



The link between poor quality nutrition and childhood antisocial behavior: A genetically informative analysis



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ABSTRACT

Purpose: The current study explores whether the association between poor quality nutrition and child antisocial behavior is robust to shared environmental and genetic influences.

Method: Data from the Early Childhood Longitudinal Study: Birth Cohort are employed, which includes a large, nationally representative sample of twin pairs. DeFries–Fulker (DF) analysis is used to test the current hypothesis.

Results: The results suggest that poor quality nutrition during preschool increases the extent of antisocial behavior during elementary school after the influence of genes and the shared environment are taken into account.

Conclusions: The relationship between poor quality nutrition and subsequent behavioral problems is robust to shared environmental and genetic influences, with variation in eating behaviors between twins predicting their relative likelihood of exhibiting antisocial behaviors.

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Research across multiple disciplines has suggested that nutritional factors are related to various aspects of development, including cognition (Connolly & Beaver, 2015; Gómez-Pinilla, 2008; Molteni, Barnard, Ying, Roberts, & Gómez-Pinilla, 2002) and behavior (Galler et al., 2011; Oddy et al., 2009; Woo et al., 2014). The adequate ingestion of various nutrients, vitamins, and minerals (e.g., folate, zinc, iron, magnesium, polyunsaturated fatty acids) is essential for optimal brain functioning (for a review, see Gómez-Pinilla, 2008). Conversely, poor nutrition appears to diminish neuropsychological functioning and dampen synaptic plasticity (see Molteni et al., 2002). Research has also revealed that such deficits in neuropsychological functioning may reduce self-control (Jackson & Beaver, 2013) and heighten the risk of misconduct (Espy, Sheffield, Wiebe, Clark, & Moehr, 2011; Riggs, Blair, & Greenberg, 2004; Schoemaker, Mulder, Deković, & Matthys, 2013). In short, research suggests that poor nutrition during childhood seems to predispose children to higher levels of aggression and related antisocial behaviors (Liu, Raine, Venables, & Mednick, 2004; Woo et al., 2014).

Notwithstanding this body of literature, criminologists have given little attention to the link between nutritional factors and antisocial behavior (however, see Liu et al., 2004; Liu & Raine, 2006). More specifically, the small number of relevant criminological studies to date have largely examined whether acute malnutrition and/or micronutrient supplementation are associated with behavioral outcomes. Criminological studies linking specific eating patterns to antisocial behavior, however, are sorely lacking. Perhaps even more importantly, observational studies have yet to adequately account for the role of both familial and

genetic confounding in the relationship between child nutrition and subsequent antisocial behavior. This limitation is particularly significant, as the association between nutritional factors and antisocial behavior may emerge as a statistical artifact once unmeasured familial and genetic confounding are taken into account. In light of these voids in the literature, the current study uses a within-family, genetically informative approach to test the relationship between poor quality nutrition and childhood antisocial behavior.

Childhood antisocial behavior as a predictor of crime and delinquency

A long line of research has examined the extent to which childhood antisocial behavior predicts the stability and severity of future antisocial behavior (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996; Tremblay, Pihl, Vitaro, & Dobkin, 1994). When age-appropriate indicators of antisocial behaviors are employed, researchers typically find evidence that antisocial children are at risk of becoming antisocial adolescents and adults (Broidy et al., 2003; Campbell, Shaw, & Gilliom, 2000; Fergusson, Boden, & Horwood, 2014; Fergusson & Horwood, 1995; Nagin & Tremblay, 1999; Nagin & Tremblay, 2001). More specifically, elevated levels of physical aggression and associated externalizing behaviors during early childhood seem to significantly increase the odds of criminal activity during adolescence and adulthood (Broidy et al., 2003; Kokko, Tremblay, Lacourse, Nagin, & Vitaro, 2006; Nagin & Tremblay, 1999; Thompson et al., 2010). To illustrate, a seminal study by Nagin and Tremblay (1999) examined the physical aggression trajectories of males from childhood to adolescence. The authors found that subjects who exhibited high levels of externalizing behavior at age 6 tended to

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engage in high levels of delinquency at age 15. The results suggest that early oppositional, externalizing and aggressive behaviors are driven by similar developmental processes, and that this cluster of traits is one of the best predictors of adolescent delinquency (Nagin & Tremblay, 1999). A follow-up study by Nagin and Tremblay (2001) found that the odds of belonging to a “high aggression” group during high school were increased by a factor of 3 for boys who frequently exhibited hyperactivity, defiance, and aggression during kindergarten.

A number of more recent studies have corroborated these results. For example, a study by Piquero, Carriaga, Diamond, Kazemian, and Farrington (2012) found that aggression during both childhood and adolescence is associated with a greater likelihood of a criminal conviction through mid-adulthood (age 40), implying a strong degree of continuity in antisocial behavior across the life course. Recent research has also indicated that bullying and externalizing behaviors during childhood are significantly predictive of official and self-report measures of property and violent offending during adulthood (Fergusson et al., 2014; see also Schaeffer, Petras, Ialongo, Poduska, & Kellam, 2003). Ultimately, both oppositional and hyperactive behaviors during childhood appear to be predictive of crime as well as several analogous behaviors, including drug abuse (Pingault et al., 2012), gambling (Shenassa, Paradis, Dolan, Wilhelm, & Buka, 2012), and risky sexual behavior (Timmermans, Van Lier, & Koot, 2008).

The role of nutritional factors in the development of antisocial behavior

Among the studies that have examined the origins of childhood antisocial behavior, relatively few of them have considered the role of nutritional factors in the development of such behaviors (Galler et al., 2011; Woo et al., 2014). The general paucity of research in this area is somewhat surprising, considering the substantial body of literature that a) links nutritional factors to brain development (Black, 2008; Gómez-Pinilla, 2008) and b) links particular aspects of brain development to childhood antisocial behavior (Riggs et al., 2004; Schoemaker et al., 2013). Nutritional factors may be especially important to cognitive and behavioral development early in the life course, as the brain is both a) experiencing exponential growth during this time and b) placing increasing demands on exogenous nutrients to supply the building blocks (e.g., proteins) that facilitate such growth (see Benton, 2008; Georgieff, 2007).

Although a large number of studies have explored the relevance of childhood nutritional factors to healthy brain development (see Bellisle, 2004 for a review), fewer studies have specifically explored whether nutrition during early childhood is associated with the development of childhood behavioral problems (Galler et al., 2011; Liu et al., 2004). Importantly, animal research suggests that deprivation of key nutrients (e.g., omega-3 fatty acids) at critical developmental periods not only reduces synaptic differentiation and formation, but also increases aggressive behavior by disadvantageously altering serotonin levels (Hibbeln, Ferguson, & Blasbalg, 2006). The limited number of studies with human subjects suggests that similar neurological and behavioral outcomes may be related to the quality of nutrition (Galler et al., 2011; Liu et al., 2004; Sinn, 2008). To illustrate, a recent study by Raine, Portnoy, Liu, Mahomed, and Hibbeln (2015) revealed that random assignment of omega-3 consumption resulted in reductions in both externalizing and internalizing behavior in children, which was partly mediated by reductions in parental antisocial and aggressive behavior.

Ultimately, the research to date has revealed that poor nutrition during early childhood might contribute to the development of a number of behavioral problems (Galler et al., 2011; Oh, Ahn, Chang, Kang, & Oh, 2013; Park et al., 2012; Woo et al., 2014). For instance, a study by Liu et al. (2004) found that children who exhibited signs of acute malnutrition (e.g., hair dyspigmentation, hair loss, and angular stomatitis) during the first few years of life engaged in more externalizing behaviors

at ages 8, 11, and 17, implying that nutritional factors during the earliest stages of the life course may indeed play a role in the development of aggressive and hyperactive behavior. Similarly, Galler et al. (2011) found that early childhood malnutrition (defined as moderate-to-severe protein-energy malnutrition) was predictive of both deficits in executive functioning and higher parent-reported aggression toward peers at ages 9–15. Thus, scholars tend to detect quite robust associations between acute malnutrition and antisocial behaviors several years into the future (see also Liu et al., 2014).

In addition to acute malnutrition, it is possible that the actual quality of the diet, or differences in the frequency with which *specific* foods (or groups of foods) are consumed, might also influence the development of behavioral problems in children. A number of studies have investigated whether children with poorer eating habits are at an increased risk of conduct problems, including ADHD symptomatology (Benton, 2008; Howard et al., 2011; Oh et al., 2013; Park et al., 2012; Woo et al., 2014). Overall, the results indicate that a western dietary pattern may be particularly conducive to the development of conduct problems (Oh et al., 2013; Park et al., 2012; Woo et al., 2014). For example, a recent study by Woo et al. (2014) revealed a traditional-healthy pattern of eating, characterized by a diet low in fat and high in fatty acids and minerals, lowered the odds of developing ADHD, whereas a snacking pattern, characterized by high consumption of sweets, snacks, and breads, increased the odds of developing ADHD. Another study revealed that high intake of sweets during childhood significantly increases behavioral problems and deficits in social skills (Oh et al., 2013). Similar results linking poor diet to conduct problems have been obtained using adolescent samples (see Howard et al., 2011; Oddy et al., 2009).

There have also been a handful of randomized control trials examining the benefits of comprehensive micronutrient supplementation in curbing antisocial behavior (Sinn, 2008). For instance, various micronutrients, including vitamin A, iodine, iron, and zinc, seem to influence the behavioral profiles of children (Schoenthaler & Bier, 2000), adolescents (Schoenthaler et al., 1997), and adults (Gesch, Hammond, Hampson, Eves, & Crowder, 2002; Zaalberg, Nijman, Bulten, Stroosma, & van der Staak, 2010). Thus, it appears that providing nutrient-dense diets through supplementation significantly reduces various forms of antisocial behavior, including fighting, vandalism, endangering others, and other aggressive behaviors. In sum, acute malnutrition and poor dietary patterns both appear to heighten the risk of various conduct problems during childhood and even into later life stages. Conversely, there is some evidence to suggest that mimicking a well-balanced diet through the use of supplementation can reduce the frequency and/or severity of antisocial behavior across the life course.

Poor nutrition and childhood antisocial behavior: the possibility of genetic and familial confounding

Although the literature is generally supportive of the link between nutritional factors and antisocial behavior, the vast majority of prior studies suffer from two key limitations. The first of these limitations is the issue of familial confounding, or selection bias stemming from factors within the family/home environment. This shortcoming is rooted in the frequent use of observational data that are based on samples of only one child per household (for examples, see Oh et al., 2013; Park et al., 2012; Woo et al., 2014). Under such circumstances, researchers typically include a number of statistical controls for potential confounding influences within the family environment to minimize the likelihood of familial confounding. Even so, since diet quality during childhood is closely related to features of the family environment (e.g., parenting, food rules/rituals, parental education, household income, etc.), it is possible that residual confounding may render the relationship between poor quality nutrition and child antisocial behavior spurious due to omitted variable bias and/or poor measurement (see Peters, Dollman, Petkov, & Parletta, 2013). Research using a within-family design would help to address this issue, as it would be capable

of detecting the degree to which an environmental factor (such as poor quality nutrition) is shared by siblings within the same household (see D'Onofrio, Lahey, Turkheimer, & Lichtenstein, 2013).

Another significant, though rarely noted limitation of this body of literature is its lack of attention to genetic confounding (see Barnes, Boutwell, Beaver, Gibson, & Wright, 2014a). Specifically, selection into nutritious or non-nutritious environments appears to be correlated with and/or influenced by genetic factors, in addition to family environmental factors (see Faith et al., 2006; Peters et al., 2013; Tholin, Rasmussen, Tynelius, & Karlsson, 2005). A number of studies have also indicated that genetic factors play a significant role in the development of childhood behavioral problems (Baker, Jacobson, Raine, Lozano, & Bezdjian, 2007; Barnes, Boutwell, Beaver, & Gibson, 2013). Therefore, if similar genetic risk factors underpin both exposure to inadequate nutrition during childhood as well as the development of antisocial behavior, then the link between nutrition and childhood antisocial behavior may be spurious due to genetic factors. It is possible, for example, that children may exhibit both poor eating habits and poor behavior as a result of a genetically influenced latent trait. Thus, the challenging eating behaviors and the challenging social behaviors may stem from a similar source (e.g., low self-control, difficult temperament) that might, at least in part, have genetic underpinnings (Beaver et al., 2009). To the extent that this process is occurring, studies of early childhood nutrition, and its effect on behavioral problems, might be misspecified. No research to date, however, has explicitly examined whether the relationship between early childhood diet and childhood antisocial behavior is robust to the influence of genetic factors.

The current study

The current study seeks to answer the call of scholars such as Jaffee, Strait, and Odgers (2012) and D'Onofrio et al. (2013), who have acknowledged the need for sibling designs and other statistical innovations in order to a) bridge the gap between the biological and the social sciences and b) more effectively distinguish between environmental correlates and causes of antisocial behavior. More specifically, the present study aims to determine whether dietary patterns during preschool are predictive of subsequent conduct problems during elementary school, independent of familial and genetic factors. Although a number of prior studies have detected associations between nutritional factors and childhood antisocial behavior, it remains to be seen whether the association between nutrition and antisocial behavior during childhood is robust to both shared environmental and genetic factors.

Method

Data

The current study uses data from the Early Childhood Longitudinal Study, Birth Cohort (ECLS-B). The ECLS-B examines a large, nationally-representative sample of children born in the United States in 2001. Using a stratified sampling approach, ECLS-B researchers sampled birth certificates registered with the National Center for Health Statistics in the year 2001, which covers approximately 99% of U.S. births that occur in a given year. Children were deemed ineligible if a) they died before the age of 9 months b) they were adopted before the age of 9 months or c) their mothers were younger than 15 at the time of birth.

Five waves of data have been collected to date, spanning several years of development (i.e., from age 9 months until the kindergarten school year). Data were collected from multiple sources, including parents, independent raters, day care providers, and school teachers. Approximately 10,600 children participated in the study at the first wave of data collection. Interviews at wave 1 were conducted between the fall of 2001 and the fall of 2002, when the children were, on average, about 9 months of age (although they ranged from about 6 to 14 months

of age). The second wave of data collection occurred between the fall of 2003 and the fall of 2004, when the children were approximately 2 years old. By the third wave of data collection, when children were roughly 4 years of age, many survey items were modified in order to reflect the enhanced autonomy and sophistication of the focal children. For example, parents were asked several questions pertaining to their child's academic preparedness, social aptitude, learning capacities, dietary practices, and behavioral problems.

Finally, the fourth and fifth waves of data collection occurred during the fall of 2007 and the fall of 2008. Subjects who had not yet entered kindergarten by the fall of 2007 were not assessed on the measures during the fourth wave of data collection, but instead were assessed during the fifth wave of data collection. Conversely, subjects who had entered kindergarten by wave 4 were not assessed at wave 5. During these waves, teachers were asked to report on their qualifications, teaching style, and classroom setting, as well as the traits and behaviors of the focal children. Specifically, questions regarding the learning, temperament, behavior, and peer relationships of focal children were asked of teachers at waves 4 and 5. Similar questions regarding the behavior of focal children were also asked of parents at these waves.

The ECLS-B is especially well-suited to the current study due to its inclusion of a large sample of approximately 1600 twins. Twins were oversampled in the ECLS-B study, which enables researchers to conduct genetically informative analyses of the data.¹ After eliminating opposite-sex twins and twins with undetermined zygosity, the sample employed in the current study consisted of nearly 1000 twins (N = 976), 238 monozygotic and 738 dizygotic.²

Measures

Childhood antisocial behavior (W4/5)

At waves 4 and 5 of data collection, parents and kindergarten teachers were asked a number of questions concerning the behavior of focal children. Measures were taken from the Preschool and Kindergarten Behavior Scales – Second Edition (PKBS-2) (Merrell, 2003). Most of the questions included in the PKBS-2 were asked of parents and teachers, with a few exceptions. Ultimately, seven parent-rated questions and six teacher-rated questions were identified as indicators of childhood antisocial behavior. Importantly, prior research using the ECLS-B has used the same items to measure childhood antisocial behavior (see Boutwell, Franklin, Barnes, & Beaver, 2011; Barnes et al., 2013). The inclusion of additional items from the PKBS-2, moreover, does not improve the observed alpha level.

Parents were asked about how often the child got angry, acted impulsively, was unable to sit still, and engaged in physically aggressive acts (e.g., hit, kick, or punch) during the 3 months prior to the interview. Parents were also asked how frequently the child threw tantrums, destroyed things, and annoyed other children during the 3 months prior to the interview. Response options ranged from 1 (never) to 5 (very often). Teachers were asked very similar questions at waves 4 and 5. Specifically, teachers were asked to report on the extent to which the child acts without thinking, engages in physical aggression, is overly active, disrupts other children and/or the class, annoys/bothers other children, and has temper tantrums. Response options for these six items also ranged from 1 (never) to 5 (very often).

Children who had not entered kindergarten by wave 4 were included in the analysis by utilizing their data from wave 5 (i.e., the wave they entered kindergarten). However, for children who had already entered kindergarten at wave 4, the wave 4 items were used.³ Both parent-rated and teacher-rated items were summed together and averaged to create a scale of antisocial behavior at wave 4/5. Importantly, the internal reliability of the items was high ($\alpha = .86$). The scale was created so that higher scores indicate greater manifestation of behavioral problems across school and family settings.

Dietary components

At wave 3 of data collection, when subjects were approximately 4–5 years of age, caregivers (usually the parent) were asked about the dietary patterns of their children. Specifically, six components of the children's diet were tapped in a series of questions in order to determine each child's eating habits. These components included vegetable consumption, fruit consumption, fast food consumption, sweets consumption, salty snack consumption, and soda consumption.⁴ Response options pertaining to the frequency of consumption of each of the dietary components included *not at all in the past 7 days* (7), *1–3 times during the past 7 days* (5), *4 to 6 times during the past 7 days* (6), *1 time per day* (1), *2 times per day* (2), *3 times per day* (3), and *4 or more times per day* (4). In the case of vegetable and fruit consumption, the items were reverse-coded so that higher scores were given to children whose parents reported that they ate vegetables and/or fruit less frequently. For each item, possible scores ranged from 0 to 6, with higher scores reflecting poorer eating behaviors within each dietary component measured.

Poor quality nutrition

In an effort to approximate the overall eating patterns of focal children in the study, a composite measure of poor quality nutrition was constructed by summing scores on the six dietary components (with the vegetable and fruit items reverse-coded). As each item has a possible range of 0–6, and there are six items, possible scores on the composite item range from 0 to 36. However, in the same-sex twin subsample, the lowest observed score was 2 and the highest observed score was 29. Subjects who scored higher on this composite measure are those who have the least healthy eating habits across the six diet domains tapped in the current study (i.e., low vegetable consumption, low fruit consumption, high fast food consumption, high sweets consumption, high salty snack consumption, high soda consumption), whereas those who scored lower have healthier overall eating habits.

Analysis

The current study employs a technique known as DeFries–Fulker (DF) analysis. DF analysis is a regression-based method that permits the estimation of the relative effects of genetic factors, shared environmental factors, and nonshared environmental factors. These estimates are obtained by using samples of sibling pairs who differ in their degree of genetic similarity (e.g., MZ and same-sex DZ twins). DF analysis decomposes the variance in the outcome variable into the proportions explained by genetic and environmental factors, while also allowing for the estimation of regression coefficients for specified nonshared environments (i.e., environments that are not shared by siblings within a kinship pair). DF analysis was utilized in the current study for two main reasons. First, DF analysis is an effective way to control for residual confounding that can be attributed to shared environmental factors (e.g., household economic disadvantage and parental education). This technique also removes the need to specify traits/environments that are shared by twins within a twin pair and include them in the model. Prior research linking nutritional factors to behavioral problems has primarily employed between-family, nonexperimental designs, which are often plagued by residual confounding due to the inability to properly control for a host of familial factors that are shared by siblings in the same household. Second, DF analysis is capable of modeling the proportion of the variance in the outcome of interest that can be attributed to genetic influences, which strengthens causal inferences about the influence of the nonshared environmental factors that are examined simultaneously (e.g., sibling differences in dietary habits). DF analysis provides a more rigorous test of environmental influences than other observational research because of its ability to a) distinguish between shared and nonshared environmental influences and b) test whether specific nonshared environmental effects are robust to the effects of genetic factors.

The DF equation has been revised since it was originally postulated by DeFries and Fulker (1985, 1988) in order to be fit for use among samples drawn from the general population (Rodgers, Rowe, & Li, 1994). Recently, Rodgers and Kohler (2005) proposed another improvement to the equation. The equation is depicted as follows:

$$K_1 = b_0 + b_1(K_2 - K_m) + b_2[R * (K_2 - K_m)] + e. \quad (1)$$

K_1 in the above equation represents the antisocial behavior score (i.e., the outcome variable) for one of the twins being analyzed, K_2 represents their cotwin's antisocial behavior score. K_m represents the mean value of K_2 (or, in this study, the mean antisocial behavior score of the cotwins). Therefore, the parenthetical statement $K_2 - K_m$ signifies that K_2 is mean-centered in this equation. R is an indicator of the genetic similarity between the kinship pair (1 for MZ twin pairs and .5 for DZ twin pairs), and $R * K_2$ is an interaction term that multiplies the cotwin's antisocial behavior score by their degree of genetic similarity with their twin. Moreover, b_0 represents the constant, b_1 represents the proportion of the variance in antisocial behavior that is explained by shared environmental influences, and b_2 represents the proportion of the variance in antisocial behavior that is explained by genetic influences. The error term (e) encompasses the effects of the nonshared environment on antisocial behavior and error.

The coefficients in the above equation do not reveal the effect of any particular gene or shared environment on antisocial behavior precisely because the coefficients signify latent factors. Nevertheless, Eq. (1) can be altered slightly to allow for the inclusion of specific nonshared environments of interest. In the current study, the following equation is employed in order to examine a number of nonshared environments related to early childhood nutrition. Doing so facilitates an examination of whether these nonshared environments have a significant influence on childhood antisocial behavior, net of genetic and shared environmental factors. The DF equation that allows researchers to include specific nonshared sources of variance is depicted as follows:

$$K_1 = b_0 + b_1(K_2 - K_m) + b_2[R * (K_2 - K_m)] + b_3ENVDIF + e. \quad (2)$$

Eq. (2) is almost an exact replication of Eq. (1). The only difference is the term ENVDIF. ENVDIF represents the difference score that is created when one twin's score on a variable is subtracted from their cotwin's score on the same variable. Thus, when b_3 is significant and positive, it suggests that the twins with higher values on the independent variable of interest tend to score higher on the outcome, relative to their cotwins. In the current study, difference scores are calculated for each of the nutritional factors in order to determine if twin differences in these variables predict differences in antisocial behavior, net of genetic and shared environmental influences. Importantly, b_3 in Eq. (2) does not represent a latent factor, but instead represents a regression coefficient, and needs to be interpreted as such (e.g., using critical t-values, and p-values).

In order to maximize the information available on twin pairs in the ECLS-B, and in line with prior research (Beaver et al., 2009; Haynie & McHugh, 2003; Rodgers, Buster, & Rowe, 2001), twins were double-entered in the current study. Double entering is the most frequent choice among researchers when using the augmented DF equations. Rodgers et al. (2001) argue that double entering is the correct approach when the specification of which siblings represent K_1 and which siblings represent K_2 is arbitrary, which is the case in the current study.⁵ Double entering allows each twin to contribute to both the independent and dependent variables in the DF analysis. Despite this advantage, double entering violates the assumption of the independence of observations (since the same observations are repeated twice). Violation of this assumption results in deflated standard errors, which biases tests of statistical significance. In line with prior research (Beaver et al., 2009; Haynie & McHugh, 2003), this bias was addressed by employing cluster-robust

standard errors, which takes account of the clustering of observations when estimating the statistical significance of the results.

Results

Table 1 displays the descriptive statistics pertaining to the DF analyses examining the influence of poor quality nutrition on externalizing behavior. The table reveals that high soda and fast food consumption are, on average, less common than low fruit and vegetable consumption. The difference scores also indicate substantial variation between twins within a twin pair on all dimensions of the diet measured.⁶ For example, in the case of both low vegetable and fruit consumption, some twin pairs emerged in which one twin ate vegetables and/or fruit four or more times a day, but their cotwin never ate vegetables and/or fruit (this would correspond to a difference score of 6 or –6). Similar variation in dietary habits between twins within a twin pair was also detected across the other dimensions of the diet. Of the dietary measures, however, fast food consumption showed the smallest range of variation between twins within a twin pair. The results suggest that, in many cases, twins living in the same household exhibit differences in their dietary patterns, even during early childhood.

In light of the sizeable number of twins who differed in their dietary behaviors, genetically informative analyses using DF modeling strategies were subsequently conducted. Table 2 contains the results of eight models exploring the influence of the shared environment, heritability, and poor quality nutrition on childhood antisocial behavior. Model 1 displays the results of the baseline model with no nonshared environmental effects specified. Model 2 includes the composite measure of poor quality nutrition during childhood as a nonshared environment in the DF equation. Finally, models 3 through 8 examine whether specific components of poor quality nutrition are more likely to lead to antisocial behavioral problems than others. The results of model 1 indicate that approximately 83% of the variance in childhood antisocial behavior can be attributed to genetic factors, with the remaining proportion of the variance being attributable to nonshared environmental factors and error. Model 2 expands model 1 by including the composite measure of poor quality nutrition as a nonshared source of variance. The results of model 2 suggest that poor quality nutrition during the preschool years is associated with a significant increase in antisocial behavior during kindergarten, even after taking genes and the shared environment into account. More precisely, the results suggest that, within twin pairs, the twin with a generally poorer diet tends to exhibit a significantly greater degree of antisocial behavior during kindergarten.

When the six nutritional components that comprise the poor quality nutrition measure are examined individually, a number of them emerge

as independent predictors of antisocial behavior during kindergarten. For example, the results from model 4 suggest that twins who consume fewer servings of fruit at age 4, relative to their cotwin, score significantly higher on the measure of childhood antisocial behavior, independent of the influence of genes. Similar findings were also obtained when high fast food consumption, high sweets consumption, and high salty snack consumption were examined (models 5, 6, and 7). However, nutritional components related to low vegetable consumption and high soda consumption did not appear to significantly influence childhood antisocial behavior once the influence of genes and the shared environment were taken into account. In sum, the results suggest that an early-childhood diet low in fruit, yet high in sweets, fast foods and salty snacks, is especially likely to heighten conduct problems during kindergarten, even when genetic factors and shared environmental factors are accounted for in the models. However, twins who consume a relatively high amount of soda and/or a relatively low amount of vegetables do not appear to exhibit significantly greater levels of behavioral problems relative to their cotwin, despite the significant influence of the composite nutrition measure on subsequent antisocial behavior.

An additional concern that emerges when examining the influence of poor quality nutrition on childhood antisocial behavior is whether the effects of poor quality nutrition on antisocial behavior are robust to prior levels of antisocial behavior. Put differently, is the link between poor diet and subsequent conduct problems merely an indicator of stability in conduct problems? Twins with unhealthier eating patterns may only have a greater degree of subsequent behavioral problems (relative to their cotwin) because their poor eating is simply a manifestation of a more challenging disposition. In short, dietary discordance between twins may suggest a household dynamic in which parents are attempting to provide a healthy, balanced diet to both twins, but the twin with the more challenging, noncompliant personality and/or behavior merely refuses to consume the healthy options that are offered to him/her. In this way, poor eating habits may be a reflection of difficult temperament and/or behavior, which was reported previously by parents at wave 3. Thus, it is possible to model the influence of preschool diet on kindergarten antisocial behavior, independent of preexisting behavioral problems, to further explore the robustness of dietary influences on childhood antisocial behavior.

The DF models testing this possibility are presented in Table 3. Model 1 of Table 3 includes the composite measure of poor quality nutrition as a nonshared source of variance, as well as a measure of existing behavioral problems at age 4. As expected, the results suggest that poor quality nutrition during the preschool years is associated with a significant increase in behavioral problems during kindergarten, even after taking genes, the shared environment, and prior antisocial behavior into account.⁷ The influence of poor quality nutrition on subsequent antisocial behavior is therefore robust to the inclusion of the measure of antisocial behavior at age 4, which buttresses the notion that the relationship detected between poor eating habits and poor behavior is not merely tapping stability in poor behavior.

Models 2 through 7 of Table 3 examine whether the individual components of the diet during preschool still have an impact on kindergarten antisocial behavior, independent of preexisting behavioral problems. Interestingly, a number of significant effects emerge, although they are not entirely consistent with the results presented in Table 2. Specifically, the results suggest that twins who consume fewer servings of vegetables at age 4, relative to their cotwin, display a significantly greater degree of behavioral problems during kindergarten. Similar findings were also obtained when low fruit consumption and high sweets consumption were examined (models 3 and 5). However, nutritional components related to fast food consumption, salty snack consumption, and soda consumption did not significantly impact antisocial behavior once genes, the shared environment, and preexisting behavioral problems were taken into account.⁸ While the significance of specific dietary components changed slightly once stability in antisocial behavior was modeled, the pattern of results across all

Table 1
Descriptive statistics.

Variable	Mean	Standard deviation	Range
<i>Full twin sample</i>			
Childhood antisocial behavior	2.15	.60	1–4.57
Poor quality nutrition	11.86	3.87	2–29
Low vegetable consumption	2.99	1.44	0–6
Low fruit consumption	2.77	1.43	0–6
High fast food consumption	1.00	1.01	0–6
High sweets consumption	2.14	1.28	0–6
High salty snack consumption	1.64	1.18	0–6
High soda consumption	1.31	1.46	0–6
<i>Predictors (difference scores)</i>			
Poor quality nutrition	0	2.64	–18–18
Low vegetable consumption	0	1.17	–6–6
Low fruit consumption	0	1.24	–6–6
High fast food consumption	0	.68	–4–4
High sweets consumption	0	.86	–5–5
High salty snack consumption	0	.98	–6–6
High soda consumption	0	1.04	–5–5

Table 2
DF analysis of the shared environment, heritability, and poor quality nutrition as predictors of childhood antisocial behavior

Childhood antisocial behavior	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6		Model 7		Model 8	
	b	SE	b	SE	b	SE	b	SE	b	SE	b	SE	b	SE	b	SE
	DF analysis components															
Shared environment	.00	.10	.00	.10	.00	.10	.00	.10	.00	.10	.00	.10	.00	.10	.00	.10
Heritability	.83**	.13	.80**	.13	.83**	.13	.84**	.13	.83**	.13	.81**	.13	.82**	.13	.83**	.13
Nonshared sources of variance																
Poor quality nutrition			.03**	.01	–	–	–	–	–	–	–	–	–	–	–	–
Low vegetable consumption			–	–	.02	.02	–	–	–	–	–	–	–	–	–	–
Low fruit consumption			–	–	–	–	.03*	.01	–	–	–	–	–	–	–	–
High fast food consumption			–	–	–	–	–	–	.06*	.02	–	–	–	–	–	–
High sweets consumption			–	–	–	–	–	–	–	–	.10**	.03	–	–	–	–
High salty snack consumption			–	–	–	–	–	–	–	–	–	–	.05*	.02	–	–
High soda consumption			–	–	–	–	–	–	–	–	–	–	–	–	.01	.02
N	760		758		760		758		760		760		760		760	
R ²	.25		.27		.25		.25		.25		.26		.25		.25	

* p < .05.
** p < .01.

models suggests that a) at least some nutritional factors significantly impact childhood antisocial behavior and b) composite scores on poor quality nutrition across dietary dimensions is consistently related to childhood antisocial behavior.⁹

Discussion and conclusion

Among the studies that have examined the origins of childhood behavioral problems, relatively few of them have considered nutritional factors as predictors of behavioral problems (Galler et al., 2011; Oh et al., 2013; Woo et al., 2014). Furthermore, none of these studies have fully explored the possibility that the association between poor nutrition and poor behavior during childhood may be overestimated and/or misleading due to unmeasured familial and genetic confounding. The present study addressed this void in the literature by employing a genetically informative, twin-based research design to examine whether twin differences in dietary practices during early childhood are predictive of twin differences in subsequent conduct problems.

The overall pattern of results in the current study suggests that poor quality nutrition during the preschool years may indeed increase the likelihood of behavioral problems once children enter elementary school. In general, twins with poorer overall eating habits during early childhood, across six dietary dimensions, subsequently exhibited significantly greater behavioral problems, relative to their co-twin. Although the statistical significance of specific dietary dimensions varied slightly across models, the results across multiple models reveal that the general

finding is robust to preexisting behavioral problems. Ultimately, the current study builds upon the body of literature linking nutritional factors to conduct problems, particularly in its examination of nutrition at an early stage of the life course and through its use of a genetically informative sibling design.

Despite the strengths of the current study in addressing the issues of familial and genetic confounding, it is not without its limitations. First, due to the use of the DF modeling strategy, the analytical sample of the present study only consisted of MZ and DZ twins.¹⁰ As a result, the current findings may not be generalizable to the singleton population. There is little reasons to believe, however, that the hypothesized relationship between poor quality nutrition and behavioral problems would operate any differently in the twin population than the singleton population. Furthermore, there are numerous strengths in the current research design that other designs lack, including the ability to account for genetic and shared environmental factors when testing the link between nutritional factors and antisocial behavior. Prior research has also revealed that studies using nationally representative samples of twins tend to be more generalizable to the singleton population than frequently assumed (for an example, see Barnes & Boutwell, 2013).

Second, the measures of early childhood nutrition were somewhat limited in their scope and specificity. That is, when examining the influence of early childhood nutrition on childhood antisocial behavior, it would have been worthwhile to also test hypotheses using measures that tap specific nutrients (e.g., omega-3 fatty acids, iron), but such measures were not available in the data. Some of the recent nutrition

Table 3
Does the effect of poor quality nutrition on childhood antisocial behavior persist independent of stability in antisocial behavior?

Childhood antisocial behavior	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6		Model 7	
	b	SE	b	SE	b	SE	b	SE	b	SE	b	SE	b	SE
	Shared environment	.16	.10	.15	.10	.14	.10	.14	.10	.15	.10	.14	.10	.14
Heritability	.64**	.13	.66**	.13	.67**	.13	.67**	.13	.65**	.13	.67**	.13	.67**	.13
Poor quality nutrition	.03**	.01	–	–	–	–	–	–	–	–	–	–	–	–
Low vegetable consumption	–	–	.04**	.02	–	–	–	–	–	–	–	–	–	–
Low fruit consumption	–	–	–	–	.04**	.01	–	–	–	–	–	–	–	–
High fast food consumption	–	–	–	–	–	–	.03	.02	–	–	–	–	–	–
High sweets consumption	–	–	–	–	–	–	–	–	.07**	.03	–	–	–	–
High salty snack consumption	–	–	–	–	–	–	–	–	–	–	.03	.02	–	–
High soda consumption	–	–	–	–	–	–	–	–	–	–	–	–	.01	.02
Antisocial behavior (W3)	.30**	.03	.31**	.03	.31**	.03	.30**	.03	.29**	.03	.30**	.03	.30**	.03
N	758		760		758		760		760		760		760	
R ²	.37		.36		.36		.35		.36		.35		.35	

** p < .01.

literature utilizes very precise measures of nutrient intake, such as omega-3 blood levels (Gow et al., 2013). Nevertheless, while such a measure would be preferable at the molecular and/or neurological level, more generalized measures of poor diet are useful in highlighting the specific dietary changes that might be made on a daily basis to minimize behavioral problems (see Park et al., 2012; Oh et al., 2013).

Finally, although the ECLS-B contains a large sample of twins, and is fairly comprehensive in scope, subjects were only followed until they were approximately 6 years of age. From a criminological perspective, it would have been informative to follow these children into adolescence and adulthood in order to examine their behavioral trajectories. The data provide no means of explicitly testing for the development of *criminal* behavior, despite the wealth of data relating to the development of *antisocial* behavior during childhood. Nevertheless, because the current study employs data that a) are quite thorough in their coverage of early childhood behaviors and processes and b) include a large sample of twins, it provides a good starting point upon which future research can build. It would certainly be worthwhile and informative for future studies to examine the applicability of this finding to antisocial and/or delinquent behavior at subsequent life stages, especially considering the body of research detecting a robust association between childhood antisocial behavior and subsequent offending patterns (Broidy et al., 2003; Campbell et al., 2000; Fergusson et al., 2014; Fergusson & Horwood, 1995; Nagin & Tremblay, 1999; Nagin & Tremblay, 2001).

The findings of the current study are generally consistent with the efforts of nutritionists worldwide to encourage healthy eating among the global population. Moreover, the results of the current study give even more reason to believe that the link between poor nutrition and behavioral problems may actually be causal, as results were robust to both shared environmental and genetic factors. Ultimately, the findings imply that interventions aimed at preventing and/or minimizing antisocial behavior will likely prove more successful if they a) are implemented during earlier stages of the life course and b) take nutritional factors into account. This is not to say that nutritional interventions during later stages of the life course are ineffective or fruitless (see Gesch et al., 2002; Zaalberg et al., 2010), or that nutritional factors are the only risk factors that should be targeted during childhood. However, the results of the current study suggest that comprehensive prevention efforts would likely benefit from incorporating nutritional components at the earliest stages of life (see Raine, Mellingen, Liu, Venables, & Mednick, 2003), as these components may help to reduce early-onset antisocial behavior and, in turn, may lower the risk of crime and delinquency. Of course, prevention efforts will not always be available and/or effective, and so some children will still end up on an antisocial path regardless, making treatment programs for adolescents and adults indispensable. Notwithstanding, the benefits of intervening before a delinquent trajectory is in full swing should not be understated. Ultimately, the findings of the current study bolsters the argument that, when it comes to biosocial risk factors such as nutrition, “early intervention is of paramount importance” (Rocque, Welsh, & Raine, 2012, p. 311).

Appendix A. Nutrition Items (Wave 3)

1. CH043: During the past 7 days, how many times did your child drink Soda pop (for example, Coke, Pepsi, or Mountain Dew), sports drinks (for example, Gatorade), or fruit drinks that are not 100% fruit juice (for example, Kool-Aid, Sunny Delight, Hi-C, Fruitopia, or Fruitworks)?
2. CH044: During the past 7 days, how many times did your child eat fresh fruit, such as apples, bananas, oranges, berries or other fruit such as applesauce, canned peaches, canned fruit cocktail, frozen berries, or dried fruit? Do not count fruit juice.
3. CH045: During the past 7 days, how many times did your child eat vegetables other than French fries and other fried potatoes? Include vegetables like those served as a stir fry, soup, or stew, in your response.

4. CH046: During the past 7 days, how many times did your child eat a meal or snack from a fast food restaurant with no wait service such as McDonald's, Pizza Hut, Burger King, Kentucky Fried Chicken, Taco Bell, Wendy's and so on? Consider both eating out, carry out, and delivery of meals in your response.
5. CH047: During the past 7 days, how many times did your child eat candy (including Fruit Roll-Ups and similar items), ice cream, cookies, cakes, brownies, or other sweets?
6. CH048: During the past 7 days, how many times did your child eat potato chips, corn chips such as Fritos or Doritos, Cheetos, pretzels, popcorn, crackers or other salty snack foods?

Notes

¹ Only same-sex twin pairs were evaluated to determine their zygosity (i.e., whether they were monozygotic or dizygotic twins), since opposite-sex twins are always dizygotic. Both parents and independent raters were asked to evaluate the similarity of the twins on several indicators (i.e., hair texture, eyes color, complexion, ear lobes, etc.). Furthermore, the blood type and Rh factors of each twin were ascertained through parental report. The process used to determine zygosity has been widely used, as it is highly reliable and valid (Goldsmith, 1991).

² Importantly, final sample sizes of specific models will vary contingent on the measures included in each model and their degree of missingness.

³ About 5% of the same-sex twin sample repeated kindergarten. However, because this did not significantly impact antisocial behavior in our sample, it was not included as a covariate in the analyses. Only a handful of twin pairs differed in their timing of entry into kindergarten. Although this predicted significant differences in antisocial behavior ($b = .49, p = .02$), such differences did not change the results of the present study in any substantive way.

⁴ For exact question wording for each of the six dietary components, see Appendix A.

⁵ Additionally, results showed no substantive variation using the alternative approach of single entering the data.

⁶ Ancillary analyses revealed that approximately 63% of twins within the same household varied in at least one dimension of their diet at age 4.

⁷ Analyses were also conducted using only parent reports of antisocial behavior at wave 4/5 (controlling for prior antisocial behavior). The results were very similar, as poor quality nutrition, low fruit consumption, sweets consumption, and salty snack consumption all predicted childhood antisocial behavior, net of the shared environment, genetic factors, and prior antisocial behavior. Low vegetable consumption also approached statistical significance ($p = .06$).

⁸ It is possible that fast food and salty snack consumption may no longer be significant in these models due to an evocative rGE. For example, infants and children with genetic predispositions toward a challenging temperament might exasperate parents in their efforts to encourage healthy eating, which may result in child-specific parental permissiveness when it comes to these types of junk foods.

⁹ MZ difference score analyses are sometimes conducted to examine the robustness of the DF results. In the current study, this method was of limited utility due to large reductions in the sample size, diminished statistical power, and reduced variation between MZ twins in eating behaviors. Nevertheless, differences in the composite diet of MZ twins were associated with differences in their antisocial behavior scores, and these differences approached statistical significance ($p = .07$).

¹⁰ Twin studies rest on certain assumptions, such as no assortative mating and the equal environments assumption. However, recent criminological research suggests that, even when/if such assumptions are violated, the findings of twin studies are not invalidated as a result (see Barnes, Wright, et al., 2014b).

References

- Baker, L. A., Jacobson, K. C., Raine, A., Lozano, D. I., & Bezdjian, S. (2007). Genetic and environmental bases of childhood antisocial behavior: A multi-informant twin study. *Journal of Abnormal Psychology, 116*(2), 219.
- Barnes, J. C., & Boutwell, B. B. (2013). A demonstration of the generalizability of twin-based research on antisocial behavior. *Behavior Genetics, 43*(2), 120–131.
- Barnes, J. C., Boutwell, B. B., Beaver, K. M., & Gibson, C. L. (2013). Analyzing the origins of childhood externalizing behavioral problems. *Developmental Psychology, 49*(12), 2272.
- Barnes, J. C., Boutwell, B. B., Beaver, K. M., Gibson, C. L., & Wright, J. P. (2014a). On the consequences of ignoring genetic influences in criminological research. *Journal of Criminal Justice, 42*(6), 471–482.
- Barnes, J. C., Wright, J. P., Boutwell, B. B., Schwartz, J. A., Connolly, E. J., Nedelec, J. L., & Beaver, K. M. (2014b). Demonstrating the validity of twin research in criminology. *Criminology, 52*(4), 588–626.
- Beaver, K. M., Schutt, J. E., Boutwell, B. B., Ratchford, M., Roberts, K., & Barnes, J. C. (2009). Genetic and environmental influences on levels of self-control and delinquent peer affiliation results from a longitudinal sample of adolescent twins. *Criminal Justice and Behavior, 36*(1), 41–60.
- Bellisle, F. (2004). Effects of diet on behaviour and cognition in children. *British Journal of Nutrition, 92*(S2), S227–S232.

- Benton, D. (2008). The influence of children's diet on their cognition and behavior. *European Journal of Nutrition*, 47(3), 25–37.
- Black, M. M. (2008). Effects of vitamin B12 and folate deficiency on brain development in children. *Food and Nutrition Bulletin*, 29(2 Suppl.), S126.
- Boutwell, B. B., Franklin, C. A., Barnes, J. C., & Beaver, K. M. (2011). Physical punishment and childhood aggression: The role of gender and gene–environment interplay. *Aggressive Behavior*, 37(6), 559–578.
- Broidy, L. M., Nagin, D. S., Tremblay, R. E., Bates, J. E., Brame, B., Dodge, K. A., ... Vitaro, F. (2003). Developmental trajectories of childhood disruptive behaviors and adolescent delinquency: A six-site, cross-national study. *Developmental Psychology*, 39(2), 222.
- Campbell, S. B., Shaw, D. S., & Gilliom, M. (2000). Early externalizing behavior problems: Toddlers and preschoolers at risk for later maladjustment. *Development and Psychopathology*, 12(03), 467–488.
- Connolly, E. J., & Beaver, K. M. (2015). Prenatal caloric intake and the development of academic achievement among US children from ages 5 to 14. *Child Development*, 86(6), 1738–1758.
- DeFries, J. C., & Fulker, D. W. (1985). Multiple regression analysis of twin data. *Behavior Genetics*, 15(5), 467–473.
- DeFries, J. C., & Fulker, D. W. (1988). Multiple regression analysis of twin data: Etiology of deviant scores versus individual differences. *Acta Geneticae Medicae et Gemellologiae: Twin Research*, 37(3–4), 205–216.
- D'Onofrio, B. M., Lahey, B. B., Turkheimer, E., & Lichtenstein, P. (2013). Critical need for family-based, quasi-experimental designs in integrating genetic and social science research. *American Journal of Public Health*, 103(S1), S46–S55.
- Espy, K. A., Sheffield, T. D., Wiebe, S. A., Clark, C. A., & Moehr, M. J. (2011). Executive control and dimensions of problem behaviors in preschool children. *Journal of Child Psychology and Psychiatry*, 52(1), 33–46.
- Faith, M. S., Berkowitz, R. I., Stallings, V. A., Kerns, J., Storey, M., & Stunkard, A. J. (2006). Eating in the absence of hunger: A genetic marker for childhood obesity in prepubertal boys? *Obesity*, 14(1), 131–138.
- Fergusson, D. M., & Horwood, L. J. (1995). Early disruptive behavior, IQ, and later school achievement and delinquent behavior. *Journal of Abnormal Child Psychology*, 23(2), 183–199.
- Fergusson, D. M., Boden, J. M., & Horwood, L. J. (2014). Bullying in childhood, externalizing behaviors, and adult offending: Evidence from a 30-year study. *Journal of School Violence*, 13(1), 146–164.
- Galler, J. R., Bryce, C. P., Waber, D. P., Medford, G., Eaglesfield, G. D., & Fitzmaurice, G. (2011). Early malnutrition predicts parent reports of externalizing behaviors at ages 9–17. *Nutritional Neuroscience*, 14(4), 138–144.
- Georgieff, M. K. (2007). Nutrition and the developing brain: Nutrient priorities and measurement. *The American Journal of Clinical Nutrition*, 85(2), 614S–620S.
- Gesch, C. B., Hammond, S. M., Hampson, S. E., Eves, A., & Crowder, M. J. (2002). Influence of supplementary vitamins, minerals and essential fatty acids on the antisocial behaviour of young adult prisoners: Randomised, placebo-controlled trial. *The British Journal of Psychiatry*, 181(1), 22–28.
- Goldsmith, H. (1991). A zygosity questionnaire for young twins: A research note. *Behavior Genetics*, 21(3), 257–269.
- Gómez-Pinilla, F. (2008). Brain foods: The effects of nutrients on brain function. *Nature Reviews Neuroscience*, 9(7), 568–578.
- Gow, R. V., Vallee-Tourangeau, F., Crawford, M. A., Taylor, E., Ghebremeskel, K., Bueno, A. A., ... Rubia, K. (2013). Omega-3 fatty acids are inversely related to callous and unemotional traits in adolescent boys with attention deficit hyperactivity disorder. *Prostaglandins, Leukotrienes and Essential Fatty Acids (PLEFA)*, 88(6), 411–418.
- Haynie, D. L., & McHugh, S. (2003). Sibling deviance: In the shadows of mutual and unique friendship effects? *Criminology*, 41(2), 355–392.
- Hibbeln, J. R., Ferguson, T. A., & Blasbalg, T. L. (2006). Omega-3 fatty acid deficiencies in neurodevelopment, aggression and autonomic dysregulation: Opportunities for intervention. *International Review of Psychiatry*, 18(2), 107–118.
- Howard, A. L., Robinson, M., Smith, G. J., Ambrosini, G. L., Piek, J. P., & Oddy, W. H. (2011). ADHD is associated with a “Western” dietary pattern in adolescents. *Journal of Attention Disorders*, 15(5), 403–411.
- Jackson, D. B., & Beaver, K. M. (2013). The influence of neuropsychological deficits in early childhood on low self-control and misconduct through early adolescence. *Journal of Criminal Justice*, 41(4), 243–251.
- Jaffee, S. R., Strait, L. B., & Odgers, C. L. (2012). From correlates to causes: Can quasi-experimental studies and statistical innovations bring us closer to identifying the causes of antisocial behavior? *Psychological Bulletin*, 138(2), 272.
- Kokko, K., Tremblay, R. E., Lacourse, E., Nagin, D. S., & Vitaro, F. (2006). Trajectories of prosocial behavior and physical aggression in middle childhood: Links to adolescent school dropout and physical violence. *Journal of Research on Adolescence*, 16(3), 403–428.
- Liu, J., & Raine, A. (2006). The effect of childhood malnutrition on externalizing behavior. *Current Opinion in Pediatrics*, 18(5), 565–570.
- Liu, J., Raine, A., Venables, P. H., & Mednick, S. A. (2004). Malnutrition at age 3 years and externalizing behavior problems at ages 8, 11, and 17 years. *American Journal of Psychiatry*, 161(11), 2005–2013.
- Liu, J., Hanlon, A., Ma, C., Zhao, S. R., Cao, S., & Compher, C. (2014). Low blood zinc, iron, and other sociodemographic factors associated with behavior problems in preschoolers. *Nutrients*, 6(2), 530–545.
- Merrell, K. W. (2003). *Preschool and kindergarten behavior scales* (2nd edition). Austin, TX: Pro-Ed.
- Moffitt, T. E., Caspi, A., Dickson, N., Silva, P., & Stanton, W. (1996). Childhood-onset versus adolescent-onset antisocial conduct problems in males: Natural history from ages 3 to 18 years. *Development and Psychopathology*, 8(02), 399–424.
- Molteni, R., Barnard, R. J., Ying, Z., Roberts, C. K., & Gómez-Pinilla, F. (2002). A high-fat, refined sugar diet reduces hippocampal brain-derived neurotrophic factor, neuronal plasticity, and learning. *Neuroscience*, 112(4), 803–814.
- Nagin, D., & Tremblay, R. E. (1999). Trajectories of boys' physical aggression, opposition, and hyperactivity on the path to physically violent and nonviolent juvenile delinquency. *Child Development*, 70(5), 1181–1196.
- Nagin, D. S., & Tremblay, R. E. (2001). Parental and early childhood predictors of persistent physical aggression in boys from kindergarten to high school. *Archives of General Psychiatry*, 58(4), 389–394.
- Oddy, W. H., Robinson, M., Ambrosini, G. L., de Klerk, N. H., Beilin, L. J., Silburn, S. R., ... Stanley, F. J. (2009). The association between dietary patterns and mental health in early adolescence. *Preventive Medicine*, 49(1), 39–44.
- Oh, S. Y., Ahn, H., Chang, N., Kang, M. H., & V Oh, J. (2013). Dietary patterns and weight status associated with behavioural problems in young children. *Public Health Nutrition*, 1–7.
- Park, S., Cho, S. C., Hong, Y. C., Oh, S. Y., Kim, J. W., Shin, M. S., ... Bhang, S. Y. (2012). Association between dietary behaviors and attention-deficit/hyperactivity disorder and learning disabilities in school-aged children. *Psychiatry Research*, 198(3), 468–476.
- Peters, J., Dollman, J., Petkov, J., & Parletta, N. (2013). Associations between parenting styles and nutrition knowledge and 2–5-year-old children's fruit, vegetable and non-core food consumption. *Public Health Nutrition*, 16(11), 1979–1987.
- Pingault, J. B., Côté, S. M., Galéra, C., Genolini, C., Falissard, B., Vitaro, F., & Tremblay, R. E. (2012). Childhood trajectories of inattention, hyperactivity and oppositional behaviors and prediction of substance abuse/dependence: A 15-year longitudinal population-based study. *Molecular Psychiatry*, 18(7), 806–812.
- Piquero, A. R., Carriaga, M. L., Diamond, B., Kazemian, L., & Farrington, D. P. (2012). Stability in aggression revisited. *Aggression and Violent Behavior*, 17(4), 365–372.
- Raine, A., Mellinger, K., Liu, J., Venables, P., & Mednick, S. A. (2003). Effects of environmental enrichment at ages 3–5 years on schizotypal personality and antisocial behavior at ages 17 and 23 years. *American Journal of Psychiatry*, 160, 1627–1635.
- Raine, A., Portnoy, J., Liu, J., Mahomed, T., & Hibbeln, J. R. (2015). Reduction in behavior problems with omega-3 supplementation in children aged 8–16 years: A randomized, double-blind, placebo-controlled, stratified, parallel-group trial. *Journal of Child Psychology and Psychiatry*, 56(5), 509–520.
- Riggs, N. R., Blair, C. B., & Greenberg, M. T. (2004). Concurrent and 2-year longitudinal relations between executive function and the behavior of 1st and 2nd grade children. *Child Neuropsychology*, 9(4), 267–276.
- Rocque, M., Welsh, B. C., & Raine, A. (2012). Biosocial criminology and modern crime prevention. *Journal of Criminal Justice*, 40(4), 306–312.
- Rodgers, J. L., & Kohler, H. P. (2005). Reformulating and simplifying the DF analysis model. *Behavior Genetics*, 35(2), 211–217.
- Rodgers, J. L., Buster, M., & Rowe, D. C. (2001). Genetic and environmental influences on delinquency: DF analysis of NLSY kinship data. *Journal of Quantitative Criminology*, 17(2), 145–168.
- Rodgers, J. L., Rowe, D. C., & Li, C. (1994). Beyond nature versus nurture: DF analysis of nonshared influences on problem behaviors. *Developmental Psychology*, 30(3), 374.
- Schaeffer, C. M., Petras, H., Ialongo, N., Poduska, J., & Kellam, S. (2003). Modeling growth in boys' aggressive behavior across elementary school: Links to later criminal involvement, conduct disorder, and antisocial personality disorder. *Developmental Psychology*, 39(6), 1020.
- Schoemaker, K., Mulder, H., Deković, M., & Matthys, W. (2013). Executive functions in preschool children with externalizing behavior problems: A meta-analysis. *Journal of Abnormal Child Psychology*, 41(3), 457–471.
- Schoenthaler, S. J., & Bier, I. D. (2000). The effect of vitamin–mineral supplementation on juvenile delinquency among American schoolchildren: A randomized, double-blind placebo-controlled trial. *The Journal of Alternative and Complementary Medicine*, 6(1), 7–17.
- Schoenthaler, S., Amos, S., Doraz, W., Kelly, M. A., Muedeking, G., & Wakefield, J. (1997). The effect of randomized vitamin–mineral supplementation on violent and non-violent antisocial behavior among incarcerated juveniles. *Journal of Nutritional & Environmental Medicine*, 7(4), 343–352.
- Shenassa, E. D., Paradis, A. D., Dolan, S. L., Wilhelm, C. S., & Buka, S. L. (2012). Childhood impulsive behavior and problem gambling by adulthood: A 30-year prospective community-based study. *Addiction*, 107(1), 160–168.
- Sinn, N. (2008). Nutritional and dietary influences on attention deficit hyperactivity disorder. *Nutrition Reviews*, 66(10), 558–568.
- Tholin, S., Rasmussen, F., Tynelius, P., & Karlsson, J. (2005). Genetic and environmental influences on eating behavior: The Swedish Young Male Twins Study. *The American Journal of Clinical Nutrition*, 81(3), 564–569.
- Thompson, R., Tabone, J. K., Litrownik, A. J., Briggs, E. C., Hussey, J. M., English, D. J., & Dubowitz, H. (2010). Early adolescent risk behavior outcomes of childhood externalizing behavioral trajectories. *Journal of Early Adolescence*, 31(2), 234–257.
- Timmermans, M., Van Lier, P. A., & Koot, H. M. (2008). Which forms of child/adolescent externalizing behaviors account for late adolescent risky sexual behavior and substance use? *Journal of Child Psychology and Psychiatry*, 49(4), 386–394.
- Tremblay, R. E., Pihl, R. O., Vitaro, F., & Dobkin, P. L. (1994). Predicting early onset of male antisocial behavior from preschool behavior. *Archives of General Psychiatry*, 51(9), 732–739.
- Woo, H. D., Kim, D. W., Hong, Y. S., Kim, Y. M., Seo, J. H., Choe, B. M., ... Kim, J. (2014). Dietary patterns in children with attention deficit/hyperactivity disorder (ADHD). *Nutrients*, 6(4), 1539–1553.
- Zaalberg, A., Nijman, H., Bulten, E., Stroosma, L., & van der Staak, C. (2010). Effects of nutritional supplements on aggression, rule-breaking, and psychopathology among young adult prisoners. *Aggressive Behavior*, 36(2), 117–126.