Diet, Physical Activity, and ADHD Symptoms in Middle Childhood: Shared and Unique Environmental and Genetic Influences

by

Emily Jurek

Fall 2021

Director: Kathryn Lemery-Chalfant, Ph.D.

Second Committee Member: Gianna Rea-Sandin, M.S.

Abstract

Attention-deficit hyperactivity disorder (ADHD) is a developmental disorder categorized by symptoms of inattention, impulsivity, and/or hyperactivity. The onset of ADHD symptoms begin to appear in childhood, tend to be persistent into adulthood, and are associated with adverse physical and mental health outcomes (Caye et al., 2016; Yoshimasu et al., 2018). The current study examined the effects of health-promoting behavior (i.e., diet and physical activity) on ADHD symptoms in middle childhood. The sample consisted of 670 twins (30.1% monozygotic, 36.5% same-sex dizygotic, 32.3% opposite-sex dizygotic) from the ongoing longitudinal study, the Arizona Twin Project. The twin's (51% female) diet, physical activity, and ADHD symptoms were assessed at age 8 (M = 8.45, SD = 0.69) and ADHD symptoms were assessed again at age 9 (M = 9.70, SD = 0.92). Mixed model regression analyses revealed that aspects of diet (i.e., protein, carbohydrates, and fiber) at age 8 negatively predicted ADHD symptoms at age 9. Similarly, sedentary behavior at age 8 negatively predicted ADHD symptoms at age 9, whereas moderate-to-vigorous activity at age 8 positively predicted ADHD symptoms at age 9. Univariate twin analyses revealed that certain aspects of diet (i.e., sugar, vegetable, and fruit consumption) were influenced by environmental factors whereas other aspects of diet (i.e., protein, carbohydrates, calories, fat, and fiber consumption) were influenced by both genetic and environmental factors. Both children's sedentary behavior and moderate-to-vigorous activity were influenced primarily by genetic factors, with the remaining variance being attributed to non-shared environmental factors. Additive genetic influences explained the majority of the variance in ADHD symptoms. Future research should examine bidirectional effects of activity and diet on ADHD symptoms across childhood.

Keywords: ADHD, physical activity, diet, childhood

Diet, Physical Activity and ADHD Symptoms in Middle Childhood: Shared and Unique Environmental and Genetic Influences

Attention-deficit/hyperactivity disorder (ADHD) is a widely researched, common, and heritable childhood disorder. The average age for ADHD onset and diagnosis is 6 years old (Kessler et al., 2005). ADHD is characterized by having 5 or more symptoms of inattention or 5 or more symptoms of hyperactivity/impulsivity (or both inattention and hyperactivity/impulsivity symptoms) for greater than or equal to 6 months (American Psychiatric Association, 2013). The prevalence of ADHD is 5.9-7.1% in children growing up in the U.S. (Willcutt, 2012). ADHD symptoms tend to be persistent into adulthood, causing problems for children and predicting problems across the lifespan (Caye et al., 2016). Persistent ADHD symptoms in adulthood are associated with a significantly higher risk for comorbidity with both internalizing and externalizing disorders with some evidence that it is also related to physical health, such as diet, and exercise (August et al., 1996; Yoshimasu et al., 2018). To prevent unpropitious outcomes from ADHD symptoms in childhood, it is essential to understand potential prevention methods from health-related factors, such as diet and exercise.

In early childhood, a healthier diet pattern and acute bouts of exercise are associated with better cognitive outcomes (Tandon et al., 2016). Previous research found that ADHD symptoms in early childhood predicted adverse health outcomes such as smoking, drug abuse, accidental injury, sleep, obesity, hypertension, diabetes, and suicidal behavior across development later in adulthood (Nigg, 2013). There are certain diet variables we examined that have not been examined in previous literature. Health-promoting behaviors may be the tool for people with ADHD in preventing adverse mental and physical health outcomes. In accordance with the biopsychosocial model, ADHD can be viewed as being influenced by biological, psychological,

and social factors (Salamanca, 2014). Therefore, the current study attempts to understand the genetic and environmental mechanisms that influence ADHD symptom development in middle childhood in order to promote better long-term outcomes for people with ADHD.

Physical Health and ADHD

High ADHD symptoms or an ADHD diagnosis in childhood is associated with increased physical health problems. Overall, children with more ADHD symptoms or a diagnosis scored significantly lower on health-related quality of life questionnaires than children without symptoms or a diagnosis (Klassen et al., 2004; Peasgood et al., 2016). Additionally, children with more ADHD symptoms slept significantly fewer hours a night than children without symptoms (Peasgood et al., 2016). Children diagnosed with ADHD have significantly more neurological health problems than children without a diagnosis (Pan & Bölte, 2020). There is an association between children diagnosed with ADHD and a higher risk for being overweight or obese and having asthma than children without a diagnosis (Chen et al., 2013; Flier et al., 2013). Similarly, children with more ADHD symptoms are associated with a higher risk of unintentional injury than children with fewer or no symptoms (Ruiz-Goikoetxea et al., 2018). Although more ADHD symptoms and diagnoses are related to poorer physical health outcomes in middle childhood, the extent to which health promoting behaviors influence ADHD in middle childhood is unknown. Diet and exercise patterns across childhood may prevent the development of symptoms of ADHD in children.

Diet and ADHD

Certain diet patterns are associated with ADHD symptoms in middle childhood. A recent review from Del-Ponte et al. (2019) concluded that in terms of ADHD and diet in children and adolescents ages 3-14, healthy diet patterns (consisting of fruits, vegetables, and whole grains)

protected against ADHD diagnoses and symptoms whereas, unhealthy diet patterns (consisting of saturated fats and refined sugars) were associated with increased risk of ADHD diagnoses and symptoms in a global sample from Europe and Asia using parent reports of diet quality. Children with more ADHD symptoms at age 6 had a lower quality diet at age 8, but overall diet quality at age 8 did not predict ADHD symptoms at age 10 in a community sample in the Netherlands using parent report of food frequency over the past 4 weeks (Mian et al., 2019). In addition, a community sample of 6-17-year-old children diagnosed with ADHD had a poorer diet regardless of taking ADHD medication compared to children without a diagnosis using a self-report of diet recall of 1 week (Bowling et al., 2017). Additionally, a recent review from Shareghfarid and colleagues (2020B) concluded that healthy diet patterns (consisting of fruits, vegetables, legumes, and fish) decreased the risk of ADHD symptoms in children ages 4-14, whereas junk food diet patterns (consisting of sweetened beverages and desserts) and Western diet patterns (consisting of red meat, refined grains, processed meats, and hydrogenated fat) increased the risk. This study was a community sample using diet recall and food frequency assessments ranging from 3 days to a year. Furthermore, Iranian children ages 6-13 diagnosed with ADHD consumed significantly fewer macronutrients (energy intake, fat, and carbohydrates) and micronutrients (calcium, magnesium, thiamin, riboflavin, niacin, vitamin B5, Vitamin C) than children from the same population without a diagnosis using a food frequency assessment from the past year (Shareghfarid et al., 2020A). In conclusion, poorer quality diet and lack of nutrients in middle childhood are positively related to ADHD symptoms.

Physical Activity and ADHD

Systemic exercise is another component of overall health and has a protective relationship with ADHD in middle childhood. A review from Cerrillo-Urbina and colleagues

(2015) concluded that short-term aerobic exercise was associated with fewer ADHD symptoms (inattention, hyperactivity, and impulsivity) in children and teens ages 6-18 diagnosed with ADHD. In addition, children with less sedentary behavior at age 7 significantly predicted more ADHD symptoms at age 14 using objective measures of activity from a large UK population-based sample (Brandt et al., 2020). Furthermore, moderate to vigorous physical activity was positively associated with cortical functioning in 7-12-year old children diagnosed with ADHD (Yu et al., 2019). This study used objective measures of physical activity and participants from elementary schools in Taipei. Moderate to vigorous physical activity for 5-12-year-old children with either an ADHD diagnosis or ADHD symptoms improved their cognitive, behavioral, motor, and social functioning when participating in exercise programs ranging from 1 day to 10 weeks (Pontifex et al., 2013; Smith et al., 2013; Verret et al., 2012). However, a community sample of children diagnosed with ADHD was 21% less likely to engage in physical activity than children without a diagnosis (Mercurio et al., 2021). This study used parent report to measure how many days a week their child participated in the recommended 60 min of daily exercise across one week. Less than a third of 6-18-year-old children diagnosed with ADHD completed at least 20 minutes of recommended daily physical activity compared to children without a diagnosis from a week-long parent report and population-based sample (Tandon et al., 2019). The literature suggests that children with ADHD symptoms and/or diagnoses are less likely to partake in exercise, but when they do, it seems to show health benefits. It is necessary to examine the relationship between physical activity, diet, and ADHD symptoms in a genetically informative design to better understand their genetic and environmental relationships.

Physical Activity, Diet, and ADHD

The literature suggests similar connections among ADHD symptoms, diet, and physical activity in middle childhood. A recent review from Quesada and colleagues (2018) concluded that dysregulated eating and lack of motivation to participate in physical activity were positively associated with ADHD symptoms of impulsivity and inattention in children and adolescents ages 0-17. In a Chinese community sample of 9-13-year-old children with ADHD symptoms using self-report, there were positive associations among increased participation in sedentary activity, increased eating, and decreased physical activity (Tong et al., 2016). In a second study of English-speaking children diagnosed with ADHD ages 8-16 who self-reported on diet, there was an association between more sedentary behavior and less consumption of healthy foods (healthy Mediterranean diet) (San Mauro Martín et al., 2018). In sum, the existing literature suggests that less physical activity and poorer diet are associated with more ADHD symptoms; however, more research is needed to understand genetic and environmental contributions to these variables and their associations.

The Twin Study Approach

Twin Methodology. To understand the direct environmental and genetic impact for certain traits, researchers use the twin method. The twin method compares traits in identical twins (monozygotic, MZ) that share 100% of their genetic makeup, to traits in fraternal twins (dizygotic twins, DZ) that share 50% of their genetic makeup (Mayo, 2009). To understand whether a trait is linked for primarily genetic reasons, the correlation between the trait of interest between the monozygotic cotwins has to be higher than the correlation between the trait of interest in dizygotic cotwins. However, similar correlations between monozygotic and dizygotic cotwins would indicate primarily environmental contributions. Based on this logic, we used the twin design to examine the extent to which genetic and environmental contributions explain

variance in our variables as well as the relationship between diet components and physical activity (sedentary activity or moderate to vigorous activity) with ADHD symptoms in children ages 8 and 9 in middle childhood.

Genetic and Environmental Influences on Children's ADHD. Individual differences in children's ADHD were explained primarily by genetic factors. A recent review from Faraone and Larsson (2019) concluded that ADHD was highly heritable (74%) in middle childhood (ages 7-10), across the reviewed literature. The remaining variance was attributed to the nonshared environment. In contrast, differences in diet were less influenced by genetic components.

Genetic and Environmental Influences on Children's Diet. There were high heritability estimates of diet patterns in a community sample of children ages 7-15 who reported their food frequency intake over an entire year (Li et al., 2016). 67.9% of the variance for diet was explained by genetics for boys and 89.8% for girls; with girls having a higher genetic influence of high fat intake (16.3%) and fried/fast food (89.8%) compared to boys (0% high fat intake and 35.2% fried/fast food). In addition, additive genetic effects explain a large part of the variation in traits related to appetite during childhood, the heritability estimate of eating behaviors (eating too much or too little, eating too fast) was 71%-89% in 2.5-year-old children and decreased to 44% for 9-year-old children (Dubois et al., 2013). Additionally, there was a high heritability estimate for additive genetic influences for food acceptance traits (refusing to eat and being fussy about food) in 9-year-old children ranging from 84-85% using a parent-report questionnaire of eating behaviors (Dubois et al., 2013). However, genetic factors explain 32% of the variance for healthy diet patterns and explained 27% of the variance of unhealthy diet patterns in a community sample of adults ages 19-92 (van den Berg et al., 2013). Participants completed a questionnaire describing how often they eat certain foods throughout the week. In addition, heritability

estimates remain stable for diet patterns among female adults ages 18-79 years old with additive genetic effects accounting for 41-48% of the variance in five food groups (fruit and vegetable, high alcohol, traditional English, dieting, low meat) from diet recall in a national sample (Teucher et al., 2007). Thus, although studies were scarce, diet quality was explained primarily by genetic factors in early childhood and tended to decrease into adulthood.

Genetic and Environmental Influences on Children's Activity Level. Genetic factors explain 57% of the variance for objectively measured physical in twin adults ages 17-27, with the rest of the variance being attributed to environmental factors (Gielen et al., 2014). Physical activity was assessed over two weeks from two independent populations in Western Europe. Similarly, heritability estimate of additive genetic factors of moderate-vigorous physical activity in adults ages 18-103, was 47% and 31% for sedentary behavior, with the remaining variance being explained primarily by nonshared environmental factors (Hoed et al., 2013). This study used a nationally representative cohort of adults and objective measures of physical activity. Genetic factors explain 26% of the variance of sedentary behavior from self-report whereas 56% were explained by objective measures of sedentary behavior in teenage and adult twins ages 16-71 (Schutte et al., 2020). Additionally, additive genetic factors explained 14% of the individual differences of moderate to vigorous physical activity from self-report whereas 46% in objective measures of moderate to vigorous physical activity in teenagers and adults over the course of a week (Schutte et al., 2020). The rest of the variance was attributed to nonshared environmental factors in both sedentary behaviors and moderate to vigorous physical activity. Variance estimates for sedentary behavior in 12-year-olds were accounted for by additive genetic (35% for boys and 19% for girls), shared environmental (29% for boys and 48% for girls), and nonshared environmental (36% for boys and 34% for girls) factors using self-report of sedentary behavior

frequency across a week (van der Aa et al., 2012). However, in the same study, the variation in sedentary behavior among 20-year-olds was accounted for by additive genetic (48% for boys and 34% for girls) and nonshared environmental (52% for boys and 66% for girls) factors, not shared environmental factors (van der Aa et al., 2012). There was little research examining individual differences of exercise in early and middle childhood. The current study filled in the gaps in the literature to provide a better understanding of how genetics influence physical activity patterns in middle childhood.

Genetic and Environmental Influences on Children's ADHD and Diet. There is some evidence of genetic and environmental influences on diet and ADHD symptoms in middle childhood. In a study by Li and colleagues (2020) regarding diet, ADHD symptoms, and genetic and environmental contributions, more ADHD symptoms of inattention and hyperactivity/impulsivity were positively associated with more consumption of seafood, high-fat food, high-sugar food, high-protein food, and unhealthy food but negatively associated with fruits, vegetables, and healthy food. The sample included 20-47-year-old adults from a nationwide population-based sample using self-report measures of food frequency intake over the past year. Genetic factors explained 40-44% of the covariance in inattention symptoms and 20-37% for the covariance of hyperactivity/impulsivity symptoms with unhealthy and high sugar dietary habits (Li et al., 2020). This pattern of results shows that identical twins with ADHD symptoms consume more similar amounts of sugar and unhealthy diet foods than fraternal twins with ADHD, illustrating the genetic influence. The current study considered these associations in childhood, that to the best of our knowledge had not been studied previously.

Genetic and Environmental Influences on Children's ADHD and Physical Activity. A study conducted by Rommel and colleagues (2015) examined whether physical activity in age

16-17 twins predicted ADHD symptoms in early adulthood ages 19-20 using self-report of physical activity as well as parent report of ADHD symptoms. Higher levels of physical activity at ages 16-17 predicted significantly fewer ADHD symptoms at ages 19-20, controlling for genetic and shared environmental influences shared between twin pairs, so the association is due to nonshared environmental influences (Rommel et al., 2015). To the best of our knowledge, there is no research examining the impact of genetic, shared, and non-shared environmental factors that explain the covariance among ADHD, diet quality, and physical activity. This study was the first to examine these associations.

Covariates

To better understand the relationship between children's ADHD, diet, and activity level, specific covariates were controlled for that may impact these relationships. Developmental research studies control for age, sex, race/ethnicity and socioeconomic status as they are commonly related to the outcomes of interest. An additional covariate that may be related to the relationship between ADHD, diet, and activity level is BMI (Bowling et al., 2017). Controlling for confounding variables diminishes the likelihood that the results are due to other related variables, strengthening the current study design.

Current Study

Research is needed to understand the associations among health indicators and children's ADHD, as well as the extent to which underlying genetic and environmental factors explain covariation among these variables. The current study explored the association between diet quality and physical activity level on ADHD symptoms in middle childhood. This study was the first to our knowledge to examine underlying genetic and environmental contributions to the relationships among diet quality, objective physical activity, and ADHD symptoms.

Based on the previous research, the current study aimed to examine 1) the association between ADHD symptoms, diet, and physical activity in middle childhood and 2) the extent to which additive genetics, shared environmental, and non-shared environmental influences explained the variance in ADHD symptoms, diet, and physical activity in middle childhood and their relationships. Since there was a positive association between ADHD symptoms, lower quality diet, and less sedentary behavior in the literature (Mercurio et al., 2021; Mian et al., 2019; Tandon et al., 2019), we hypothesized that 1) an unhealthy diet pattern (high sugar) would be positively related to ADHD symptoms in middle childhood, 2) that sedentary behavior would be negatively related to ADHD symptoms and activity level would be positively related to ADHD symptoms in middle childhood, and 3) ADHD symptoms would be concurrently and longitudinally related. Also, we hypothesized that 1) more unhealthy diet patterns at age 8 would be positively associated with more ADHD symptoms at age 9 and would be related for primarily non-shared environmental reasons, 2) more moderate-to-vigorous activity at age 8 would be positively associated with more ADHD symptoms at age 9 and would be related for primarily genetic reasons, and 3) more unhealthy diet patterns would be positively associated with more ADHD symptoms at age 9 and would be primarily related for non-shared environmental reasons and moderate-to-vigorous activity at age 8 would be positively associated with more ADHD symptoms at age 9 and would be related for primarily genetic reasons.

Methods

Participants

The current study consisted of 677 children (51% female) assessed at age 8 years (M = 8.45, SD = .69), and 651 children assessed at age 9 years (M = 9.70, SD = .92). These children were twins (30.1 % monozygotic twins, 36.5% same-sex dizygotic twins, 32.3% opposite-sex dizygotic twins) from an ongoing longitudinal twin study, the Arizona Twin Project (Lemery-

Chalfant et al., 2019). The sample was ethnically diverse at age 8 (approximately 23.7 % Hispanic/Latinx, 58.5% non-Hispanic white/Caucasian, and 0.9% other) and age 9 (approximately 17.1% Hispanic/Latinx, 42.2% non-Hispanic white/Caucasian, 0.6% other, and 28% unknown). Based on the income-to-needs ratio, using household income, family size, and poverty thresholds, families were economically diverse at age 8 (53% were middle to upper class, 23% were at or near the poverty line, 16% were lower middle class, 8% lived below the poverty line) and at age 9 (53% were middle to upper class, 23% were at or near the poverty line, 16% were lower middle class, 8% lived below the poverty line). Lastly, primary caregiver's education levels were diverse (0.6% % less than high school, 8.7% high school degree, 25.3% some college, 34.5% college degrees, 3.4% 2 or more years of graduate school, and 21.8% graduate or professional degree). See Table 1 for participant demographics.

Procedures

Primary caregivers were invited to complete a phone interview when the twins were 12 months old, which included a zygosity questionnaire to determine the zygosity of the twins. Families were contacted again to participate in a twin study examining children's sleep and health when the children were 8 years old. The assessment at 8 years included an online survey, two in-home visits, an interview with each twin, and three days to a week of daily assessment including diet recall and actigraphy. The online survey included the Health and Behavior Questionnaire (HBQ) that includes questions about ADHD symptoms which was completed by the primary caregiver, secondary caregiver, and teacher prior to the first home visit. After receiving written consent from the primary caregiver and verbal assent from each twin, the first in-home assessment was conducted. During the home visit, two trained researchers administered health assessments to each twin separately. Height and weight were measured at the home visit in order to assess body mass index.

Additionally, primary caregivers were asked to complete a daily diary of the food intake of each of the twins for three consecutive days. Instructions on how to complete the daily dairy were covered in the first home visit. Daily dairies could be completed on paper or an online survey depending on the preference of the primary caregiver. Primary caregivers were trained on how to appropriately fill out the food dairies for each twin by going through an entire diary entry step-by-step.

Lastly, the research assistants trained the primary caregiver on the procedure with the actigraphy watches that measured activity level and sleep during the first home visit and picked up the watches at least a week later. Twins were asked to wear the watches on their non-dominant wrists for 24 hours over six days and seven nights except in water (bathing, swimming, and showering). Most of the twins began wearing the watches on a Sunday and removed the watches the following Sunday. During this week, research assistants checked in with the families about the watches and sent text reminders to fill out daily diaries throughout the week.

Families were contacted again when the children were 9 years old to participate in the next wave of the study. This next wave was conducted to follow up on the twin's health and sleep behaviors. The 9-year assessment consisted of the second home visit and online survey where the primary caregiver again completed the HBQ including questions about ADHD symptoms prior to the home visit. Also, the twins participated in the Berkeley Puppet Interview (BPI) including questions about ADHD symptoms which was administered by a trained researcher during the home visit.

Data collected at the 8-year assessment was used to assess the twin's diet quality, physical activity level, and ADHD symptoms from both caregivers and teacher reports. Data collected at the 9-year assessment was used to assess the twin's ADHD symptoms from self-report (BPI) and primary caregiver measures (HBQ).

Measures

Demographics. Primary and secondary caregivers completed demographic information on their children's age, sex, race/ethnicity, and socio-economic status (SES) during both the 8- and 9-year assessments. SES was assessed using a composite of income-to-needs ratio and primary and secondary caregiver education completed by the primary caregiver. The income-to-needs ratio was calculated for each family based on the number of individuals in the household and federal poverty standards published for 2017 (U.S. Department of Health and Human Services, 2017).

Zygosity. The zygosity of the twins was assessed using The Zygosity Questionnaire for Young Twins completed by the primary caregiver when the twins were 12 months old (Goldsmith, 1991). The zygosity questionnaire consisted of 32 items on twin gestation, the birth of the twins, physical similarities over time, and observable differences that demonstrate about 93% accuracy of zygosity when compared to genotyping (Forget-Dubois et al., 2003). Zygosity was confirmed through photos taken during the home visits at the age 8 and 9-year assessments.

Diet at age 8. Diet was assessed by primary caregivers using a 24-hour food recall diary at the age 8 assessment. The diet diaries included reports of food, beverage, time of day, amount, type, and additional comments (calories, etc.). The diet diaries were coded using https://fdc.nal.usda.gov/ to determine calories, protein, and carbohydrates. If the food item could not be located in the US Department of Agriculture database, then research assistants obtained diet information directly from the brand's website or a fitness website (e.g., MyFitnessPal). Diet composites categorized the food into eight major categories, total fat, protein, carbohydrates, sugar, fiber, calories, fruits, and vegetables. For the purpose of this study, diet was categorized as either healthy (total fiber, fruits, and vegetables), unhealthy (total sugar), and the remaining

variables were included by not categorized (calories, protein, fat, and carbohydrates). The quality of the food diaries was also coded on a 1-3 Likert scale, with 1 indicating very little to no detail and 3 indicating a great deal of detail.

Physical Activity at age 8. Physical Activity was assessed at age 8 using Micro MotionLogger Watches, which are wrist-based accelerometers designed to distinguish between periods of sleep and activity (Ambulatory Monitoring, Inc, Ardsley, NY USA). The Sadeh algorithm in Action W-2 software version 2.7.1 (Ambulatory Monitoring) program was used to analyze the data (Sadeh, Sharkey, & Carskadon, 1994). Specifically, we used the proportional-integrating mode (PIM) which measured the total amount of movement in 60-second epochs. We used specific cutoff points to categorize 4 levels of activity; sedentary (1-100 counts per 60s epochs), light (101- 2292 counts per 60s epochs), moderate (2293-4008 counts per 60s epochs), and vigorous (4009 counts per 60s epochs) (Evenson et al., 2008). For the purpose of this study, we used sedentary and moderate-to-vigorous activity composites.

ADHD symptoms at age 8 and 9. ADHD symptoms were assessed by both primary and secondary caregiver and teacher at the 8-year assessment and by the primary caregiver and self at the 9-year assessment using the HBQ at ages 8 and 9 and the BPI for self-report at age 9 (Armstrong & Goldstein, 2003; Measelle et al., 1998). The HBQ ADHD composite consisted of 15 items, 9 relating to impulsivity symptoms and 6 items relating to inattention symptoms which were completed by both caregivers and teacher. The items were measured using a scale of 0-2, where 0 = “Never or not true”, 1 = “sometimes or somewhat true”, and 2 = “often or very true.” Parents reported how often these behaviors occurred during the past 6 months where teachers reported behavior frequency over the past 3 months. The BPI consisted of 12 items of ADHD symptoms, 6 relating to symptoms of impulsivity, and 6 relating to intention symptoms (Measelle et al., 1998). The twins were asked to choose which statement applies to them more

(e.g., “Your teacher doesn’t tell you to pay attention. -OR- Your teacher is always telling you to pay attention.”), then based on that choice the twins were asked to explain if that choice “sort of describes you” or “really describes you.”.

Covariates. Potential covariates included child age, sex, SES, race/ethnicity, and body mass index (BMI).

Statistical Approach.

Descriptive statistics were collected for each variable to determine if the variables were normally distributed. We ran descriptive statistics (mean, standard deviation, minimum, maximum, skewness, and kurtosis) for all study variables. This included five variables for ADHD symptoms, (primary caregiver-report at age 8, secondary caregiver report at age 8, teacher report at age 8, primary caregiver-report at age 9, and self-report at age 9), eight variables for diet quality (total fat, carbohydrates, sugar, fiber, fruits, vegetables, protein, and calories at age 8), two variables of physical activity (sedentary behavior and moderate-to-vigorous physical activity at age 8), and potential covariates (age at 8 and 9, SES at age 8 and 9, sex and race/ethnicity age 8, and BMI at age 8). Fat, carbohydrates, and vegetables were skewed because of outliers, some children consumed high amounts of these diet factors. Therefore, these variables were winsorized to three standard deviations from the mean. The fiber variable was extremely skewed, so a natural log transformation was performed on this variable. Child sex, age, race/ethnicity, BMI, and family SES were assessed as potential covariates. Child sex was dichotomized (female = 1, male = 0). Race/ethnicity was also dichotomized (not non-Hispanic white = 0, non-Hispanic white = 1).

Correlations were run in SPSS between ADHD symptoms (primary caregiver-report at age 8, secondary caregiver report at age 8, teacher report at age 8, primary caregiver-report at

age 9, and self-report at age 9), diet quality (total fat, carbohydrates, sugar, fiber, fruits, vegetables, protein, and calories at age 8), physical activity (sedentary behavior and moderate-to-vigorous physical activity at age 8), and potential covariates (age assessed at 8 and 9, SES assessed at age 8 and 9, sex and race/ethnicity assessed at age 8, and BMI assessed at age 8). Additionally, multi-level regression was run to examine 1) the impact diet and/or physical activity has on ADHD symptoms, controlling for covariates, and 2) whether certain aspects of diet and/or physical activity predict ADHD symptoms at age 9, controlling for ADHD symptoms at age 8 and potential covariates.

To further explore the genetic contributions to these variables, univariate models using OpenMx in R (Neale et al., 2016) were fit to determine the additive genetic (A), shared (common) environment (C), and non-shared (unique) environment (E; also includes measurement error) contributions to variance in ADHD symptoms, diet, and physical activity. In the ACE model, A is correlated at 1.00 for monozygotic twins (MZ; share 100% of their genes) and 0.50 for dizygotic twins (DZ; share half of their segregating genes), whereas C is correlated at 1.00 for both MZ and DZ twins because cotwins are growing up together in the same home. E was uncorrelated because it comprises attributes of the environment that contribute to differences between cotwins as well as measurement error.

Results

Preliminary Analyses

Means and standard deviations are reported in Table 2. Fiber, fat, vegetables, and carbohydrates were positively skewed and kurtotic. Fat, vegetables, and carbohydrates were winsorized to three SDs, and a log transformation was applied to fiber to approach normality.

Table 3 gives the zero-order correlations with all study variables. All three measures of ADHD symptoms at age 8 were positively correlated with both measures of ADHD symptoms at

age 9. Moderate to vigorous activity was negatively correlated with sedentary behavior. Moderate to vigorous activity was positively correlated with both caregivers' reports of ADHD symptoms at ages 8 and primary-caregiver reports of ADHD symptoms at age 9. There was no significant correlation between sedentary behavior and ADHD symptoms. Diet composites of total fiber, sugar, fruit, and calories were negatively correlated with sedentary behavior. However, there were no significant correlations between diet and moderate to vigorous activity. Diet composites of fruits, carbohydrates, and calories were negatively correlated with primary caregiver-reported ADHD symptoms at age 8. Similarly, fruit consumption was negatively correlated with teacher-reported ADHD symptoms at age 8. Lastly, protein consumption was negatively correlated with self-reported ADHD symptoms at age 9. The correlations between diet and physical activity with ADHD symptoms were too low to conduct bivariate analyses so only univariate analyses were run.

Phenotypic Associations Among Diet, Activity, and ADHD

Mixed model regression analyses were run to examine the main effects of physical activity on ADHD symptoms. Child sex, age, BMI, SES, and race/ethnicity were included in the analyses as covariates. Moderate to vigorous physical activity positively predicted symptoms of ADHD for all measures of ADHD symptoms (primary caregiver-reported at age 8 and 9, secondary caregiver-reported at age 8, and teacher-reported at age 8) except for self-reported ADHD symptoms at age 9. In addition, sedentary behavior negatively predicted primary caregiver reported ADHD symptoms at age 8. Male sex was associated with more ADHD symptoms for all reports of ADHD symptoms except self-report symptoms at age 9. SES was negatively associated with primary-caregiver and teacher-report ADHD symptoms at age 8. BMI was positively associated with primary caregiver reported ADHD symptoms at age 8. Similar analyses were run to examine the main effects of diet components on ADHD symptoms. Child

sex, age, BMI, SES, and race/ethnicity were included in the analysis as covariates. More protein consumption predicted fewer primary caregiver reported ADHD symptoms at ages 8 and 9 and self-report symptoms at age 9.

Further analyses were run to examine if physical activity and diet were predictive of ADHD symptoms at age 9, controlling for ADHD symptoms at age 8. After controlling for potential covariates and previous ADHD symptoms at age 8 (primary and secondary caregiver-reported symptoms and teacher-reported symptoms), carbohydrate and fiber consumption negatively predicted primary caregiver reported ADHD symptoms at age 9.

Genetic and Environmental Influences on Diet, Activity, and ADHD

Table 6 provides fit statistics and parameter estimates of the full and reduced univariate ACE models for diet quality (i.e., total fat, carbohydrates, sugar, fiber, fruits, vegetables, protein, and calories at age 8), and physical activity (i.e., sedentary behavior and moderate-to-vigorous physical activity at age 8) and ADHD symptoms (i.e., primary caregiver-report at age 8, secondary caregiver report at age 8, teacher report at age 8, primary caregiver-report at age 9, and self-report at age 9).

The full ACE model was the best fit for protein, fat, carbohydrates, and calories (refer to Table 6 for fit statistics). However, the reduced CE model fit best for sugar, fruit, and vegetables. The majority of the variance was attributed to shared environmental factors for protein (62%), fat (64%), carbohydrates (69%), and calories (97%), the rest was attributed to genetic factors (29% for protein, 21% for fat, 21% carbohydrates, and 3% for calories) and nonshared environmental factors (9% for protein, 15% for fat, and 1% for carbohydrates). However, for fiber, 58% of the variance was attributed to genetic factors, 22% was attributed to shared environmental factors, and the remaining 2% was attributed to nonshared environmental factors. For sugar, fruit, and vegetables, most of the variance was attributed to shared environmental influences (84% for

sugar, 80% for fruit, and 87% for vegetables) and the remaining variance was attributed to nonshared environmental influences (16% for sugar, 20% for fruit, and 13% for vegetables).

The reduced AE model fit best for sedentary behavior and moderate-to-vigorous activity (refer to table 6 for fit statistics). For sedentary behavior, 64% of the variance is attributed to genetic factors, and the remaining 36% is attributed to shared environmental influences. However, moderate-to-vigorous activity had a stronger genetic influence with 78% of the variance being attributed to genetic factors and the remaining 22% is explained by non-shared environmental influences.

The reduced AE model fit best for primary caregiver-reported ADHD symptoms at ages 8 and 9, teacher-reported symptoms at age 8, and self-reported symptoms at age 9 (refer to Table 6 for fit statistics). The AE model indicates that genetic and nonshared environmental influences account for the variance in these variables. However, the reduced CE model fit best for secondary caregiver-reported symptoms at age 8. The CE model indicates that shared and nonshared environmental influences account for the variance in this variable, with no genetic influence. For primary caregiver reported symptoms, variance attributable to additive genetic factors increased from 87% at age 8 to 94% at age 9 and the variance attributed to unique environmental influences decreased from 13% and age 8 to 6% at age 9. For teacher-reported symptoms at age 8, the additive genetic influence was also high (78%). For self-reported symptoms at age 9, variance attributed to nonshared environmental influences was higher (69%) than additive genetic influences (31%). For secondary caregiver-reported symptoms at age 8, 65% of the variance was attributed to shared environmental influences and the remaining 35% was explained by nonshared environmental influences.

Discussion

The current study examined if aspects of diet and physical activity were associated with more ADHD symptoms in middle childhood. We also examined the genetic and environmental contributions to childhood diet, physical activity, and ADHD. The hypothesis that an unhealthy diet (high sugar) was positively associated with ADHD symptoms was partially supported, as aspects of a child's diet (fruit, carbohydrates, protein, and calories) were negatively associated with ADHD symptoms. Additionally, protein consumption negatively predicted ADHD symptoms at age 8 and 9, however, when controlling for ADHD symptoms at age 8, carbohydrate and fiber consumption negatively predicted ADHD symptoms at age 9. Consistent with our hypothesis, sedentary behavior was negatively associated with ADHD symptoms at age 8. Moderate-to-vigorous activity was positively associated with ADHD symptoms at age 8 and 9 and more moderate-to-vigorous activity at age 8 predicted more symptoms of ADHD at age 9, but not when controlling for ADHD symptoms at age 8. We hypothesized that high sugar consumption would be related to ADHD symptoms for primarily nonshared environmental reasons and more moderate-to-vigorous activity would be related to ADHD symptoms for primarily genetic reasons in middle childhood. Bivariate models were not conducted due to low phenotypic correlations between diet and ADHD, and activity level and ADHD. However, the univariate models showed that carbohydrates, protein, fiber, fat, and calories were heritable whereas fruit, vegetables, and sugar were mostly influenced by environmental factors.

Diet and ADHD symptoms

Although an association between aspects of a healthy or unhealthy diet pattern and ADHD symptoms was not found, specific aspects of the overall diet (more protein, carbohydrates, and fiber consumption) predicted fewer ADHD symptoms. This is evidence that there may be a relationship between specific macronutrients and fewer ADHD symptoms in middle childhood. A previous study from Zhou et al. (2016), observed a significant negative

relationship between protein (specifically white-fish meat) and risk of an ADHD diagnosis in 6-14-year-old children from China. In addition, another study observed an association between less carbohydrate and protein consumption and improvement in ADHD symptoms for 6-9-year-old children diagnosed with ADHD (Abd El Baaki et al., 2021).

Healthy diet patterns have previously been shown to decrease ADHD symptoms in middle childhood, the results of our study suggest that energy-rich macronutrients may have similar effects (Del-Ponte et al., 2019; Shareghfarid et al. 2020B). Carbohydrates, protein, and unsaturated fats are the main macronutrients responsible for providing our bodies with energy (National Research Council, 1989). In a study by Mueller et al. (2010), prepubertal boys with an ADHD diagnosis had a significantly higher resting energy expenditure than boys with a diagnosis. The results of this study suggest that ADHD boys may need more energy intake because they are exerting more energy throughout the day, even when resting. These findings are consistent with our results as it suggests that children with ADHD symptoms need to consume foods that give them steady energy.

Physical activity and ADHD

The hypothesis for moderate to vigorous activity and ADHD symptoms was fully supported. More moderate-to-vigorous activity was associated with more ADHD symptoms and more sedentary behavior was associated with fewer ADHD symptoms in middle childhood. These results may be evidence that ADHD children are more physically active. Similar results were found in a study that found that sedentary behavior at age 7 negatively predicted ADHD symptoms at age 14, moderate activity at age 7 positively predicted ADHD symptoms at age 14, and less sedentary activity at age 7 predicted an ADHD diagnosis at age 14 (Brandt et al., 2020). One reason for this outcome may come from using a wrist-based accelerometer to measure physical activity in the sample. In a study conducted by Dane, Schachar, & Tannock (2000),

children with an ADHD diagnosis were more active throughout the school day than children without a diagnosis. Children with ADHD may fidget more and the actigraphy watches may interpret the movement as activity even when the child is not exerting a lot of energy.

Twin models for diet, physical activity, and ADHD symptoms

Twin models of diet. The additive genetic shared environmental, and non-shared environmental influences for the diet components were not consistent across every diet variable. Shared environmental influences explained the majority of variance for vegetables, fruit, and sugar with the remaining being attributed to non-shared environmental influences. When we think about the measure, we used to assess diet, we assessed children in middle childhood, where caregivers were responsible for choosing foods that households will consume. So, our results suggest that parents play a large role in integrating fruit, vegetables, and sugar into their children diet

The remaining diet components (protein, fat, carbohydrates, calories, and fiber) had variance explained by genetic, shared-environmental, and non-shared environmental influences. Similar to vegetables, fruit, and sugar, shared environmental influences explained the majority of the variance for protein, fat, carbohydrates, and calories, however, the rest of the variance was attributed to additive genetic followed by non-shared environmental influences. For fiber, additive genetic influences explained the majority of the variance with the rest being split between shared and non-shared environmental influences. Our bodies rely on fat, carbs, and protein as our main sources of energy. Genetic influences could reflect this biological need for these nutrients, with MZ twins consuming similar amounts compared to DZ twins. In conclusion, we found evidence of heritable influences on aspects of children's diets. Previous genetically informed literature has focused on adults and found moderate heritability estimates for overall diet patterns (Li et al., 2016 & van den Berg et al., 2013). The current study's finding of

heritability of diet in middle childhood is important as it is the first time genetic and environmental influences of diet recall have been examined, to the best of our knowledge.

ACE models of physical activity. We found consistent genetic and environmental influences on both sedentary behavior and moderate-to-vigorous activity. Additive genetic factors explained the majority of variance for both measures of activity level with the remaining being attributed to non-shared environmental influences, which best fit in the AE model. Previous literature has found moderate heritability estimates for activity level in adults (Brandt et al., 2020). Our study found higher heritability estimates in middle childhood. However, objective measures of physical activity are shown to be more heritable than self-reported measures of activity in adults, potentially explaining why our estimates were higher (Schutte et al., 2020).

ACE models of ADHD. Consistent with previous heritability estimates from Faraone & Larsson, 2019, additive genetic influences explain the majority of variance for most measures of ADHD symptoms in childhood, with the rest of the variance being attributed to unique environmental influences. However, there were differences in heritability estimates for secondary caregiver and self-reported measures of ADHD symptoms. Most of the variance for secondary caregiver reported ADHD symptoms was attributed to shared environmental influences with the remaining variance attributed to nonshared environmental influences. Nonshared environmental influences explained most of the variance for self-reported ADHD symptoms with the rest being attributed to additive genetic influences.

In a study by Bied, Biederman, and Faraone, (2017), parents and teacher reports of ADHD symptoms were “statistically indistinguishable” from each other in terms of accuracy of ADHD diagnosis. This may explain why primary caregiver and teacher-reported measures of ADHD symptoms have similar genetic and environmental influences. Previous literature also indicates that mothers and fathers differ in reports of ADHD symptoms for their children. There

has been an association between mothers reporting more ADHD symptoms for children than fathers did (Caye, Machado, & Rohde, 2017; Langberg et al., 2010; Sollie, Larsson, & Mørch, 2013). Primary and secondary caregiver heritability estimates of ADHD symptoms varied in our results, so caregivers may be picking up on different characteristics of their children's behavior. Lastly, previous literature has observed that self-reported ADHD symptoms were not correlated with objective measures of ADHD symptoms for children and adolescence (Du Rietz et al., 2016). Parents reported significantly more ADHD symptoms than children reported about themselves which may help explain the result of the current study. Additionally, children are 9 years old when self-reporting their ADHD symptoms, so they may be thinking more concretely (how they are behaving right now) instead of abstractly on their behaviors (average behavior across months).

Because the phenotypic correlations among diet, physical activity, and ADHD were low, we did not examine genetic and environmental underpinnings of these associations. Our study examined different genetic and environmental influences for each diet variable. This suggests that there are distinct genetic and environmental influences on certain aspects of diet and future research should explore this relationship and find potential mechanisms to explain these differences.

Strengths and Limitations

The current study has several strengths. To the best of our knowledge, this study was the first to examine diet, physical activity, and ADHD symptoms in middle childhood. Diet was assessed using a 24-hour recall diet diary controlling for quality of the diet reports, eliminating bias. Additionally, caregivers are the ones buying food and preparing the meals for their children, so they would be the best ones to report on their children diet. Physical activity was measured objectively through accelerometer devices, allowing a more precise measurement than

self-report measures. ADHD symptoms were reported by multiple people, including their caregivers, teacher, and self. We used a large representative sample of twins, allowing us to examine genetic and environmental influences on diet, physical activity, and ADHD symptoms in middle childhood. With a sample of twins were we able to assess genetic and environmental influences on diet, physical activity, and ADHD symptoms in middle childhood.

However, the current study has several limitations. The sample was assessed one year apart, which may have not been enough time to examine a difference in ADHD symptoms. In addition, this study measures ADHD symptoms instead of ADHD diagnoses in middle childhood, which may result in stronger effects. Since this study measured ADHD symptoms, we did not control for stimulant use, which may affect the measures of activity and diet. Lastly, the longitudinal design of the study does not allow for causal associations between variables.

Future Directions

Our study suggests many avenues for future directions. First, our study examined diet composites of major food groups. Future research should look at diet composites that examine specific nutrients in food, to see if it is associated ADHD symptoms in middle childhood. Additionally, future research should examine ratios of diet composites, for example, protein to carb ratios to understand how it could influence ADHD symptoms in middle childhood.

Our study examined the association between wrist based activity and ADHD symptoms in middle childhood. Previous research has found an association between children diagnosed with ADHD and participation in less exercise (Tandon et al., 2019). Future research should examine energy expending exercise (e.g. sports) to examine if it impacts ADHD symptoms in middle childhood. Similarly, there may be bidirectional effects of activity level on ADHD symptoms, so future research should examine whether ADHD symptoms influence physical activity.

Implications

Our study suggests many implications for children with ADHD. This study is in-line with the biopsychosocial model that theorizes that ADHD is influenced by multiple domains (Salamanca, 2014), such as diet and exercise. Previous research has found effectiveness of reducing behavioral and internalizing problem of children (ages 4-12) with ADHD symptoms from behavioral parent training, however did not help alleviate ADHD symptoms (Van Den Hoofdakker, 2007) This study suggests that alleviating ADHD symptoms requires intervening on various lifestyle factors, rather than just prescribing children stimulant medication. Lastly, this study also suggests that providing information for parents about diet and exercise could be an effective target for interventions aiming to reduce children's ADHD symptoms.

Conclusion

The current study examined the effects of diet and exercise on ADHD symptoms in middle childhood. Some aspects of diet (protein, carbohydrates, and fiber) and more sedentary behavior predicted less ADHD symptoms in middle childhood. Whereas moderate to vigorous activity predicted less ADHD symptoms. Some aspects of diet (fiber, carbohydrates, protein, fat, and calories), activity (sedentary and moderate to vigorous activity), and ADHD symptoms were primarily influenced by genetic factors and some environmental influence whereas other aspects of diet (sugar, vegetables, and fruit) were influenced by solely environmental factors. Understanding ways to help alleviate ADHD symptoms in children can help them long-term.

References

Abd El Baaki, O. M., Abd El Hamid, E. R., Zaki, S. T., Alwakkad, A. S. E. D., Sabry, R. N., & Elsheikh, E. M. (2021). Diet modification impact on ADHD outcome. *Bulletin of the National Research Centre*, *45*(1), 1-8.

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). https://doi.org/10.1176/appi.books.9780890425596

Armstrong, J. M., & Goldstein, L. H. (2003). MacArthur Working Group on Outcome Assessment, *Manual for the MacArthur Health and Behavior Questionnaire (HBQ 1.0)*. MacArthur Foundation Research Network on Psychopathology and Development, University of Pittsburgh.

August, G. J., Realmuto, G. M., MacDonald, A. W., Nugent, S. M., & Crosby, R. (1996). Prevalence of ADHD and comorbid disorders among elementary school children screened for disruptive behavior. *Journal of Abnormal Child Psychology*, *24*(5), 571-595. https://doi.org/10.1007/BF01670101

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

Bied, A., Biederman, J., & Faraone, S. (2017). Parent-based diagnosis of ADHD is as accurate as a teacher-based diagnosis of ADHD. *Postgraduate Medicine*, *129*(3), 375-381.

Bowling, A., Davison, K., Haneuse, S., Beardslee, W., & Miller, D. P. (2017). ADHD medication, dietary patterns, physical activity, and BMI in children: a longitudinal analysis of the ECLS-K study. *Obesity*, *25*(10), 1802-1808. https://doi.org/10.1002/oby.21949

Breen, F. M., Plomin, R., & Wardle, J. (2006). Heritability of food preferences in young children. *Physiology & Behavior*, *88*(4-5), 443-447.

Brandt, V., Patalay, P., & Kerner auch Koerner, J. (2020). Predicting ADHD symptoms and diagnosis at age 14 from objective activity levels at age 7 in a large UK cohort. *European Child & Adolescent Psychiatry*, 1-8.

Caye, A., Machado, J. D., & Rohde, L. A. (2017). Evaluating parental disagreement in ADHD diagnosis: Can we rely on a single report from home?. *Journal of Attention Disorders*, *21*(7), 561-566.

Caye, A., Swanson, J., Thapar, A., Sibley, M., Arseneault, L., Hechtman, L., Arnold, L. E., Niclasen, J., Moffitt, T., & Rohde, L. A. (2016). Life span studies of ADHD-conceptual challenges and predictors of persistence and outcome. *Current Psychiatry Reports*, *18*(12), 111. https://doi.org/10.1007/s11920-016-0750-x

Cerrillo-Urbina, A. J., García-Hermoso, A., Sánchez-López, M., Pardo-Guijarro, M. J., Santos Gómez, J. L., & Martínez-Vizcaíno, V. (2015). The effects of physical exercise in children with attention deficit hyperactivity disorder: A systematic review and meta-analysis of randomized control trials. *Child: Care, Health and Development*, *41*(6), 779-788. https://doi.org/10.1111/cch.12255

Chen, M. H., Su, T. P., Chen, Y. S., Hsu, J. W., Huang, K. L., Chang, W. H., ... & Bai, Y. M. (2013). Asthma and attention-deficit/hyperactivity disorder: a nationwide population-based prospective cohort study. *Journal of Child Psychology and Psychiatry*, *54*(11), 1208-1214. https://doi.org/10.1111/jcpp.12087

Cornelius, C., Fedewa, A. L., & Ahn, S. (2017). The effect of physical activity on children with ADHD: a quantitative review of the literature. *Journal of Applied School Psychology*, *33*(2), 136-170.

Dane, A. V., Schachar, R. J., & Tannock, R. (2000). Does actigraphy differentiate ADHD subtypes in a clinical research setting?. *Journal of the American Academy of Child & Adolescent Psychiatry*, *39*(6), 752-760.

Del-Ponte, B., Quinte, G. C., Cruz, S., Grellert, M., & Santos, I. S. (2019). Dietary patterns and attention deficit/hyperactivity disorder (ADHD): a systematic review and meta-analysis. *Journal of Affective Disorders*, *252*, 160-173. https://doi.org/10.1016/j.jad.2019.04.061

Dubois, L., Diasparra, M., Bédard, B., Kaprio, J., Fontaine-Bisson, B., Tremblay, R., ... & Pérusse, D. (2013). Genetic and environmental influences on eating behaviors in 2.5-and 9-year-old children: a longitudinal twin study. *International Journal of Behavioral Nutrition and Physical Activity*, *10*(1), 1-12. https://doi.org/10.1186/1479-5868-10-134

Du Rietz, E., Cheung, C. H., McLoughlin, G., Brandeis, D., Banaschewski, T., Asherson, P., & Kuntsi, J. (2016). Self-report of ADHD shows limited agreement with objective markers of persistence and remittance. *Journal of Psychiatric Research*, *82*, 91-99.

Evenson, K. R., Catellier, D. J., Gill, K., Ondrak, K. S., & McMurray, R. G. (2008). Calibration of two objective measures of physical activity for children. *Journal of Sports Sciences*, *26*(14), 1557-1565.

Faraone, S. V., & Larsson, H. (2019). Genetics of attention deficit hyperactivity disorder. *Molecular Psychiatry*, *24*(4), 562-575. https://doi.org/10.1038/s41380-018-0070-0

Fliers, E. A., Buitelaar, J. K., Maras, A., Bul, K., Höhle, E., Faraone, S. V., ... & Rommelse, N. N. (2013). ADHD is a risk factor for overweight and obesity in children. *Journal of*

Developmental and Behavioral Pediatrics: JDBP, *34*(8). doi:10.1097/DBP.0b013e3182a50a67

Flory, K., Molina, B. S., Pelham, Jr, W. E., Gnagy, E., & Smith, B. (2006). Childhood ADHD predicts risky sexual behavior in young adulthood. *Journal of Clinical Child and Adolescent Psychology*, *35*(4), 571-577. https://doi.org/10.1207/s15374424jccp3504_8

Forget-Dubois, N., Perusse, D., Tuerecki G, Girard, A., Bilette J.M., Rouleau, G., Boivin, M., Malo, J., and Tremblay, R.E. (2003). Diagnosing zygosity in infant twins: Physical similarity, genotryping and chorionicity. *Twin Research*. 6(6), 479-485.

Gielen, M., Westerterp-Plantenga, M. S., Bouwman, F. G., Joosen, A. M. C. P., Vlietinck, R., Derom, C., ... & Westerterp, K. R. (2014). Heritability and genetic etiology of habitual physical activity: a twin study with objective measures. *Genes & Nutrition*, *9*(4), 415.

Goldsmith, H. H. (1991). A zygosity questionnaire for young twins: A research note. *Behavior Genetics*, *21*(3), 257-269.

Hoed, M. D., Brage, S., Zhao, J. H., Westgate, K., Nessa, A., Ekelund, U., ... & Loos, R. J. (2013). Heritability of objectively assessed daily physical activity and sedentary behavior. *The American Journal of Clinical Nutrition*, 98(5), 1317-1325. https://doi.org/10.3945/ajcn.113.069849

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 593-602. doi:10.1001/archpsyc.62.6.593

Klassen, A. F., Miller, A., & Fine, S. (2004). Health-related quality of life in children and adolescents who have a diagnosis of attention-deficit/hyperactivity disorder. *Pediatrics, 114*(5), e541-e547. https://doi.org/10.1542/peds.2004-0844

Langberg, J. M., Epstein, J. N., Simon, J. O., Loren, R. E., Arnold, L. E., Hechtman, L., ... & Wigal, T. (2010). Parent agreement on ratings of children's attention deficit/hyperactivity disorder and broadband externalizing behaviors. *Journal of Emotional and Behavioral Disorders*, *18*(1), 41-50.

Lemery-Chalfant, K., Oro, V., Rea-Sandin, G., Miadich, S., Lecarie, E., Clifford, S., ... & Davis, M. C. (2019). Arizona Twin Project: Specificity in risk and resilience for developmental psychopathology and health. *Twin Research and Human Genetics*, *22*(6), 681-685. https://doi.org/10.1017/thg.2019.113

Li, J., Liu, H., Beaty, T. H., Chen, H., Caballero, B., & Wang, Y. (2016). Heritability of children's dietary intakes: A population-based twin study in China. *Twin Research and Human Genetics*, *19*(5), 472-484. doi:10.1017/thg.2016.61

Li, L., Taylor, M. J., Bälter, K., Kuja-Halkola, R., Chen, Q., Hegvik, T. A., ... & Larsson, H. (2020). Attention-deficit/hyperactivity disorder symptoms and dietary habits in adulthood: A large population-based twin study in Sweden. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, *183*(8), 475-485. https://doi.org/10.1002/ajmg.b.32825

Mayo, O. (2009). Early research on human genetics using the twin method: who really invented the method?. *Twin Research and Human Genetics*, *12*(3), 237-245. https://doi.org/10.1375/twin.12.3.237

Mian, A., Jansen, P. W., Nguyen, A. N., Bowling, A., Renders, C. M., & Voortman, T. (2019). Children's Attention-Deficit/Hyperactivity Disorder symptoms predict lower diet quality but not vice versa: results from bidirectional analyses in a population-based cohort. *The Journal of Nutrition*, 149(4), 642-648. https://doi.org/10.1093/jn/nxy273

Measelle, J.R., Ablow, J.C., Cowan, P.A., & Cowan, C.P. (1998). Assessing young children's views of their academic, social, and emotional lives; An evaluation of the self-perception scales of the Berkeley Puppet Interview. *Child Development, 69(6),* 1556-1576. https://doi.org/10.2307/1132132

Mercurio, L. Y., Amanullah, S., Gill, N., & Gjelsvik, A. (2021). Children with ADHD engage in less physical activity. *Journal of Attention Disorders*, 25(8), 1187-1195. https://doi.org/10.1177/1087054719887789

Mueller, T. F., Brielmaier, S., Domsch, H., Luyckx, V. A., Ehlers, T., & Krowatschek, D. (2010). Increased resting energy expenditure in children with attention-deficithyperactivity disorder. *Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity*, *15*(3), e144-e151.

National Research Council. (1989). *Diet and Health: Implications for Reducing Chronic Disease Risk.* National Academies Press, Washington, DC.

Neale, M. C., Hunter, M. D., Pritkin, J., Zahery, M., Brick, T. R., Kirkpatrick, R. M., Estabrook, R., Bates, T. C., Maes, H. H., & Boker, S. M. (2016). OpenMx 2.0: Extended structural equation and statistical modeling. *Psychometrika*, *81*, 535–549. https://doi.org/10.1007/s11336-014-9435-8

Nigg, J. T. (2013). Attention-deficit/hyperactivity disorder and adverse health outcomes. *Clinical Psychology Review*, *33*(2), 215-228. https://doi.org/10.1016/j.cpr.2012.11.005

Pan, P. Y., & Bölte, S. (2020). The association between ADHD and physical health: a co-twin control study. *Scientific Reports*, *10*(1), 1-13. https://doi.org/10.1038/s41598-020-78627-1

Peasgood, T., Bhardwaj, A., Biggs, K., Brazier, J. E., Coghill, D., Cooper, C. L., ... & Sonuga-Barke, E. J. (2016). The impact of ADHD on the health and well-being of ADHD children and their siblings. *European Child & Adolescent Psychiatry*, *25*(11), 1217-1231. https://doi.org/10.1007/s00787-016-0841-6

Pontifex, M. B., Saliba, B. J., Raine, L. B., Picchietti, D. L., & Hillman, C. H. (2013). Exercise improves behavioral, neurocognitive, and scholastic performance in children with attention-deficit/hyperactivity disorder. *The Journal of Pediatrics*, *162*(3), 543-551. https://doi.org/10.1016/j.jpeds.2012.08.036

Quesada, D., Ahmed, N. U., Fennie, K. P., Gollub, E. L., & Ibrahimou, B. (2018). A review: Associations between attention-deficit/hyperactivity disorder, physical activity, medication use, eating behaviors, and obesity in children and adolescents. *Archives of Psychiatric Nursing*, *32*(3), 495-504. https://doi.org/10.1016/j.apnu.2018.01.006

Rommel, A. S., Lichtenstein, P., Rydell, M., Kuja-Halkola, R., Asherson, P., Kuntsi, J., & Larsson, H. (2015). Is physical activity causally associated with symptoms of attention-deficit/hyperactivity disorder?. *Journal of the American Academy of Child & Adolescent Psychiatry*, *54*(7), 565-570. https://doi.org/10.1016/j.jaac.2015.04.011

Ruiz-Goikoetxea, M., Cortese, S., Aznarez-Sanado, M., Magallón, S., Zallo, N. A., Luis, E. O., ... & Arrondo, G. (2018). Risk of unintentional injuries in children and adolescents with ADHD and the impact of ADHD medications: A systematic review and meta-analysis.

Neuroscience & Biobehavioral Reviews, *84*, 63-71. https://doi.org/10.1016/j.neubiorev.2017.11.007

Sadeh, A., Sharkey, K.M., & Carskadon, M.A. (1994). Activity-based sleep-wake identification: An empirical test of methodological issues. *Sleep*, 17, 201–207. https://doi.org/10.1093/sleep/17.3.201

Salamanca, L. M. (2014). Biopsychosocial perspective of ADHD. *Open Journal of Epidemiology*, 4, 1-6.

San Mauro Martín, I., Blumenfeld Olivares, J. A., Garicano Vilar, E., Echeverry López, M., García Bernat, M., Quevedo Santos, Y., ... & Rincón Barrado, M. (2018). Nutritional and environmental factors in attention-deficit hyperactivity disorder (ADHD): A cross-sectional study. *Nutritional Neuroscience*, *21*(9), 641-647. https://doi.org/10.1080/1028415X.2017.1331952

Schutte, N. M., Huppertz, C., Doornweerd, S., Bartels, M., de Geus, E. J., & van Der Ploeg, H. P. (2020). Heritability of objectively assessed and self-reported sedentary behavior. *Scandinavian Journal of Medicine & Science inSsports*, *30*(7), 1237-1247. https://doi.org/10.1111/sms.13658

Shareghfarid, E., Sangsefidi, Z. S., Hosseinzadeh, M., Salehi-Abargouei, A., & Mohammadi, M. (2020A). Comparison of nutrients' intakes in children with and without Attention Deficit Hyperactivity Disorder. *Journal of Nutrition and Food Security*, *5*(2), 132-140. doi:10.18502/jnfs.v5i2.2799

Shareghfarid, E., Sangsefidi, Z. S., Salehi-Abargouei, A., & Hosseinzadeh, M. (2020B). Empirically derived dietary patterns and food groups intake in relation with Attention

Deficit/Hyperactivity Disorder (ADHD): A systematic review and meta-analysis. *Clinical Nutrition ESPEN*, *36*, 28-35. https://doi.org/10.1016/j.clnesp.2019.10.013

Smith, A. L., Hoza, B., Linnea, K., McQuade, J. D., Tomb, M., Vaughn, A. J., ... & Hook, H. (2013). Pilot physical activity intervention reduces severity of ADHD symptoms in young children. *Journal of Attention Disorders*, *17*(1), 70-82. https://doi.org/10.1177/1087054711417395

Sollie, H., Larsson, B., & Mørch, W. T. (2013). Comparison of mother, father, and teacher reports of ADHD core symptoms in a sample of child psychiatric outpatients. *Journal of Attention Disorders*, *17*(8), 699-710.

Tandon, P. S., Tovar, A., Jayasuriya, A. T., Welker, E., Schober, D. J., Copeland, K., ... & Ward, D. S. (2016). The relationship between physical activity and diet and young children's cognitive development: A systematic review. *Preventive Medicine Reports*, *3*, 379-390. https://doi.org/10.1016/j.pmedr.2016.04.003

Tandon, P. S., Sasser, T., Gonzalez, E. S., Whitlock, K. B., Christakis, D. A., & Stein, M. A. (2019). Physical activity, screen time, and sleep in children with ADHD. *Journal of Physical Activity and Health*, *16*(6), 416-422. https://doi.org/10.1123/jpah.2018-0215

Teucher, B., Skinner, J., Skidmore, P. M., Cassidy, A., Fairweather-Tait, S. J., Hooper, L., ... & MacGregor, A. J. (2007). Dietary patterns and heritability of food choice in a UK female twin cohort. *Twin Research and Human Genetics*, *10*(5), 734-748. https://doi.org/10.1375/twin.10.5.734

Tong, L., Xiong, X., & Tan, H. (2016). Attention-deficit/hyperactivity disorder and lifestyle-related behaviors in children. *PloS one*, *11*(9), e0163434. https://doi.org/10.1371/journal.pone.0163434

U.S. Department of Health and Human Services (2009). The HHS poverty guidelines. Washington, DC: U.S. *Department of Health and Human Services*. Retrieved from https://aspe.hhs.gov/poverty-guidelines.

van den Berg, L., Henneman, P., van Dijk, K. W., Delemarre-van de Waal, H. A., Oostra, B. A., van Duijn, C. M., & Janssens, A. C. J. (2013). Heritability of dietary food intake patterns. *Acta Diabetologica, 50*(5), 721-726. doi:10.1007/s00592-012-0387-0

Van Den Hoofdakker, B. J., Van der Veen-Mulders, L., Sytema, S., Emmelkamp, P. M., Minderaa, R. B., & Nauta, M. H. (2007). Effectiveness of behavioral parent training for children with ADHD in routine clinical practice: a randomized controlled study. *Journal of the American Academy of Child & Adolescent Psychiatry*, *46*(10), 1263-1271.

van der Aa, N., Bartels, M., te Velde, S. J., Boomsma, D. I., de Geus, E. J., & Brug, J. (2012). Genetic and environmental influences on individual differences in sedentary behavior during adolescence: A twin-family study. *Archives of Pediatrics & Adolescent Medicine, 166*(6), 509-514. doi:10.1001/archpediatrics.2011.1658

Verret, C., Guay, M. C., Berthiaume, C., Gardiner, P., & Béliveau, L. (2012). A physical activity program improves behavior and cognitive functions in children with ADHD: an exploratory study. *Journal of Attention Disorders*, *16*(1), 71-80. https://doi.org/10.1177/1087054710379735

Willcutt, E. G. (2012). The prevalence of DSM-IV attention-deficit/hyperactivity disorder: a meta-analytic review. *Neurotherapeutics*, *9*(3), 490-499. doi: 10.1007/s13311-012-0135-8

Yoshimasu, K., Barbaresi, W. J., Colligan, R. C., Voigt, R. G., Killian, J. M., Weaver, A. L., & Katusic, S. K. (2018). Adults with persistent ADHD: Gender and psychiatric

comorbidities—A population-based longitudinal study. *Journal of Attention Disorders*, *22*(6), 535-546. https://doi.org/10.1177/1087054716676342

Yu, C. L., Chueh, T. Y., Hsieh, S. S., Tsai, Y. J., Hung, C. L., Huang, C. J., ... & Hung, T. M. (2019). Motor competence moderates relationship between moderate to vigorous physical activity and resting EEG in children with ADHD. *Mental Health and Physical Activity*, *17*, 100302. https://doi.org/10.1177/1087054716676342

Zhou, F., Wu, F., Zou, S., Chen, Y., Feng, C., & Fan, G. (2016). Dietary, nutrient patterns and blood essential elements in Chinese children with ADHD. *Nutrients*, *8*(6), 352.

Table 1

Demographic of Study Sample (N = 670)

Variable		
Age at 8-year assessment in year, *M* (*SD*)		8.45 (.69)
Age at 9-year assessment in year, M (SD)		9.70 (.92)
Sex, *n* (%)		
	Female	560 (47.5)
	Male	506 (52.5)
Zygosity, *n* (%)		
	Monozygotic twins	282 (30.1)
	Same-sex dizygotic twins	342 (36.9)
	Opposite-sex dizygotic twins	303 (32.3)
	Unknown zygosity	10 (1.1)
Ethnicity at age 8, *n* (%)		
	Hispanic/Latinx	160 (23.7)
	Asian/Asian American	21 (3.1)
	Black/African American	26 (3.9)
	Native American	18 (2.7)
	Native Hawaiian or Pacific Islander	6 (.9)
	Non-Hispanic White/Caucasian	395 (58.5)
	Multiracial/ethnic or other	49 (7.3)
	Unknown	262 (28)
Education level of primary caregiver, *n* (%)		
	Less than a high school education	4 (0.6)
	High school degree	62 (8.7)
	Some college	181 (25.3)
	College degrees (e.g., BS or BA)	247 (34.5)
	2+ years of graduate school	24 (3.4)
	Graduate or professional degree	156 (21.8)
	Unknown	42 (5.9)
Income-to-needs ratio (at 12 months), *n* (%)		
	Below 1 (under poverty line)	42 (5.9)
	1-2 (near the poverty line)	126 (17.6)
	2-3 (lower middle class)	92 (12.8)
	Greater than 3 (middle to upper class)	304 (42.5)

Note. Percentages for income-to-needs ratios based on available data.

Table 2

Descriptive Statistics on Main study Variables (N=)

Variable	N	Mean	Standard Deviation	Minimum	Maximum	Skewness	Kurtosis
Primary caregiver-reported ADHD Symptoms at age-8	771	1.5	0.39	1	3	0.91	0.25
Secondary caregiver-reported ADHD Symptoms at age-8	506	1.64	0.4	1	3	0.51	-0.07
Teacher-reported ADHD Symptoms at age-8	536	1.4	0.45	1	3	1.24	0.68
Primary caregiver-reported ADHD Symptoms at age-9	728	2.13	0.54	1	3.92	0.34	-0.05
Child self-reported ADHD Symptoms at age-9	728	2.13	0.54	1	3.92	0.34	-0.05
Sedentary behavior	510	660.83	435.58	8.98	3637.01	1.58	5
Moderate-to-vigorous activity	516	1020.33	639.6	0	4001.97	1	1.49
Total fruit consumption	565	3.45	3.01	0	18	0.96	0.97
Total vegetable consumption	565	2.72	2.28	0	8	0.72	-0.28
Total fiber consumption	565	1.62	0.25	0	3.76	0.58	13.53
Total protein consumption	653	184.04	79.61	7	610.56	1.41	4.84
Total sugar consumption	653	287.85	128.49	10	917.98	0.79	1.97
Total carbohydrates consumption	566	683.71	249.31	38	1934.16	0.87	3.75
Total fat consumption	565	190.42	73.82	4.87	504.91	0.39	0.96
Total calorie consumption	653	5011.07	1637.28	220	11946.73	0.12	1.31
Age at 8-year assessment (in years)	800	8.44	0.7	6.96	10.26	-0.23	-0.44
Age at 9-year assessment (in years)	728	9.66	0.94	7.75	12.01	0.35	0.02
Sex	1066	-	-	-	-	-0.1	-1.99
SES composite at 8-year assessment	798	-0.02	0.8	-1.72	3.34	0.46	0.28
SES composite at 9-year assessment	809	-0.01	0.76	-1.53	3.33	0.53	0.51
Body mass index	686	16.67	2.83	12.11	34.93	1.97	5.98
Race/ethnicity at age-8 assessment	796	0.59	0.49	0	1	0.37	-1.87
Race/ethnicity at age-9 assessment	870	0.6	0.49	0	1	-0.42	-1.83

Note. Socioeconomic status (SES; mean composite of primary caregiver education, secondary caregiver education, and an income-to-needs ratio at 8- and

Table 3

Zero-Order Correlations of Main Study Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. Primary-caregiver-reported ADHD symptoms at age 8	-																		
2. Secondary-caregiver-reported ADHD symptoms at age 8	.53**	-																	
3. Teacher-reported ADHD symptoms at age 8	.44**	.28**	-																
4. Primary-caregiver-reported ADHD symptoms at age 9	.77**	.45**	.42**	-															
5. Self-reported ADHD symptoms at age 9	.34**	.29**	.32**	.30**	-														
6. Sedentary behavior	-.06	-.07	.00	-.08	-.03	-													
7. Moderate-to-vigorous activity	.11*	.14*	.09	.13*	.04	-.45**	-												
8. Fruit consumption	-.09*	.01	-.13**	-.04	-.05	-.12**	-.00	-											
9. Vegetable consumption	-.01	-.00	-.09	.02	-.01	-.07	-.02	.30**	-										
10. Fiber consumption	-.06	-.04	-.03	-.06	.01	-.10*	.05	.45**	.45**	-									
11. Protein consumption	-.07	.03	-.01	-.04	-.09*	-.03	.02	.30**	.41**	.49**	-								
12. Sugar consumption	-.07	.04	.04	-.02	-.07	'-.10*	.03	.45**	.25**	.44**	.44**	-							
13. Carbohydrate consumption	-.09*	-.00	-.04	0.06	-.06	-.08	.04	.42**	.36**	.59**	.55**	.82**	-						
14. Fat consumption	-.04	.02	-.02	-.04	-.01	-.08	.09	.18**	.36**	.50**	.64**	.51**	.60**	-					
15. Calorie consumption	-.08*	.03	-.02	-.04	-.07	-.10*	.07	.35**	.40**	.61**	.69**	.77**	.87**	.83**	-				
16. Age at 8-year assessment (in years)	-.06	-.04	.01	-.09*	0.01	.20**	-.06	-.15**	.00	-.06	.02	-.09*	-.02	-.05	-.06	-			
17. Age at 9-year assessment (in years)	-.10*	-.01	-.05	-.08	-.00	.16**	-.01	-.06	-.03	-.03	.06	-.04	.04	-.03	.02	.93**	-		
18. Sex	-.18**	-.18**	-.27**	-.22**	-.13**	.08	-.06	.05	.03	.02	-.09*	-.07	-.04	-.07	-.05	.03	-.03	-	
19. SES composite at 8-year assessment	-.15**	-.10*	-.12**	-.10*	-.07	-.10	.03	.32**	.12**	.14**	.08*	.21**	.14**	.06	.12**	-.10**	-.05	.04	-
20. SES composite at 9-year assessment	-.11**	-.05	-.11*	-.04	-.07	.01	.01	.27**	.15**	.15**	.12**	.18**	.18**	.05	.11*	-.04	-.08*	.05	.94
21. Body mass index	.12**	.09	.05	.06	.06	.10*	.01	-.00	-.03	.05	.02	-.05	-.00	-.00	-.02	.15**	.08	.01	-.13
22. Race/ethnicity at age-8 assessment	.09*	.06	.02	.05	.03	-.12**	.11*	.06	-.02	.10*	.07	.13**	.07	.09*	.08	-.17**	-.15**	-.05	.25
22. Race/ethnicity at age-9 assessment	.07	.06	.01	.07*	.03	-.07	.08	.07	-.04	.05	.09*	.14**	.10*	.06	.07	-.14**	-.14**	-.06	.22

Note. Socioeconomic status (SES; mean composite of primary caregiver education, secondary caregiver education, and income-to-needs ratio at 8- and 9- year assessments); Sex: Female = 1, Male = 0; Race/ethnicity: Not

Table 4

Mixed model regression coefficients estimating different reports of child ADHD symptoms at age 8 and 9 from diet, physical, activity, and covariates.

	Report of ADHD symptoms											
	Primary caregiver reported at age 8			Secondary caregiver-reported at age 8			Teacher-reported at age 8			Primary caregiver-reported at age 9		
	Est.	*SE*	*95% CI*	Est.	*SE*	*95% CI*	Est.	*SE*	*95% CI*	Est.	*SE*	*95% CI*
Sex	-0.14**	0.03	(-0.20,-0.08)	-0.14**	0.03	(-0.2, -0.9)	-2.50**	0.04	(-0.33, -0.19)	-0.15**	0.03	(-0.22, -0.09)
Age	-0.04	0.03	(-0.10,0.03)	-0.03	0.03	(-0.1, 0.03)	0.04	0.04	(-0.04, 0.13)	-0.03	0.04	(-0.1, 0.04)
SES composite	-0.07*	0.03	(-0.13, -0.02)	-0.07	0.03	(0.13,-0.02)	-0.06	0.03	(-0.12, 0.01)	-0.06	0.04	(-0.13, 0.13)
BMI	0.02*	0.01	(0.01, 0.03)	0.02**	0.01	(0.01, 0.03)	0.01	0.01	(0, 0.12)	0.02	0.01	(0,0)
Race/ethnicity	0.09*	0.05	(0, 0.19)	0.1*	0.05	(0.01, .19)	0.05	0.05	(-0.06, -0.19)	0.08	0.05	(-0.02, 0.19)
Fiber	-0.05	0.07	(-0.19, 0.09)	-0.05	0.12	(-0.30, 0.19)	0.04	0.08	(-0.12, 0.20)	-0.09	0.07	(-0.23, 0.05)
Fat	0.00	0.00	(0,0)	0.00	0.00	(0,0)	0.00	0.00	(0,0)	0.00	0.00	(0,0)
Carbohydrates	0.00	0.00	(0,0)	0.00	0.00	(0,0)	0.00	0.00	(0,0)	0.00	0.00	(0,0)
Protein	0.00*	0.00	(0,0)	0.00	0.00	(0,0)	0.00	0.00	(0,0)	0.00	0.00	(0,0)
Sugar	0.00	0.00	(0,0)	0.00	0.00	(0,0)	0.00	0.00	(0,0)	0.00	0.00	(0,0)
Calories	0.00	0.00	(0,0)	0.00	0.00	(0,0)	0.00	0.00	(0,0)	0.00	0.00	(0, 0.03)
Vegetables	0.00	0.01	(-0.02, 0.02)	0.00	0.01	(-0.02 ,0.03)	0.00	0.01	(-0.03, 0.01)	0.00	0.00	(-0.02, 0.02)
Fruit	-0.01	0.01	(-0.02, 0)	0.00	0.01	(-0.01, 0.02)	-0.01	0.01	(-0.03, 0)	-0.01	0.01	(-0.03, 0)
Sedentary Behavior	0.00*	0.00	(0,0)	0.00	0.00	(0,0)	0.00	0.00	(0,0)	0.00	0.00	(0,0)
Moderate-to-vigorous activity	0.00*	0.00	(0,0)	0.00*	0.00	(0,0)	0.00*	0.00	(0,0)	0.00*	0.00	(0,0)

Note. N = 670 twins. The outcome for each model is a different report of child ADHD symptoms and the predictors include sex, age, socioeconomic status (SES composite; mean composite of primary caregiver education, secondary caregiver education, and an income-to-needs ratio at 8- and 9-year assessments), familial risk of ADHD, and positive parenting. Est. = unstandardized partial regression coefficient. SE = standard error of the unstandardized coefficient. 95% CI = 95% confidence intervals. * $p < .05$; ** $p < .001$.

Table 5

Mixed model regression coefficients estimating different reports of child ADHD symptoms at age 9 from diet,
physical, activity, ADHD symptoms at age 8 and covariates.

	Report of ADHD Symptoms					
	Primary caregiver reported at age 9			Self-reported at age 9		
	Est.	*SE*	*95% CI*	Est.	*SE*	*95% CI*
Sex	-0.01	0.02	(-0.05, 0.01)	0.06	0.08	(-0.09, 0.21)
Age	-0.09*	0.03	(-0.17, -0.01)	-0.02	0.07	(-0.15, 0.11)
SES composite	-0.03	0.03	(-0.10, 0.04)	-0.02	0.06	(-0.14, 0.10)
BMI	0.00	0.00	(-0.01, 0.01)	0.00	0.01	(-0.03, 0.02)
Race/ethnicity	-0.02	0.06	(-0.13, 0.09)	0.10	0.10	(-0.09, 0.29)
Fiber	-0.23*	0.09	(-0.40, -0.06)	0.19	0.18	(-0.17, 0.56)
Fat	0.00	0.00	(0,0)	0.00	0.00	(0,0)
Carbohydrates	0.00*	0.00	(0,0)	0.00	0.00	(0,0)
Protein	0.00	0.00	(0,0)	0.00	0.00	(0,0)
Sugar	0.00	0.00	(0,0)	0.00	0.00	(0,0)
Calories	0.00	0.00	(0,0)	0.00	0.00	(0,0)
Vegetables	-0.01	0.01	(-0.03, 0.01)	-0.01	0.02	(-0.04 ,0.03)
Fruit	0.00	0.01	(-0.02, 0.01)	0.02	0.01	(-0.01, 0.05)
Sedentary Behavior	0.00	0.00	(0,0)	0.00	0.00	(0,0)
Moderate-to-vigorous activity	0.00	0.00	(0,0)	0.00	0.00	(0,0)

Note. N = 670 twins. The outcome for each model is a different report of child ADHD symptoms and the predictors include sex, age, socioeconomic status (SES composite; mean composite of primary caregiver education, secondary caregiver education, and an income-to-needs ratio at 8- and 9-year assessments), familial risk of ADHD, and positive parenting. Est. = unstandardized partial regression coefficient. SE = standard error of the unstandardized coefficient. 95% CI = 95% confidence intervals. * $p < .05$; ** $p < .001$.

Table 6

Univariate twin models and fit statistics

Scale	Model	-2LL	*df*	AIC	Δ-2LL	*p*	A	C	E
Primary caregiver reported ADHD symptoms age 8	ACE	394.17	644	402.17	-	-	0.76 (0.08, 0.14)	0.1 (-0.01, 0.04)	0.13 (0.01, 0.03)
	AE	**395.18**	**645**	**401.18**	**1.01**	**0.32**	**0.87 (0.82, 0.90)**	**0**	**0.13 (0.10, 0.18)**
	CE	447.01	645	435.01	52.84	<.001	0	0.6 (0.53, 0.67)	0.4 (0.33, 0.47)
	E	593.57	646	597.57	199.4	<.001	0	0	1
Secondary caregiver reported ADHD symptoms age 8	ACE	310.75	406	318.75	-	-	0.01 (-0.05, 0.05)	0.64 (0.07, 0.15)	0.35 (0.04, 0.08)
	AE	336.48	406	342.48	25.73	<.001	0.71 (0.61, 0.79)	0	0.29 (0.21, 0.39)
	CE	**310.74**	**406**	**316.74**	**-0.01**	**1**	**0**	**0.65 (0.56, 0.72)**	**0.35 (0.28, 0.44)**
	E	420.09	407	424.09	109.34	<.001	0	0	1
Teacher-reported ADHD symptoms age 8	ACE	417.36	446	425.36	-	-	0.73 (0.08, 0.18)	0.05 (-0.04, 0.06)	0.22 (0.03, 0.05)
	AE	**417.48**	**447**	**423.48**	**0.12**	**0.73**	**0.78 (0.68, 0.85)**	**0**	**0.22 (0.15, 0.32)**
	CE	434.89	447	440.89	17.53	<.001	0	0.51 (0.40, 0.61)	0.49 (0.39, 0.60)
	E	494.82	448	498.82	77.47	<.001	0	0	1
Primary caregiver reported ADHD symptoms age 9	ACE	305.89	598	313.89	-	-	0.8 (-0.15, 0.39)	0.14 (-0.008, 0.05)	0.06 (0.009, 0.01)
	AE	**307.84**	**599**	**313.84**	**1.95**	**0.16**	**0.94 (0.91, 0.95)**	**0**	**0.06 (.05, .09)**
	CE	399.29	599	405.29	93.4	<.001	0	0.66 (0.59, 0.72)	0.34 (0.28, 0.41)
	E	570.11	600	574.11	264.21	<.001	0	0	1
Self-reported ADHD symptoms age 9	ACE	1018.1	642	1026.1	-	-	0.31 (-0.5, .22)	0 (-0.09, 0.09)	0.69 (0.14, 0.26)
	AE	**1018.05**	**643**	**1024.05**	**-0.04**	**1**	**0.31 (0.15, 0.46)**	**0**	**0.69 (0.54, 0.85)**
	CE	1021.96	643	1027.96	3.87	0.05	0	0.17 (0.06, 0.28)	0.83 (0.72, 0.94)
	E	1031.71	644	1035.71	13.61	0.001	0	0	1
Protein	**ACE**	**5923.92**	**538**	**5931.92**	**-**	**-**	**0.29 (1070.74, 2338.04)**	**0.62 (2608.43, 4600.23)**	**0.09 (358.35, 671.25)**
	AE	5976	539	5982	52.09	<.001	0.91 (0.89, 0.93)	0	0.09 (0.07, 0.11)

	CE	5947.57	539	5953.57	23.65	<.001	0	0.81 (0.78, 0.83)	0.19 (0.17, 0.22)
	E	6230.91	540	6234.91	306.99	<.001	0	0	1
Sugar	ACE	6442.63	538	6450.63	-	-	0.13 (144.33, 3264.93)	0.73 (7718, 11931.14)	0.14 (1333.94, 2438.04)
	AE	6519.91	539	6525.91	77.29	<.001	0.88 (0.85, 0.90)	0	0.12 (0.10, 0.15)
	CE	**6442.73**	**539**	**6448.73**	**0.1**	**0.75**	**0**	**0.84 (0.81, 0.86)**	**0.16 (0.14, 0.19)**
	E	6760.95	540	6764.95	318.33	<.001	0	0	1
Fat	**ACE**	**5902.61**	**538**	**5910.6**	**-**	**-**	**0.21 (393.25, 1689.35)**	**0.64 (2284.77, 3945.33)**	**0.15 (499.16, 918.56)**
	AE	5953.42	539	5959.42	50.81	<.001	0.87 (0.83, 0.89)	0	0.13 (0.11, 0.17)
	CE	5910.17	539	5916.17	7.56	<.001	0	0.79 (0.76, 0.82)	0.21 (0.18, 0.24)
	E	6175.1	540	6179.1	272.5	<.001	0	0	1
Carbohydrates	**ACE**	**7120.3**	**538**	**7128.3**	**-**	**-**	**0.21 (5030.19, 14810.01**	**0.69 (25798.38, 40409.20)**	**0.1 (3434.75, 6265.15)**
	AE	7189.96	539	7195.96	69.66	<.001	0.91 (0.88, 0.93)	0	0.09 (0.07, 0.12)
	CE	7139.48	539	7145.48	19.18	<.001	0	0.82 (0.78, 0.84)	0.18 (0.16, 0.22
	E	7475.91	540	7479.91	355.62	<.001	0	0	1
Fiber	**ACE**	**-85.69**	**538**	**-77.68**	**-**	**-**	**0.58 (0.02, 0.53)**	**0.22 (0, 0.03)**	**0.2 (-0.01, 0.04)**
	AE	-81.98	539	-75.98	3.71	<.001	0.81 (0.74, 0.86)	0	0.19 (0.14, 0.26)
	CE	-65.09	539	-59.09	20.6	<.001	0	0.6 (0.52, 0.67)	0.4 (0.33, 0.48)
	E	51.55	540	55.55	137.24	<.001	0	0	1
Fruit	ACE	2422.41	536	2430.41	-	-	0.06 (-0.73, 1.67)	0.76 (4.91, 8.01)	0.19 (1.11, 2.05)
	AE	2489.45	537	2495.45	67.04	<.001	0.83 (0.78, 0.87)	0	0.17 (0.13, 0.17)
	CE	**2422.97**	**537**	**2428.97**	**0.57**	**0.45**	**0**	**0.8 (0.75, 0.84)**	**0.2 (0.16, 0.25)**
	E	2692.7	538	2696.7	270.29	<.001	0	0	1
Vegetables	ACE	2293.29	536	2301.29	-	-	0.05 (-0.27, 1.15)	0.84 (5.55, 8.37)	0.11 (0.64, 1.18)
	AE	2404.4	537	2410.4	111.12	<.001	0.89 (0.86, 0.92)	0	0.11 (0.08, 0.14)
	CE	**2294.62**	**537**	**2300.62**	**1.34**	**0.25**	**0**	**0.87 (0.84, 0.90)**	**0.13 (0.10, 0.16)**

	E	2680.89	538	2684.89	387.61	<.001	0	0	1
Calories	**ACE**	**6167.54**	**669**	**6175.54**	**-**	**-**	**0.03 (69.31, 120.39)**	**0.97 (2920.74, 3989.44)**	**0 (12.73, 22.29)**
	AE	6705.24	670	6711.24	537.7	<.001	0.99 (0.99, 0.10)	0	0.01 (0.005, 0.009)
	CE	6217.35	670	6223.35	49.81	<.001	0	0.99 (0.98, 0.99)	0.01 (0.01, 0.02)
	E	7415.52	671	7419.52	1247.98	<.001	0	0	1
Sedentary behavior	ACE	2782.05	489	2790.05	-	-	0.63 (4.66, 18.30)	0.01 (-4.92, 5.32)	0.36 (-1.69, 3.09)
	AE	**2782.06**	**490**	**2788.06**	**0.01**	**0.94**	**0.64 (0.50, 0.74)**	**0**	**0.36 (0.26, 0.50)**
	CE	2790.22	490	2796.22	8.17	<.001	0	0.39 (0.28, 0.49)	0.61 (0.51, 0.72)
	E	2828.67	491	2832.67	46.62	<.001	0	0	1
Moderate-to-vigorous activity	ACE	3165.07	493	3172.07	-	-	0.68 (26.97, 29.29)	0.09 (-6.71, 12.79)	0.23 (6.11, 12.77)
	AE	**3165.66**	**494**	**3171.66**	**0.59**	**0.78**	**0.78 (0.69, 0.83)**	**0**	**0.22 (0.17, 0.31)**
	CE	3181.2	494	3187.2	16.13	<.001	0	0.50 (0.40, 0.58)	0.5 (0.42, 0.60)
	E	3249.47	495	3253.47	84.4	<.001	0	0	1

Note. A, C, and E are standardized squared parameter estimates for additive genetic, shared environmental, and nonshared