previously diagnosed adolescent children (M=140.6, SD=17.9, in months) and a matched control group (M=140.7, SD=17.8, in months) on several neuropsychological assessments. The authors found that the ADHD group obtained significantly lower scores on measures of intellectual functioning, verbal working memory and attention, nonverbal fluency, and visuo-constructional abilities and visual memory when compared to the control group. This cross-sectional study limits ability to draw conclusions about temporal relation between these constructs. Halperin and colleagues (Halperin et al., 2008) found that children who had been diagnosed with ADHD in childhood performed more poorly on executive functioning measures in adolescence/young adulthood than a control group who had never had ADHD. When analyses were re-run by adult clinical status, individuals' whose ADHD persisted from childhood to adulthood showed the worst performance. Although a weakness of this study is that participants' neuropsychological functioning was not measured in childhood, this adds support to the idea that growth in neuropsychological functioning is associated with improved clinical status over time.

What is Obesity?

Obesity is an excess of weight beyond what is considered normal. It is often measured by body mass index (BMI) (Centers for Disease Control and Prevention, 2017b). BMI is calculated the same for children and adults by using the formula weight (lb) / [height (in)]^2 x 703 or weight (kg) /

[height (m)]^2 (Centers for Disease Control and Prevention, 2017b). However, the categorical interpretation of BMI differs for children and adults. For children, weight status is determined by using age and sex-specific percentiles. An underweight child would have a BMI at less than the 5th percentile for children of the same age and sex. The 5th percentile to less than the 85th percentile is considered "normal" BMI, the 85th percentile to less than the 95th percentile is considered "overweight", and the 95th percentile or greater is considered "obese" (Centers for Disease Control and Prevention, 2018b). Regarding adults, a BMI below 18.5 is considered "underweight", 18.5 to 24.9 is a "healthy weight", 25.0 to 29.9 is "overweight", and 30 or higher is "obese" (Centers for Disease Control and Prevention, 2017b).

Obesity is now believed to be a public health epidemic in America, with 2 in 3 adults being considered overweight or obese and 1 in 6 children and adolescents considered to have obesity (National Institute of Diabetes and Digestive and Kidney Diseases-NIH, 2017). Obesity rates are moderated by race and income. That is, the rates of obesity decreased as the white population of a state increased and the rates of obesity increased as income decreased (Menifield, Doty, & Fletcher, 2008).

Multiple factors are believed to contribute to obesity, including the physical environment, a phenomenon now coined "obesogenic environments" (Lake & Townshend, 2006). Briggs, Black, Lucas, Siewers and Fairfield (2019) assessed these risks by analyzing previously collected demographic data, lifestyle behaviors/ health factors (including BMI), and food availability variables. Food availability variables included density of "fast-food restaurants", density of "full-service restaurants", density of "grocery stores", density of "convenience stores", number of "fitness and recreation facilities" and county percent of households with no car and low store access as a "food store access" metric. Statistical analyses showed that a greater proportion of individuals living in areas with high-density fast food restaurants and full-service restaurants were also obese. Even after controlling for variables such as sex, age, education, and income, environmental characteristics were

positively associated with obesity. This data supports that the built environment can influence obesity rates.

These risk factors also extend from early life prenatal factors to modifiable lifestyle factors during adolescence and adulthood. For instance, maternal pre-pregnancy and gestational excessive BMI, maternal smoking during pregnancy, prenatal exposure to antibiotics, and birthing method (specifically caesarian section birth) are all associated with greater risk of childhood obesity in the offspring (Larqué, et. al., 2019). It is also suggested that a sedentary lifestyle, lack of physical activity, and smoking are significantly associated with obesity among adolescents (Hu, Ramachandran, Bhattacharya, & Nunna, 2018). In adults, unhealthy lifestyle habits such as lack of physical activity, unhealthy eating behaviors, lack of sleep, and high levels of stress have been noted as risk factors for overweight and obesity (National Heart, Lung, and Blood Institute- NIH, n.d.).

Along with environmental and behavioral risk factors, obesity may have an underlying biological component. Current research suggests that the variants of several candidate genes are responsible for irregular production of molecules, such as leptin, insulin, and adiponectin, which are involved in biological mechanisms related to control of human body fat, energy intake, and nutrient partitioning (O'Rahilly & Farooqi, 2006). For example, Thorleifsson et. al. (2009) implemented a genome-wide association study approach to determine the variants associated with measures of obesity (BMI or weight). The researchers found 11 loci that were significantly associated with obesity and or weight. When explaining the role of these genes that were significantly associated with measures of obesity, the researchers noted that more of the genes are involved with neural development or activity and fewer of the genes are involved in metabolic processes. This included variants that are expressed in regions of the brain related to feeding regulation.

Obesity is often associated with an increased risk of metabolic health conditions and even death. In fact, it has been suggested that obesity is a significant predictor of chronic medical conditions and poor physical health even more so than poverty or smoking (Sturm & Wells, 2001). A

literature review by Tagliabue, Principi, Giavoli, and Esposito (2016) explored the hypothesis that obesity is associated with other health conditions because obesity is associated with immune system dysregulation. It is well established that adipose tissue or fat is crucial in secreting biomolecules such as lipids, fatty acids, and other pro- and anti- inflammatory proteins. Collectively these molecules are called adipokines. It has been shown that the various adipokines are able to communicate with and influence other organs and cells including immune cells, the brain, and the heart. When there is an increase of adipose tissue present in the body, adipokine secretion does not function normally, which is believed to lead to chronic inflammation, modifications of immune system regulation, and therefore an increased susceptibility to infection (Grant & Dixit, 2015; Ouchi, Parker, Lugus, & Walsh, 2011; Romacho, Elsen, Röhrborn, & Eckel, 2014; Tagliabue, Principi, Giavoli, & Esposito, 2016).

This increased risk for disease is noted in several research studies. One study by the National Task Force on the Prevention and Treatment of Obesity (2000) found that of an estimated 15.6 million adults in the United States who have type 2 diabetes, 67% have a BMI of at least 27 and 46% have a BMI of at least 30. This positive correlation indicates that risk for the development of Type 2 diabetes increases as weight increases. Obesity is also shown to impact risk factors that are associated with coronary heart disease. Furthermore, increasing body weight is associated with nonalcoholic liver disease, ischemic stroke in men (Kurth, Gaziano, Berger, & Kase, 2002) and among women, irregular menstruation, gestational diabetes, certain forms of cancer, and an increased risk for death mostly due to cardiovascular causes (Overweight, Obesity, and Health Risk, 2000).

Unfortunately, the detrimental effects of obesity are noted as early as childhood. A follow-up study to a longitudinal cohort study found that men, but not women, who were overweight during adolescence had a higher relative risk for mortality (males RR=1.8, 95% CI [1.2,2.7]). However, men and women who were overweight as adolescents showed an increase in rates of diabetes, heart disease, atherosclerosis, and gout (Dietz, 1998). Another cohort study (Baker, Olsen, & Sørensen,

2007) found that higher childhood BMI heightened the risk for a heart disease event (fatal or nonfatal) in adulthood. The association strengthened with increasing age of the child. The authors speculate that BMI in late childhood reflects a greater accumulation of fat which increases the risk for heart disease in adulthood.

What is SES?

The American Psychological Association (APA, n.d.) describes socioeconomic status (herein referred to as SES) as the social standing or class of an individual or group. SES can be measured by assessing various factors, but most often includes occupational status, educational attainment, and/or income. For a more in-depth measure of SES, an individual's perceived social status/class, wealth, and home ownership status may be implicated. Gender and race/ethnicity are moderators of social status and therefore may also be considered in determining SES and various outcomes. For instance, The National Women's Law Center (Patrick, 2017) reports that women in the United States are 38% more likely than men to live in poverty and extreme poverty; with women of color being disproportionately represented in this population. Additionally, after analyzing survey data, Kingston and Smith (1997) found that African Americans and Latinos trail whites substantially in socioeconomic factors such as schooling, marriage rates, wealth, income, and report higher rates of chronic disease. Further the researchers found that socioeconomic factors play a major role in African American and Latino individual's ability to function with chronic illness.

SES is considered a consistent and reliable predictor of well-being and physical/mental health outcomes across the lifespan. Higher SES is often associated with access to good healthcare, important social connections, higher academic achievement, and overall better health. Lower SES is associated with decreased educational achievement, poverty, and poorer health habits (American Psychological Association, n.d.; APA Task Force on Socioeconomic Status, 2007; Chiu, 2016).

The results of Kittleson et. al. (2006) study makes evident the relation between SES and health outcomes exists across a lifetime. The researchers sought to measure incidence of coronary

heart disease (CHD) in adult white men as a result of childhood SES. To control for SES the researchers chose their sample from a population of graduated medical students at Johns Hopkins University; this way participants would be considered high SES in adulthood and the health outcome is not likely to be attributed to SES variations in adulthood. The researchers then measured childhood SES based on paternal occupation. For example, if the participant's father was a farmer, they would be considered low SES in childhood. If the participant's father was a physician, they would be considered high SES in childhood. Results revealed that of all male physicians who had experienced an event of CHD on or before the age of 50, the incidence rate was consistently significantly higher for men of a low SES childhood background, than in those of higher childhood SES. This risk for CHD associated with childhood SES was present even when controlling for other risk factors for CHD such as BMI and hypertension. These results highlight the continuous deleterious effects of low SES across the lifespan.

Menec, Shooshtari, Nowicki, and Fournier (2010) would agree with the results of Kittleson et. al. (2006), but further suggest that the relation between SES and health outcomes persists beyond the age of 50 years. Participants in the study included nursing home residents aged 65 years and older. After analysis of participant health measures and SES, the results showed that for certain health conditions, SES effects were present for individuals 65 to 74 years of age. Thus, confirming the importance of SES in later adulthood.

It is unclear how this SES-health connection occurs. Evans and Kim (2010) propose that this relation is due to multiple risk exposure. Multiple risk exposure refers to an individual experiencing more than one risk at a time. Risks include factors such as crowded and noisy living environments, high conflict families, and harsh and unresponsive parenting. These risks may simultaneously trigger other adverse events and circumstances such as job loss, divorce, teenage pregnancy, dropping out of high school, residential relocation, trauma, or a major illness. Each of these circumstances are inversely related to SES and are capable of compromising health. This theory further proposes that

persons of lower SES are exposed to more stressful life events, which are directly related to SES and multiple risk exposure. In other words, stressful life events are part of the index for multiple risks that contribute to SES. The authors posit that SES is related to multiple risk and multiple risk is related to health outcomes, making multiple risk a mediating variable of SES and health outcomes. This study emphasizes the complexities of studying SES and understanding how it affects health outcomes.

Chen and Paterson (2006) also highlight the complexities of SES by suggesting that SES is multidimensional and each level (individual, familial, and community) captures a unique aspect of SES. Often, social science research utilizes individual-level factors as their metric of SES while neglecting to understand SES at the community level. Community-level SES is the aggregate measure of social indicators of a group of individuals living in a defined community (Chen & Paterson, 2006; Quon & McGrath, 2015). Like the outcomes of individual level SES, community level SES is related to community members' health status and behaviors. For instance, a longitudinal study conducted by Roux et. al. (2001) found that adult participants who developed heart-disease were more likely to live in disadvantaged neighborhoods. While the reverse trend is noted in more advantaged neighborhoods. Another study suggests that neighborhoods of lower SES are associated with higher adolescent BMI. The researchers postulate that this is due to a lack of neighborhood resources such as parks and affordable healthy food options; as well as a lack of knowledge about healthy behaviors (Chen & Paterson, 2006). Furthermore, understanding community level SES is necessary for implementing policies that better the community and for decreasing negative health outcomes in a defined area.

Obesity and SES

There are multiple factors that contribute to obesity. One factor that is consistently associated with obesity is socioeconomic status. However, recent research highlights the complexity and variation of the relation between obesity and SES.

For instance, according to data from the CDC's National Health and Nutrition Examination Survey 2005-2008 (Ogden, Lamb, Carroll, & Flegal, 2010), in the United States obesity and SES differ by race and gender. Obesity prevalence among men is slightly higher at higher income levels, whereas, among women, obesity prevalence increases as income and education decrease.

This trend does not always persist when analyzing this relation globally. More specifically, these differential relations are seen to depend on the region being analyzed and the SES indicator chosen. That is, if the region is of a developed country or developing country and if the indicator is education, income, wealth etc. For instance, McLaren's (2007) review of this literature found that in highly developed countries, women of a lower SES were more likely to have higher BMI. In developing countries, there was a strong association between women with higher SES (usually indicated by education) and a higher BMI. For men, the association between SES and obesity was once again nonsignificant except in the case of developed countries where a negative association was noted when education was the indicator for SES. For men in countries that were rated "between developed and developing", a positive association was noted when income was the indicator for SES.

Several studies attempt to explain why obesity is associated with SES. Dubowitz et. al. (2012) propose evidence that the built environment is to blame. Using data from postmenopausal women in the Women's Health Initiative Clinical Trial, they created a "food environment index" (which included the availability of grocery stores and supermarkets and major fast-food restaurants), and a neighborhood SES construct (NSES; measured by (i) percent of adults older than 25 with less than a high school education; (ii) percent of males who were unemployed; (iii) percent of households with income below the poverty line; (iv) percent of households receiving public assistance; (v) percent of households with children headed by a woman; (iv) and median household income). The study sought to focus on the influence of neighborhood factors such as NSES and the food environment on BMI, with the underlying assumption being that access to resources conducive to a healthy lifestyle is associated with NSES. Regression analysis showed that as NSES increased, BMI

decreased, as the availability of grocery stores and supermarkets improved, the odds of being obese decreased, and as the availability of fast food chains increased, the odds of being obese increased. These results suggest that neighborhood variables (food environment and NSES) are consistently associated with BMI and obesity.

Another study, based in Singapore, interestingly implies that the experience of low subjective SES (SSES) potentially increases perceived deprivation, which stimulates appetite and contributes to the SES-obesity gradient (Cheon & Hong, 2017). To test this hypothesis, the researchers conducted three separate studies. The first involved manipulating SSES by prompting participants to either make a comparison between themselves and people who are relatively well off (low SSES condition) or worse off (high SSES condition). After this comparison, participants were asked to describe how it would be to have an interaction with the person to whom they compared themselves. After this manipulation, participants were asked what they would eat for their next meal if at a buffet. Results showed a marginal interaction in that individuals experiencing low SSES showed lower level of cognitive restraint towards food, which was predictive of a trend towards selecting high calorie foods. The second study involved participants performing the same SSES manipulation task, but then completing an implicit association task (IAT) in which they were required to categorize high and low caloric foods (i.e., pizza, fruits respectively) into categories using pleasant and unpleasant words (tasty, awful respectively). Participants in the low SSES condition tended to show a stronger implicit preference for high calories foods over low calories foods when compared to those in the high SSES condition. Two other studies were conducted consistently showing similar results in the food consumption patterns of individuals of the low SSES condition compared to the high SSES condition. The researchers further speculate that subjective perception of a lower SES and increased food intake may be of a preadaptation survival mechanism because this association is also noted across species.

Early life experiences are also thought to underly the association between obesity and SES (Wijlaars, Johnson,Van Jaarsveld, & Wardle, 2011). An analysis of infant weight and SES in a population of UK children found that at birth, there was no significant association between SES and obesity. However, at three months of age, infants from lower SES families had higher weights than infants from high SES families. Further, infants from lower SES families also had an increased chance of rapid growth. There are numerous factors that may underly the relation between birthweight and SES, including maternal smoking during pregnancy. Notably, the researchers found that the significance of this relation was diminished after accounting for breastfeeding. However, this study warrants further investigation on the effects of early infancy on the association between SES and obesity as this association is complex and varying.

ADHD and SES

Many studies find an association between ADHD and socioeconomic status although the SES-ADHD relation is complex and varying depending on the variable/s used to measure SES. The following studies explain the relation between SES and ADHD.

A population-based birth cohort study (Larsson, Sariaslan, Långström, D'Onofrio, & Lichtenstein, 2014) utilized data from a Swedish government population registry to determine the influence of family income on ADHD. Specifically, family income from the first five years of the child's life was used as an SES indicator. Then family income was divided into quartiles. An ADHD diagnosis was determined by either being prescribed a non-/stimulant for ADHD or being diagnosed with ADHD. Regression results revealed that ADHD seemed to be associated with lower family income during early childhood, suggesting that young children exposed to lower levels of family income are at an increased risk for ADHD. This association persisted even when accounting for covariates such as sex, parental mental health history, and maternal age at birth of the child.

A longitudinal study based in the United Kingdom shared similar results. Russell, Ford, and Russell (2018) analyzed the ADHD-SES relationship by measuring financial difficulty as the SES

indicator and parent-reported data on the hyperactivity subscale of the Strengths and Difficulties Questionnaire (SDQ) to measure ADHD symptoms. Financial difficulty was constructed by asking mothers to rate their current difficulty affording food, clothes, heating, rent/mortgage and other things considered essential for the child. Responses were then scored with higher scores indicating more difficulty. Financial difficulty was grouped by "no difficulty", "decreasing difficulty", "increasing difficulty", and "in difficulty". Analysis of the data showed a significant association between the "in difficulty" group and higher ADHD symptom scores than for any other group. Also, relative to the "no difficulty" group, the "increasing difficulty" and "decreasing difficulty" groups were associated with higher ADHD symptom scores. More so, as financial difficulty was defined more stringently, the hyperactivity score increased, suggesting that an experience of financial difficulty is associated with more severe ADHD symptoms.

Other studies utilizing a composite of SES variables also report an association between SES and ADHD. For instance, an analysis of data from a United Kingdom birth cohort measured SES by: parents' highest educational qualification, social class, family size, type of housing tenure (income based housing), family income (adjusted for number of children in the family), poverty status, family structure (single parent household), and an "index of SES" made from variables that were relatively stable over time (father's social class, mother's social class and paternal and maternal education). Results of the analysis indicated that ADHD diagnosis was associated with maternal education, family structure, housing tenure, and index of SES (with a higher score indicating lower SES) (Russell, Ford, Rosenberg, & Kelly, 2014). Likewise, Russell, Ford and Russell (2015) found that lower level of maternal education, lower income bands, lower housing bands, maternal marital status, and report of financial difficulty were associated with a large proportion of ADHD cases; with housing tenure, marital status, and financial difficulty being significant predictors of ADHD. Furthermore, financial difficulties proved to be the strongest predicator of ADHD.

Overall, the above evidence suggests an increased risk of ADHD in socioeconomically disadvantaged populations; quite consistently, with a financial indicator being a significant predictor of this risk. The causal workings of this relationship are not yet understood. Therefore, more research investigating ADHD and SES associations is needed.

Obesity and ADHD

Comorbidity between ADHD and obesity is increasingly documented in research literature though the causes of this relationship are unknown. For instance, in a study of the association between obesity and comorbid mental health in children aged 10-17, researchers found that ADHD was significantly associated with overweight and obesity (Halfon, Larson, & Slusser, 2013). This relation strengthened when controlling for stimulant use. These results are not surprising as stimulant medication is known to cause appetite suppression (Aguirre Castaneda et. al., 2016). The authors hypothesized that stimulant medication may reduce the risk of obesity by decreasing appetite and increasing impulse control.

An association between childhood ADHD and obesity in adulthood is also noted. A longitudinal study of childhood ADHD and non-ADHD controls reported that female participants with childhood ADHD are more likely than controls to be obese during childhood and young adulthood (Aguirre Castaneda et. al., 2016). Interestingly, the authors found no difference in obesity rates between ADHD cases treated with stimulants and ADHD cases not treated with stimulants. Also, among those treated with stimulants, obesity risk was not significantly associated with duration of treatment. Even more so, the researchers noted a higher BMI in ADHD cases that began stimulant treatment earlier. Based on these finding, the researchers conclude that treatment with stimulants during childhood does not appear to effect obesity into young adulthood.

Further, a population-based study found that obesity was most prevalent among participants with adult ADHD (met full childhood ADHD criteria with current symptoms) than among those with a history of childhood ADHD (met full childhood ADHD criteria with no current symptoms) or no

ADHD history (Pagoto et. al., 2009). Results also indicated that adult ADHD is significantly associated with binge eating disorder in the last year. Though the authors added stimulant use as a covariate, they did not report further analysis of its effects. The authors concluded that having adult ADHD increases the odds of being overweight and obese and that binge eating disorder in the past year mediates this association.

Again, it is unclear why ADHD populations have a high prevalence of obesity. One study suggests that this relation is due to impaired executive functioning which leads to lack of impulse control and therefore increased emotional eating (Dempsey, Dyehouse, & Schafer, 2011). This reasoning may explain why Pagoto et. al. (2009) found that binge eating disorder mediates the relation between obesity and ADHD. This reason is also supported by a study conducted by Graziano et. al. (2012). Graziano and colleagues analyzed the ADHD symptoms, BMI, executive functioning (EF), and medication use of children aged 10-18 years with a diagnosis of ADHD. Results showed a significant effect of medication use and BMI with children in the stimulant group having a lower BMI than children taking a non-stimulant or children who never took a psychotropic medication. Results also showed a very slight effect for EF on children's BMI. Specifically, children with better EF scores had a lower BMI. The researchers also found that EF scores could differentiate children's weight status. These results indicate co-occurrence of ADHD and obesity may indeed be due to impairment of executive function. However, it is also important to note that it is unclear if the effects of obesity are causing cognitive impairment or if EF impairment is a driver of ADHD and/or obesity.

Other studies point out that deletion of a specified chromosomal region is associated with childhood obesity and neurodevelopmental disorders such as ADHD (Shinawi et.al., 2011). Yet a review of the literature suggests that ADHD behaviors simply lead to poorer health behaviors and exercise habits (Nigg, 2013). Overall, these are complex relations with many moderating and mediating variables likely affecting risk for the disorders.

Current Study

This review of the literature highlights the intricacies underlying the relation between ADHD and obesity. To date, very few studies have looked at the ADHD-obesity relation in college students. Further, we find no reports on the moderating effects of SES on the ADHD-obesity association in college students. This population is important to study given the high number of individuals attending college with elevated ADHD (see Green & Rabiner, 2012, for a review), and how little is known about this population. We aimed to examine the association between ADHD and obesity among a diverse sample of college students attending a large public university. Additionally, we sought to examine socioeconomic status as a possible moderating variable of this ADHD-obesity relation, with particular emphasis on how neighborhood-level SES can add to our understanding of the relation between ADHD and obesity. We hypothesized that individuals with high ADHD symptoms would be more likely to be overweight/obese. We also hypothesized that this relation would be moderated by neighborhood-level SES, such that the ADHD-obesity relation would be stronger among individuals from more socioeconomically disadvantaged communities.

Methods

Participants

Individuals were recruited on a volunteer basis through emails to City College students, flyers, and the “SONA system”. SONA is an online system used to manage participant sign up to experiments taking place across the college and compensation for their participation. Participants were included if they were aged 18-40 years, were fluent in English, and were enrolled at CCNY part-time or full-time. They were excluded from the study if they had ever taken a neuroleptic or mood stabilizing medication (e.g. Risperdal, Zyprexa, Haldol, Clozapine, Lithium); had a history of major mental health problems or a neurological condition (e.g., history of traumatic brain injury, epilepsy, schizophrenia, bipolar disorder); had taken medicine for ADHD in the past 3 months; or

had smoked cigarettes or chewed tobacco in the past 3 months. Persons who had taken ADHD medication or used a nicotine product were excluded because of the appetite suppressant effects of these substances (Audrain-Mcgovern & Benowitz, 2011; Jo, Talmage, & Role, 2002; Wolraich, McGuinn, & Doffing, 2007; Zachor, Roberts, Bart Hodgens, Isaacs, & Merrick, 2006), and therefore potential to weaken the association between ADHD and obesity.

Of participants who signed up for the study (N=703), three people consented to take part, but then did not answer any screening questions. These participants were considered to have withdrawn from the study. Another 112 participants[1] were excluded because they were ineligible based on screening questions: younger than 18 or older than 40 years (n=22), not fluent in English (n=6), not a CCNY student (n=9), taken ADHD medicine in the past 3 months (n=17) or ever taken a mood stabilizer or neuroleptic medication (n=28), smoked cigarettes in the past 3 months (n=48), and history of TBI/major mental illness (n=18). Therefore, 568 participants were admitted into the study. The mean age of admitted participants was 20.74 years (SD = 3.20; min=18.01, max=39.46). The sample consisted of 166 people identifying as male (29.2%) and 402 people identifying as female (70.8%). The sample was ethnically (Latinx, n=224, 39.4%) and racially diverse. Eleven (1.9%) eligible participants self-identified as Native American or Native Alaskan, 168 (29.6%) as Asian, 136 (23.8%) as Black/African American, 68 (11.9%) as White, 2 (0.4%) as Native Hawaiian or Pacific Islander, 99 (17.7%) as biracial/multiracial or as a race other than those listed; 84 (14.7%) chose not to disclose their race. Most participants were enrolled at The City College of New York at the undergraduate level (n=564, 99.3%), 3 (0.5%) were at the master level, and 1 (0.2%) at the doctoral level. Regarding income, 62.6% (n=356) indicated having an annual household income of $39,999 or less, while 37.3% (n=212) had a household income of $40,000 of greater. Of the 568 participants, 70

[1] N for each reason for withdrawal listed may be greater than when summed due to great transparency.

(12.3%) indicated they were taking some sort of medication, with the highest frequencies being an antidepressant/SSRI (n=7), contraceptive (n=19), or vitamin/mineral (n=12).

Materials/Measures

BMI

Body mass index was measured using self-reported height (inches) and weight (pounds). This data was then converted into meters and kilograms and used to calculate BMI using the following formula (Freedman, Horlick, & Berenson, 2013):

$$BMI = weight\ (kg) / [height\ (m)]^2$$

BMI is interpreted using standard categories which are the same for adult men and women. A BMI <18.5 is categorized as "underweight," a BMI between 18.5 – 24.9 is categorized as "normal/typical," a BMI between 25.0 – 29.9 is categorized as "overweight," and a BMI ≥30 is categorized as "obese" (CDC, 2017b).

ADHD

Risk for attention-deficit/hyperactivity disorder was assessed using the ADHD Index scale of the Barkley Deficits in Executive Functioning Scale (BDEFS, Barkley, 2011). As a whole, the BDEFS is an empirically based assessment tool to evaluate executive functioning (EF) deficits in the areas of self-organization/ problem-solving, self-restraint, self-motivation, self-regulation of emotion, and self-management to time among adults aged 18-81 years. Respondents rate how often certain behaviors occur, with response options: "never or rarely," "sometimes," "often," and "very often". The current study employed the 11-item ADHD-EF Index score, which comprises items characteristic of adults with ADHD and is designed to measure risk for ADHD. Individual responses are summed to get a total score, which is converted to an age- and sex-normed percentile. Individuals were classified as Low or High ADHD in order to identify persons who were more likely to have ADHD. By dichotomizing this measure, the clinical implications are clearer (Barkley, 2011). This index has shown to be reliable for its use in clinical and research settings (Barkley, 2011).

Neighborhood Socioeconomic Status (NSES)

Participants provided the zip code of their current address, which was then converted to zip code tabulation area (ZCTA) and community district (CD) using Census Bureau data and a conversion file created by Newman Library at Baruch College, CUNY (https://www.baruch.cuny.edu/confluence/display/geoportal/Baruch+Geoportal+Home). ZCTAs are a trademark of the U.S. Census Bureau. They are generalized representations of Unites States Postal Service (USPS) zip code areas. One ZCTA can encompass many USPS zip codes. CDs are areas of the five New York City boroughs that are dependent on 59 community boards. Socioeconomic factors are the main determinants of a defined community district (NYC Planning, n.d.).

Zip codes, ZCTAs and CDs were then used to code community deprivation index of multi-variate indicators of neighborhood socioeconomic status (NSES). Domains and indicators for this NSES index are based on several commonly used multiple deprivation indices from around the world. These indices include: The English Indices of Deprivation 2019, The Social Deprivation Index (SDI), The CDC's Social Vulnerability Index (2016), and The Area Deprivation Index. Four domains were regularly used among the indices: education, housing, income, and living environment. For the purpose of this study, the domains used to determine NSES are education, housing/housing characteristics, family composition, economic security, and living environment. Specific indicators for each domain are listed in Table 1 (please refer to *Appendix A* for further details).

Table 1.

Neighborhood Socioeconomic Status Indicators

Indicator	Measure	Data Source
Education	Percent of population aged 25 years and older that has completed less than 9th grade or has no high school diploma	US Census 2012-2016 American Community Survey 5-Year Estimate
Housing	Percent of population with rent or mortgage ≥30% of household income	US Census 2012-2016 American Community Survey 5-Year Estimate
	Overcrowding; that is, the percent of population with more than 1 occupant per room	
Family Composition	Percent of population with female head of household	US Census 2012-2016 American Community Survey 5-Year Estimate
Economic Security	Percent of population receiving supplemental security income (SSI), cash public assistance, and food stamps/SNAP benefits (in the past 12 months)	US Census 2012-2016 American Community Survey 5-Year Estimate
	Percent of population with income in the past 12 months below the poverty level	
	Percent of the population unemployed	
Living Environment	Percent of population rating their neighborhood as not safe	NYC.gov Community Health Survey 2016
	Percent of population with a BMI of 30 or greater	
	Percent of population indicating they have consumed no fruits or vegetables yesterday	

Procedure

Participants signed up for the study through an online platform called the SONA System. They read a short abstract of the study and then clicked on a link to take them to the questionnaire. They completed online consenting and if they agreed to take part in the study, they answered a 7-question screening questionnaire to determine eligibility for the study. Participants who passed the screening questions were admitted into the study and directed to complete an hour-long online assessment survey covering ADHD, anthropometry, and demographic variables among other measures. Participants who signed up through SONA received 1 SONA credit, which they could exchange for extra credit in their courses. All other participants who signed up through emails or flyers were included in a draw for one of two $50 Amazon gift vouchers. This study was approved by

the CUNY Integrated IRB. Participants who chose this study were able to select from several experiments, thus they did not have to choose this study in order to get their extra credit. Alternatively, participants were able to complete other academic work to achieve extra credit rather than complete any experiment at all. Furthermore, participants who selected this study were able to opt out at any time without penalty.

Missing Data

Of the 703 individuals who signed up for the study, 579 were eligible and admitted into the study. However, 11 eligible individuals were excluded because they did not provide a valid zip code. Independent sample t-tests showed that there were no significant differences between those who did and did not provide a valid zip code for age, $F(1,577)=0.98$ and BMI, $F(1,577)=2.40$. Chi square analyses showed that there were no significant differences between those who did and did not provide a valid zip code for: gender, $\chi^2(1, N=579)=0.02$; ethnicity, $\chi^2(1, N=579)=0.04$; race, $\chi^2(1, N=493)=2.72$; enrollment status, $\chi^2(1, N=579)=0.08$; income band, $\chi^2(1, N=579)=1.07$; and medication use, $\chi^2(1, N=579)=1.54$ (all $p > .05$).

Data Analysis

First, ADHD severity and BMI were checked for normality. As they were not normally distributed, median analyses or non-parametric analyses were carried out where appropriate. To test the hypotheses that individuals with high ADHD symptoms would be more likely to be overweight/obese and that this relation would be moderated by neighborhood-level SES, we first calculated the median ADHD severity and median BMI. The association between ADHD severity and BMI was examined using a Spearman's rho correlation analysis. An Independent-samples Kruskal-Wallis test was performed to test group differences (control group, high BMI only group, high ADHD only group, comorbid group; independent variables) on weight, height, BMI, and ADHD severity (dependent variables). A chi square analysis examined the relationship between the categorical variables ADHD (high or low) and BMI (high or low).

To test the second part of our hypothesis, the variables used to measure neighborhood socioeconomic status (NSES) were first square root transformed to fit normality. Group differences (control group, high BMI only group, high ADHD only group, comorbid group; independent variables) in NSES were then observed by performing a univariate analysis of variance for each NSES variable (dependent variable). A Pearson product-moment correlation was conducted to test the relations among the NSES variables. To determine if our socioeconomic status variables loaded onto an underlying neighborhood SES latent construct, we carried out a maximum likelihood factor analysis with direct oblimin rotation. Next, a Pearson product-moment correlation was run to examine the relationship between the resultant two factors. We then performed a univariate analysis of variance for each yielded factor (dependent variable) to test between-group (control group versus high BMI only group versus high ADHD only group versus comorbid group; independent variables) differences in the mean factor score. A multivariate analysis of variance was also carried out to examine mean differences of the yielded factor scores (dependent variables) by ADHD severity (low versus high group; independent variables). Finally, we tested the interaction of ADHD and NSES (predictor variables) on BMI status (low and high; response variable). All analyses were completed in IBM SPSS Statistics v. 25.

Results

Association Between ADHD and BMI

Participants rated the frequency with which ADHD behaviors have occurred over the past six months. Based on their reports, the median (interquartile range) ADHD severity was 19 (15-22) (Table 2). Using a cut-off threshold of a raw score 1 or more standard deviations above the mean of the normative sample, 75.6% (n=428) of the sample fell into the Low ADHD group, while 24.4% (n=138) were classified as High ADHD.

Participants also self-reported their height (inches) and weight (pounds), from which BMI was calculated according to the CDC (2017b) (Table 2). Participants' median (interquartile range)

BMI was 23 (20.60-26.60), which falls in the typical range (CDC, 2017b). A small minority of participants had BMI in the "Underweight" range (n=39, 6.9%). The majority of participants' BMI fell in the "Typical" range (n=334, 59.2%). These groups were combined to create a "Low BMI" group (n=373, 66.1%). Approximately one third of participants had elevated BMIs, with 23.0% (n=130) of participants categorized as "Overweight" and 10.8% (n=61) participants categorized as "Obese." These two groups were combined to form a High BMI group (n=191, 33.8%).

The association between participants' ADHD severity and BMI was examined using a Spearman's rho correlation analysis. A non-significant, positive correlation emerged between BMI score and ADHD index score when looking across the entire sample, $r_s(560)=.042$, $p= 0.32$.

Individuals were classified as Low/High ADHD and Low/High BMI based on established cut off scores (see Method section). Approximately half of the sample fell in the "Control" group (n=280, 49.8%); these individuals had underweight to normal BMI and an ADHD index score <1SD above the mean. The "High BMI only" group (n=145, 25.8%) included individuals with an overweight to obese BMI and an ADHD index score <1SD above the mean. The "High ADHD only" group (n=91, 16.2%) included individuals with an underweight to normal BMI and an ADHD index score ≥1SD above the mean. The "Comorbid" group (n=46, 8.2%) included individuals with an overweight to obese BMI and an ADHD index score ≥1SD above the mean. Table 2 shows the median (interquartile range) for height, weight, BMI, and ADHD severity as a function of group.

Table 2.

Descriptive Statistics of Anthropometric Measures Across the Whole Sample (N=568)[a]

Variable	Total	Controls (1)	High BMI Only (2)	High ADHD Only (3)	Comorbid (4)	*H* (df)	*p*	Pairwise Comparisons
	Median (IQR)[b]	Median (IQR)	Median (IQR)	Median (IQR)	Median (IQR)			
ADHD Severity	19 (15-22)	17 (14.25-20)	17 (15-20)	26 (24-28)	26 (24-29)	311.85 (3)	<.0001	1=2<3=4
Height (in.)	65 (62-68)	65 (63-68)	64 (61.2-68)	65 (63-68)	64.5 (61.38-68)	5.05 (3)	.17	
Weight (lbs.)	140 (120-165)	126.9 (114.25-144.9)	168.5 (150-185.25)	130 (116-143)	167.05 (148.75-206.25)	227.62 (3)	<.0001	1=3< 2=4
BMI	23.03 (20.60 – 26.60)	21.41 (19.92-23.03)	27.79 (26.42-30.86)	21.82 (19.23-23.03)	28.21 (26.76-31.49)	377.68 (3)	<.0001	1=3< 2=4

[a]Ns may differ due to missing values; [b]IQR=interquartile range.
Note. ADHD Severity measured with the Barkley Deficits in Executive Functioning Scale (BDEFS) ADHD Index (Barkley, 2011). Height (inches) and weight (lbs) self-reported by participants and used to calculate BMI: BMI=weight (kg)/[height (m)]^2.

Results of an Independent-samples Kruskal-Wallis test (Table 2) shows significant group differences in average rank for weight, BMI, and ADHD severity, but not height. For weight and BMI, Controls and High ADHD Only groups did not differ from each other. Their average rank of weight and BMI were significantly lower than High BMI Only and Comorbid group participants, who did not differ from each other. For ADHD severity, the average rank for Controls and High BMI Only was significantly lower than for High ADHD Only and Comorbid groups, who did not differ from each other.

The association between ADHD and BMI group status was also examined, $\chi^2(1, N=562)=0.014$, p =.91, V=.005. Individuals with High ADHD were no more likely to be in the High BMI group (n=46, 33.6%) than individuals with Low ADHD (n=145, 34.1%) (see Figure 1).

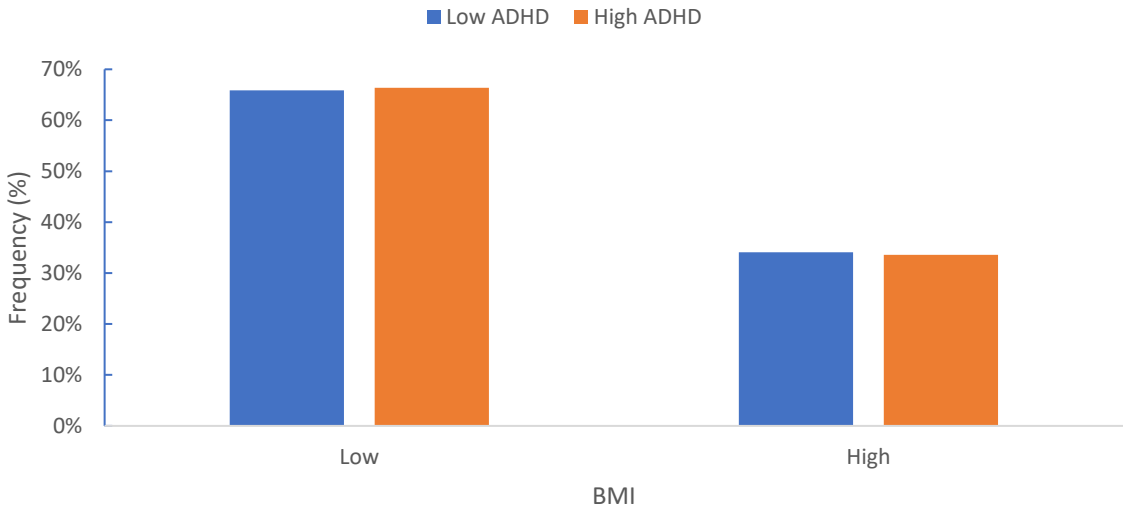

Fig 1. *Frequency of Participants Classified as Low/High BMI as a Function of ADHD Status (Low, High).*

Neighborhood Socioeconomic Status (NSES)

Participants provided their zip code which was used to determine neighborhood socioeconomic status (NSES). NSES variables including: percent receiving public assistance; percent with >1 person per room; percent who perceive neighborhood as unsafe; percent with income in the past 12 months below the poverty line; unemployment rate; and percent of female head of householders, were square root transformed to better fit assumptions of normality. To test for group differences in NSES variables, a univariate analysis of variance was performed for each NSES variable (see Table 3). The analysis indicated that a significant group difference was detected only for percentage of individuals who did not consume fruits or vegetables yesterday, such that individuals in the High BMI Only group were from neighborhoods where significantly fewer fruits/vegetables were consumed compared to those in the High ADHD Only group.

Table 3.

Neighborhood SES Variables as a Function of Group

Neighborhood SES Variable	Total	Controls (1)	High BMI Only (2)	High ADHD Only (3)	Comorbid (4)	F (df)	P	ES
Mean Percent of Female Head of Householders	20.64 (9.21)	20.16 (9.09)	21.98 (9.61)	19.85 (9.01)	20.64 (8.87)	1.398 (3, 557)	0.242	0.007
Unemployment Rate	9.44 (3.29)	9.21 (3.17)	9.89 (3.57)	9.30 (3.32)	9.69 (3.29)	1.446 (3, 557)	0.229	0.008
Mean Percent with Income in the Past 12 Months Below the Poverty Line	21.59 (10.15)	21.07 (10.35)	22.33 (10.26)	21.27 (9.38)	23.08 (10.14)	1.064 (3, 557)	0.364	0.006
Mean Percent Who Perceive Neighborhood as Unsafe	17.75 (10.60)	17.61 (10.22)	18.41 (10.92)	16.40 (10.59)	19.23 (11.79)	0.904 (3, 486)	0.439	0.006
Mean Percent with a BMI ≥ 30	25.31 (7.44)	25.21 (7.48)	26.32 (7.10)	23.98 (7.64)	25.53 (7.63)	1.717 (3, 490)	0.163	0.010
Mean Percent Who Did Not Consume Fruits or Vegetables Yesterday	13.70 (4.62)	13.61 (4.60)	14.54 (4.57)	12.73 (4.82)	13.64 (4.21)	2.689 (3, 490)	0.046, 2>3	0.016
Mean Percent Without a Highschool Diploma	22.77 (9.84)	22.46 (9.83)	22.69 (10.26)	22.75 (9.25)	24.93 (9.70)	0.834 (3, 557)	0.476	0.004
Mean Percent with Housing Costs Over 35% of Gross Household Income	44.13 (9.62)	44.25 (9.43)	43.85 (9.77)	44.51 (9.45)	43.51 (10.92)	0.165 (3, 557)	0.920	0.001
Mean Percent with >1 Person Per Room	10.53 (5.70)	10.28 (5.98)	10.64 (5.51)	11.04 (5.51)	10.64 (4.90)	0.853 (3, 557)	0.465	0.005
Mean Percent Receiving Public Assistance	12.41 (6.67)	12.05 (6.70)	12.89 (6.93)	12.41 (6.32)	13.13 (6.38)	0.840 (3, 557)	0.472	0.005

Note. For ease of interpretation raw means (SDs) are shown.

Table 4.

Pearson Correlations Among Neighborhood SES Variables

Neighborhood SES Variable	1	2	3	4	5	6	7	8	9	10
1. Mean Percent of Female Head of Householders	-									
2. Unemployment Rate	.76*	-								
3. Mean Percent with Income in the Past 12 Months Below the Poverty Line	.65*	.60*	-							
4. Mean Percent Who Perceive Neighborhood as Unsafe	.75*	.64*	.80*	-						
5. Mean Percent with a BMI ≥ 30	.83*	.72*	.47*	.72*	-					
6. Mean Percent Who Did Not Consume Fruits or Vegetables Yesterday	.72*	.67*	.53*	.72*	.75*	-				
7. Mean Percent Without a Highschool Diploma	.58*	.50*	.81*	.66*	.38*	.40*	-			
8. Mean Percent with Housing Costs Over 35% of Gross Household Income	.32*	.23*	.35*	.33*	.32*	.17*	.56*	-		
9. Mean Percent with >1 Person Per Room	.37*	.32*	.65*	.43*	.22*	.27*	.79*	.66*	-	
10. Mean Percent Receiving Public Assistance	.78*	.74*	.91*	.82*	.64*	.64*	.79*	.33*	.58*	-

Note. The following correlations use the square root transformed variable.

*p<.01.

The relations among NSES variables were analyzed using the Pearson product-moment correlation. Results showed positive significant correlations amongst all NSES variables (Table 4). That is, higher levels in one NSES variable was associated with higher levels in the other variables.

A maximum likelihood factor analysis with direct oblimin rotation was carried out to reduce the number of factors to underlying latent constructs. Two factors emerged, accounting for 57.90% and 13.18% of the variance. Table 5 shows the resultant structure matrix, representing the correlations between each variable and the underlying factors.

Table 5.

Structure Matrix for Factor Analysis

Neighborhood SES Variables	Factor 1	Factor 2
	"Health and Safety"	"Economic Security"
Mean Percent of Female Head of Householders	0.902	0.424
Mean Percent with a BMI ≥ 30	0.865	0.282
Mean Percent Who Perceive Neighborhood as Unsafe	0.843	0.648
Mean Percent Who Did Not Consume Fruits or Vegetables Yesterday	0.804	0.335
Unemployment Rate	0.821	0.342
Mean Percent with Income in the Past 12 Months Below the Poverty Line	0.700	0.818
Mean Percent Without a Highschool Diploma	0.524	0.918
Mean Percent Receiving Public Assistance	0.846	0.740
Mean Percent with >1 Person Per Room	0.250	0.769
Mean Percent with Housing Costs Over 35% of Gross Household Income	0.210	0.508

The variables "Mean Percent of Female Head of Householders", "Mean Percent with a BMI ≥ 30", "Mean Percent Who Did Not Consume Fruits or Vegetables Yesterday", "Mean Percent Who Perceive Neighborhood as Unsafe," and "Unemployment Rate" loaded heavily on

Factor 1. Factor 2 showed high loadings for the variables “Mean Percent with Housing Costs Over 35% of Gross Household Income”, “Mean Percent with >1 Person Per Room”, and “Mean Percent Without a Highschool Diploma”. The variables related to income - “Mean Percent with Income in the Past 12 Months Below the Poverty Line” and “Mean Percent Receiving Public Assistance” – split their variance across Factor 1 and Factor 2.

Based on the structure matrix we concluded that Factor 1 represents health and safety. It comprises the variables: “Mean Percent of Female Head of Householders”; “Mean Percent with a BMI ≥ 30”; “Mean Percent Who Perceive Neighborhood as Unsafe”; “Mean Percent Who Did Not Consume Fruits or Vegetables Yesterday”; “Unemployment Rate”; as well as the cross-loadings from “Mean Percent with Income in the Past 12 Months Below the Poverty Line” and “Mean Percent Receiving Public Assistance”. Factor 2 variables appear related to Economic Security. It comprises housing (“Mean Percent with >1 Person Per Room” and “Mean Percent with Housing Costs Over 35% of Gross Household Income”), education attainment (“Mean Percent Without a Highschool Diploma”) and the cross-loadings from the income variables (“Mean Percent with Income in the Past 12 Months Below the Poverty Line” and “Mean Percent Receiving Public Assistance”).

A Pearson product-moment correlation was run to examine the relationship between the two factors. The correlation coefficient suggests that factor 1 and factor 2 share a moderate positive significant association, $r(493)=.501$, $p<.01$. That is to say, higher levels in factor 1 are associated with higher levels in factor 2.

Given the two factors were significantly correlated, analysis of the association between ADHD status (high/low; independent variables) and NSES (factor 1 and factor 2; dependent variables) was conducted using MANOVA. No significant multivariate effects were found on

factor 1 or factor 2, Pillai's Trace= .004, $F(2,490)=.972$, p=.379. This means that NSES did not differ by ADHD status.

To test the interaction of ADHD and NSES on BMI, a logistic regression was computed. More specifically, we modeled the relationship between ADHD (low), NSES (factor 1 and factor 2), the interaction between ADHD and factor 1, and the interaction between ADHD and factor 2 as predictor variables of BMI (low or high). Table 6 shows that the variables were not significantly associated with BMI status.

Table 6.

Logistic Regression Results for the Interaction of ADHD (Low, High) and Neighborhood Socioeconomic Status on BMI (Low, High)

Variable	B	SE	Wald	df	Sig	OR	95% CI
ADHD	-0.03	0.22	0.02	1	0.898	0.97	0.63 – 1.50
Factor 1	0.22	0.13	2.77	1	0.096	1.25	0.96 – 1.62
Factor 2	-0.16	0.14	1.47	1	0.226	0.85	0.65 – 1.12
ADHD x Factor 1	-0.11	0.26	0.18	1	0.673	0.90	0.54 – 1.50
ADHD x Factor 2	0.28	0.27	1.08	1	0.300	1.32	0.78 – 2.25
Constant	-0.65	0.11	34.63	1	0.000	0.52	

Note. ADHD is coded 0 (Low, indicator), 1 (High).

Discussion

The ADHD-Obesity Relation

The purpose of the current study was to examine the ADHD-obesity association and to consider neighborhood-level socioeconomic status (NSES) as a moderating variable of this relation. Our sample data indicates that college students with current high levels of ADHD behaviors are not more likely to be overweight or obese. This finding contradicts previous findings which report an association between excess weight and adult ADHD (Alfonsson,

Parling, & Ghaderi, 2012; Instanes, Klungsøyr, Halmøy, Fasmer, & Haavik, 2018; Pagoto et. al., 2009).

Differences in sampling strategy may explain this discrepancy. For instance, a review of the literature on the adult ADHD-obesity relation notes that studies conducted in a clinical setting tend to find an association between adult ADHD and obesity or above average BMI (Instanes, Klungsøyr, Halmøy, Fasmer, & Haavik, 2018). However, these results are less consistent in a community-based sample, which was used in the current study. Furthermore, our participants being college students may be another issue that affected the likelihood of obtaining significant results. According to previous research, college students with ADHD tend to have higher levels of cognitive functioning, better coping skills, and higher ability levels compared to their non-college counterparts (Frazier, Youngstrom, Glutting, & Watkins, 2007; Gray, Fettes, Woltering, Mawjee, & Tannock, 2016). Given that executive functioning deficits, common to both ADHD and obesity, is one of the proposed mechanisms accounting for higher obesity levels among those with ADHD (Cortese & Morcillo Peñalver, 2010) the stronger executive skills of college students with elevated ADHD behaviors may explain the non-significant findings in this study.

This inconsistency between previous literature and current findings may also be explained by accounting for comorbidity of other disorders such as binge eating disorder and depression. For example, Alfonsson, Parling, and Ghaderi (2012) found that adult ADHD was more common in bariatric surgery candidates (10.2%) than in the general population (4.7%; Murphy & Barkley, 1996b). The authors further report that screening positive for adult ADHD was significantly associated with symptoms of anxiety and depression (Alfonsson et al., 2012). Similarly, Pagoto et al. (2009) found that while adults with ADHD were more likely to be

overweight or obese than controls, binge eating disorder partially mediated this association. A longitudinal study conducted by Biederman et. al. (2010) found that ADHD was not significantly associated with weight outcomes, but rather comorbid major depression better explained weight gain in adult females with ADHD.

Since ADHD manifestation and outcomes vary by gender, our majority female sample may have skewed the hypothesized results (Quinn & Madhoo, 2014). Research shows that females with ADHD are predominately diagnosed with the inattentive ADHD subtype, have predominately internalizing symptoms, and tend to have more effective coping strategies (Quinn, 2005; Quinn & Madhoo, 2014). Given that we used a rating scale to identify ADHD-like behaviors rather than the gold-standard psychiatric interview, females with internalizing problems (which share many symptoms with ADHD – poor attention, restlessness for example) may have appeared to have elevated ADHD, when in fact they did not. This may account, in part, for the weakened relation between ADHD and obesity observed in this study.

Regarding the methodology of the current study, it is possible that our eligibility criteria restricted ADHD positive adults with comorbid disorders from participating in the study. Our exclusion of participants who take ADHD medications may have also limited us from examining individuals with most severe ADHD. Loosening eligibility criteria may have allowed us to detect a relation between ADHD and obesity, although this would have created other difficulties, including how to manage participants' medication use in analyses.

NSES as a Moderator of the ADHD-Obesity Relation

The hypothesis that the ADHD-obesity relation would be moderated by neighborhood-level SES was also not supported by the current data. Literature examining socioeconomic status, let alone community level socioeconomic status, as a moderator of the relation between ADHD

and obesity is scarce. One large scale study documenting the association between ADHD, obesity, and socioeconomic status indicates that children with ADHD are more likely to be overweight and of a lower socioeconomic background than their non-ADHD counterparts (Waring & Lapane, 2008). Most of the literature, however, focuses on the direct associations between ADHD and SES or obesity and SES.

It has been documented that adults with ADHD tend to have a lower socioeconomic standing, measured as household income, when compared to controls (Biederman & Faraone, 2006). An analysis of longitudinal data (Fletcher, 2014) reports that a diagnosis of childhood ADHD is associated with decreased earnings in adulthood when compared to sibling controls. Similarly, Altszuler et al. (2016) found that young adults with ADHD were more likely to experience financial difficulties and childhood ADHD diagnosis was a significant predictor of these negative financial outcomes.

The relation between SES and obesity is well known. As stated in the literature review, this association may differ by sex and SES indicator. In America, the obesity prevalence of men is similar across all income levels, with obesity prevalence tending to be higher for men of higher income levels (Ogden, Lamb, Carroll, & Flegal, 2010). Although, McLaren (2007) suggested that this association among men will vary by SES indicator. For instance, a negative association is observed when SES is measured as education attainment (McLaren, 2007). For women, lower income is associated with an increase in obesity prevalence (Ogden, Lamb, Carroll, & Flegal, 2010). Furthermore, women's obesity prevalence increased as education decreased.

Our approach to measuring SES differed from methods used in previous research. Past studies have primarily looked at socioeconomic standing at the individual level by measuring

variables such as personal income, family income, job attainment, or financial stress/difficulty. The current study differs in that we focused on socioeconomic standing from a community level with the assumption being that the economic standing of a neighborhood will influence individual outcomes, like obesity and mental health.

The concept of the built environment supports our theory that neighborhood social standing effects public health outcomes (Briggs, Black, Lucas, Siewers and Fairfield, 2019; Hatch et.al., 2011; Silva, Loureiro, & Cardoso, 2016). The research on outcomes of the built environment suggest that a poor-quality physical environment may expose individuals to risk factors that contribute to poor health (Perdue, Stone, & Gostin, 2003). Further, low income communities tend to be associated with more negative characteristics of the built environment (Abramovitz & Albrecht, 2013) such as less access to fresh foods (Briggs, Black, Lucas, Siewers and Fairfield, 2019) and poorer municipality services (Evans & Kantrowitz, 2002). Health disparities by socioeconomic status are further exaggerated when considering the health of racial and ethnic minorities in relation to whites (Merkin et.al., 2009). These inequalities emphasize the importance of understanding the built environment and socioeconomic factors of a neighborhood in relation to public health outcomes (Perdue, Stone, & Gostin, 2003). Moreover, there is an increasing effort for social research to focus on socioeconomic determinants of health and health disparities (Boyce & Olster, 2011; Danis & Pesce, 2012). Our study tackled this task by examining health outcomes of neighborhood-level SES in a diverse population of college students. To our knowledge, this is the first study to examine these associations.

Although assessing socioeconomic status in relation to health is important. Our data did not support our proposed hypothesis for this association. This may be the case because we did not consider any individual level socioeconomic measures, which may interact with

neighborhood-level characteristics. The approach of previous studies has been to measure some form of individual-level income alongside their index of neighborhood socioeconomic variables. For instance, Wainwright and Surtees (2004) measured the impact of community level socioeconomic status on health by analyzing the interaction of individual level measures (i.e., employment status, social class, and education attainment) and area level measures (using a 32-variable index of multiple deprivations). The authors found that combinations of individual-level factors and area level factors contributed to lowest levels of physical health. The authors also report that the magnitude of association at the area level was modest compared to the magnitude of association at the individual level.

It is also possible that our hypothesis was not supported because individual level socioeconomic factors, such as education, are protective against the effects of living in a more disadvantaged neighborhood. This is especially important when considering that our sample comprised individuals currently attending a 4-year university. Research supports that lower level of education is significantly associated with worse mental health (Silva, Loureiro, & Cardoso, 2016) and an increased obesity prevalence among women (Ogden, Lamb, Carroll, & Flegal, 2010). This may partially explain why our hypothesis on the association between SES-obesity and SES-ADHD was not supported by the results of this study.

Limitations

Multiple limitations exist in our current study. The first relates to how we measured ADHD. ADHD symptoms and severity often emerge during childhood, and persistence through adolescence and/or adulthood often signals more severe manifestations of the disorder. One potential limitation of our study is that ADHD severity during childhood was not accounted for. This measure is important because ADHD severity during childhood tends to be a significant

predictor of outcomes in adolescence and adulthood (Miller, Nevado-Montenegro, & Hinshaw, 2012; Yang, Tai, Yang, & Gau, 2013). Much research assessing ADHD outcomes analyze ADHD from the childhood perspective, including research on the ADHD-SES and ADHD-obesity relations. Therefore, capturing childhood ADHD severity and current ADHD severity may have better explained the ADHD-obesity relation in this study.

BMI and ADHD are both continuous constructs, which are often classified into groups. The group classifications of BMI are based on standard cut points for underweight, normal weight, overweight, and obese groups. Individuals classified as having ADHD meet a set number of symptoms according to the DSM-5 (APA, 2013). In the current study, we dichotomized BMI and ADHD into high and low levels using cut-off scores, consequently sacrificing power. Using continuous measures of ADHD and BMI in our analyses may have allowed for better sensitivity.

There is also the possibility that our measure of ADHD was not reliable. Since several symptoms of ADHD are common to other mental health disorders, including anxiety and depression (e.g., restless, difficulty sustaining attention) (Montano & Weisler, 2011), it is possible that our "High ADHD" group may have included individuals presenting with severe symptoms of comorbid disorders. In this case the relation between ADHD-obesity and SES may have been weakened. Previous work has suggested that ADHD in adulthood with no childhood history of the symptoms is often explained by a comorbid disorder, including depression, anxiety and substance use (Sibley et al., 2018). A possible solution to overcome this limitation of the present study may be to implement a more stringent measure of ADHD and give a psychiatric interview to assess each ADHD symptom in more detail, and to ascertain levels and nature of comorbidity.

Our measure of geographic area may have also skewed our intended results. Zip code tabulation area is a generalized representation made up of multiple zip codes. This means that the geographic area we measured may conflate zip codes that have varying community socioeconomic standings. ZCTA may be too large of a measure to indicate differences of SES. It may have been beneficial to use individual street addresses rather than zip code to have a more sensitive measure of NSES. In addition, we asked participants for their zip code and assumed that the zip code was for their permanent residence when it may have been for a temporary residence. We could have mitigated this issue by asking participants if the zip code they provided was for their permanent address. This may have allowed for a more accurate measure of the effects of neighborhood SES.

Another limitation of the current study is that we only had access to participants' self-reported height and weight to determine BMI status. This is problematic in that female and overweight individuals tend to under report their weight and males tend to overstate their height (Bowring et. al., 2012; Elgar, Roberts, Tudor-Smith, & Moore, 2005; Sherry, Jefferds, & Grummer-Strawn, 2007). Due to potential inaccurate reports of height and weight, participant BMI status for our sample may not be as accurate as if it had been measured objectively.

A final limitation of this study may be that most of the participants in our sample fell within the control group. Having a larger sample may have allowed for a more even distribution of participants in each comparison group and an increased likelihood of obtaining valid results for ADHD severity on NSES. Having fewer participants in the "High BMI Only", "High ADHD Only", and "Comorbid" group may have limited our ability to detect significant results.

Contributions of the Current Study

In recent years there has been greater effort to study how ADHD-like behaviors in childhood impact later functioning. ADHD-SES and ADHD-obesity studies especially, tend to focus on how ADHD symptoms in childhood impact an outcome, rather than analyzing these associations from an adult ADHD symptom severity perspective. This study contributes to our current understanding of the ADHD-obesity association in a diverse population of young adults.

The diversity of our sample is especially notable because diversity of variables such as gender, age, race, and ethnicity, is often ignored in ADHD research (Gingerich, Turnock, Litfin, & Rosén, 1998). For instance, demographics of national level data on ADHD in children and adolescents show that majority of participants were male, white, and non-Hispanic/Latino (Danielson, Visser, Chronis-Tuscano, & DuPaul, 2018). Another large-scale national survey on ADHD in children and adolescents showed participant demographics were equal for sex, but not for race, with white participants out numbering participants who identified as Black or "other" (Danielson et. al., 2018). A study on adult ADHD shows a similar lack of diversity with most participants being male and non-Hispanic white (Kessler et. al., 2006). Participants of the current study were ethnically and racially diverse with most participants being young adult females. Our study, therefore, contributes to the little availability of ADHD research with diverse samples.

In addition, this study emphasizes an increasingly important topic in the field of psychology, socioeconomic status, specifically at the neighborhood level. Socioeconomic status across all levels (individual, communal, and familial) is a significant determinant of physical and mental health outcomes across the life span (Oakes & Rossi, 2003). We benefit from an understanding of lower socioeconomic status and outcomes of low socioeconomic status by gaining perspective on social structure and public health in our society (American Psychological Association, Task Force on Socioeconomic Status, 2007). Insight on the SES of communities in

New York City is also of great importance for implementing policies that help reduce socioeconomic and health disparities in the city.

Future Direction

Future direction of this study should continue to focus on NSES as a moderator of the ADHD-obesity relation. It may be beneficial to broaden the index of SES variables in accord with recommendations from the American Psychological Association (APA), Task Force on Socioeconomic Status (2007). This should include measuring social class. Specifically taking into consideration, how one's connection to institutions, social networks, communities, and social policies may benefit some while oppressing others. Contributions of individual level SES variables should also be of focus. This includes individual education attainment, income, and occupation status. According to the APA (2007), education may be one of the most essential aspects of SES.

Further, the APA (2007) recommends the assessment of different dimensions of SES and understanding of how each dimension contributes to an outcome, rather than trying to understand the contribution of a composite measure of SES on an outcome. For example, the domain in the current study, "living environment", should include more variables related to the outcome of obesity (i.e., number of fresh food grocers, access to private or public transit, access to clean parks). Based on the APA recommendation, this domain alone, is sufficient to measure a dimension of socioeconomic standing and associated outcomes.

The model used in this study (NSES as a moderator of the ADHD-BMI relation) assumes linearity between ADHD severity and BMI status. However, it is possible that a curvilinear relationship exists between ADHD and BMI. Alternative models of the relations among ADHD, SES, and BMI should be examined, including potential mediating or moderating effects of other

risk and protective factors. For example, since SES and BMI tend to vary by gender, it may be beneficial to consider the moderating effects of gender on the current model. Research on mediation- moderation models for prevention science does not recommend testing moderators and mediators in the same analyses due to difficulty of justifying the model and interpreting the effects (Fairchild & MacKinnon, 2009; Mackinnon, 2011).

The protective effects of culture and ethnicity should also be examined in future research. Epidemiological studies in the United States suggest that racial and ethnic minorities tend to have a lower lifetime risk of psychiatric disorders compared to non-Hispanic whites (Breslau et. al., 2006). This lower prevalence suggests that childhood protective factors have generalized effects on mental health (Breslau et. al., 2006). Schei, Nøvik, Thomsen, Indredavik, and Jozefiak (2015) also find that family cohesion is protective for adolescents with ADHD. Family cohesion may have varying protective effects by culture and ethnicity (Rivera et. al., 2008; Weisman, Rosales, Kymalainen, & Armesto, 2005). For example, Weisman and colleagues (2005) found that family cohesion amongst Latinos and African Americans, but not Anglo-Americans, moderates emotional distress. Future models should consider family dynamic as a protective factor against severe ADHD symptoms in diverse samples.

Future studies on ADHD-SES and ADHD-obesity should strive to utilize a diverse adult sample as was done in the current study. The scarcity of research on these topics pertaining to adult ADHD makes it difficult to form well-grounded conclusions about the current data. Further research on these matters may allow us to better understand the moderating effects of neighborhood socioeconomic status on the ADHD-obesity relation in a diverse sample of young adults and the implications of the results of the current study.

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Appendix A: Sources of Neighborhood Socioeconomic Status Variables

Domain	Variable	Table ID/ Indicator	Source of Info/ Website link
Education	Population aged 25 years and older that has completed less than 9th grade and has no diploma	S1501	Census' 2012-2016 American Community Survey 5-Year Estimates/ https://data.census.gov/cedsci/
Housing Characteristics	Population with rent or mortgage that is ≥30% of household income	DP04	Census' 2012-2016 American Community Survey 5-Year Estimates/ https://data.census.gov/cedsci/
	Population with more than 1 occupant per room	DP04	Census' 2012-2016 American Community Survey 5-Year Estimates/ https://data.census.gov/cedsci/
	Population with female head of household	S2201	Census' 2012-2016 American Community Survey 5-Year Estimates/ https://data.census.gov/cedsci/
Income/ Employment	Population receiving supplemental security income (SSI), cash public assistance, and food stamps/SNAP benefits (in the past 12 months)	S1902/ S2201	Census' 2012-2016 American Community Survey 5-Year Estimates/ https://data.census.gov/cedsci/
	Population with income in the past 12 months below the poverty level	S1701	Census' 2012-2016 American Community Survey 5-Year Estimates/ https://data.census.gov/cedsci/
	Population unemployed	S2301	Census' 2012-2016 American Community Survey 5-Year Estimates/ https://data.census.gov/cedsci/
Living Environment	Population rating their neighborhood as not safe	"Neighborhood is safe or not"	NYC Community Health Survey 2016/ https://a816-health.nyc.gov/hdi/epiquery/visualizations?PageType=ps&PopulationSource=CHS
	Population with a BMI of 30 or greater	"Overweight and obesity"	NYC Community Health Survey 2016/ https://a816-health.nyc.gov/hdi/epiquery/visualizations?PageType=ps&PopulationSource=CHS
	Population indicating they have consumed no fruits or vegetables yesterday	"Fruit/vegetable consumption"	NYC Community Health Survey 2016/ https://a816-health.nyc.gov/hdi/epiquery/visualizations?PageType=ps&PopulationSource=CHS

Note. Since the beginning of the data coding for this study, the U.S. Census Bureau and NYC government have altered their data sources. Therefore, current data tables displayed on these websites may not reflect the data previously available.