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Three dopaminergic polymorphisms are associated with academic achievement in middle and high school

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ABSTRACT

Although academic achievement is a heritable construct, to date research has yet to explore its molecular genetic underpinnings. Drawing on data from the National Longitudinal Study of Adolescent Health, the current longitudinal study investigated the associations between polymorphisms in three dopaminergic genes (DAT1, DRD2, and DRD4) and academic achievement during middle and high school (Ns ranged between 622 and 2181). Findings revealed statistically significant associations between the three dopaminergic polymorphisms and a composite genetic risk index with English, math, history, and science grades.

1. Introduction

A substantial body of empirical research has examined the role academic achievement has on later-life outcomes. The results of these studies have unequivocally revealed that poor academic achievement in high school is associated with a range of antisocial behaviors, including increased drug use, alcohol abuse, and delinquent involvement (Maguin & Loeber, 1996; Schulenberg, Bachman, O'Malley, & Johnston, 1994). In contrast, students who perform well at school have an increased probability of attending college, of being socially and emotionally adjusted, and of earning a relatively high annual salary (Ganderton & Santos, 1995; Herrnstein & Murray, 1994; Steinberg, 1996). Given the robust link between high school educational achievement and positive and negative outcomes, there has been a significant amount of interest in unpacking the factors that promote academic performance. Much of this research has focused on examining the effects of environmental factors, such as family social class and school poverty status (Mau, 1997; Rivkin, Hanushek, & Kain, 2005; Sun & Li, 2009), or individual-level factors, such as motivation and intelligence (Deary, Strand, Smith, & Fernandes, 2007; Mitchell, 1992; Rohde & Thompson, 2007; Watkins, Lei, & Canivez, 2007). Although the evidence garnered from these inquiries has provided much needed insight into the potential environmental- and individual-level causes of educational achievement, comparatively less is known about the genetic underpinnings to academic performance during middle and high school.

2. Behavioral genetics and academic performance

A number of behavioral genetic studies have been conducted to estimate the relative effects of genetic, shared environmental, and nonshared environmental influences on variation in measures of high school academic achievement.

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Results from these studies have pointed to the importance of both genetic and environmental contributors to school performance (Devlin, Daniels, & Roeder, 1997; Plomin & Kovas, 2005; Plomin & Petrill, 1997). For example, Johnson, McGue, and Iacono (2006) analyzed a sample of twins drawn from the Minnesota Twin Family study to examine genetic and environmental influences on academic achievement as indexed by grades. They estimated separate statistical models for girls and boys at the ages of 11, 12, 13, and 14. Although the heritability estimates varied between genders and over time, the heritability estimates tended to converge around .50, meaning that approximately 50% of the variance in academic achievement was explainable by genetic factors and 50% was the result of environmental factors (and error). Importantly, other studies that have employed different samples and different measures of academic achievement and performance have also reported a significant genetic effect (Van Den Oord & Rowe, 1998; Wainright, Wright, Geffen, Luciano, & Martin, 2005).

With the available behavioral genetic research suggesting that academic achievement is influenced by genetic factors, the next step is to examine the influence candidate genes may have on academic performance (Plomin, 2003). Trying to identify which genes are linked to school achievement and performance is a difficult enterprise because academic achievement and cognitive performance represent polygenic phenotypes, where multiple gene systems are involved and where single genes likely have relatively small effects. The problem of identifying candidate genes for educational achievement is compounded by the fact that the genes do not have direct effects on educational achievement, but rather are mediated by endophenotypes, such as working memory, processing speed, and selective attention (van Leeuwen, van den Berg, Hoekstra, & Boomsma, 2007). As a result, the genes that are most likely to be associated with educational achievement are those that have been found to affect endophenotypes previously linked to educational achievement.

3. The dopaminergic system and academic performance

The dopaminergic system, due to its widespread effects on learning, memory, motivation, and reward, is theoretically useful in understanding educational performance. The extant literature provides at least three reasons to suspect that genes from the dopaminergic system are promising candidate genes that are partially responsible for producing variation in educational achievement and performance. First, a wealth of research has revealed that there is a strong positive relationship between scores on intelligence tests and achievement in school (Deary et al., 2007; Rohde & Thompson, 2007; Watkins et al., 2007). In addition, intelligence tends to be one of the most heritable of all human phenotypes (Plomin, 1999, 2003; Plomin & Spinath, 2002, 2004), and there is emerging evidence that the dopaminergic system, including certain dopaminergic genes, is partially responsible for creating variation in intelligence and cognitive abilities (Mill et al., 2006; Previc, 1999; Qiang et al., 2010). For example, Berman and Noble (1995) reported that the A1 allele of the DRD2 gene was related to reduced visuospatial performance in children. This study, along with others (Mill et al., 2006; Qiang et al., 2010), suggests that dopaminergic genes might have an effect on academic performance via their effects on intelligence and motivation. Nonetheless, it is important to point out that studies examining the association between dopaminergic polymorphisms and cognitive abilities and intelligence often fail to detect a statistically significant effect (e.g., Ball et al., 1998; Moises et al., 2001; Petrill, Plomin, McClearn, Smith, Vignetti et al., 1997).

Second, existing research findings implicate the dopaminergic system in learning and memory formation (Dayan & Balleine, 2002; Klein et al., 2007; Wise, 2004). For instance, Jocham et al. (2009) reported that the A1 allele was associated with impaired scores on a reversal learning task. Other dopaminergic genes, such as the DRD4 gene, moreover, have been shown to have effects on memory formation and learning processes (Herrmann et al., 2007). Taken together, the available evidence suggests that dopaminergic genes may have an effect on academic performance because of the effects that these genes have on the capacity for learning and on retaining newly acquired information.

The third reason why dopaminergic genes are hypothesized to be related with educational achievement and performance is because of their association with various psychopathologies, especially antisocial behaviors. A significant amount of developmental research has revealed that youths who engage in antisocial behavior or who have problems with impulse control and self-regulation are at very high risk for poor school performance, including dropping out of school, being suspended from school, and being expelled from school (Biederman, Monuteaux, Doyle, Seidman, Wilens et al., 2004; Cairns & Cairns, 1994; Maguin & Loeber, 1996). Available research reveals that the association between psychopathology and educational performance could be driven, in part, by genes from the dopaminergic system. For instance, several studies have reported that polymorphisms in certain dopaminergic genes, such as DAT1, DRD2, and DRD4, confer an increased risk to violence, aggression, and other forms of adolescent delinquency (Beaver, 2008a; DeLisi, Beaver, Vaughn, & Wright, 2009; Guo, Roettger, & Shih, 2007; Vaughn, DeLisi, Beaver, & Wright, 2009), as well as problems with impulse control and self-regulation (Faraone, Doyle, Mick, & Biederman, 2001; Sagvolden, Johansen, Aase, & Russell, 2005), and with binge drinking (Vaughn, Beaver, DeLisi, Howard, & Perron, 2009). Consequentially, genes from the dopaminergic system could be involved in school performance by way of the indirect effects that these genes have on various psychopathologies.

The evidence presented above suggests that a number of dopaminergic genes may be associated with endophenotypes that have previously been linked to academic performance and achievement. As a result, the current study examines the association between polymorphisms in the DAT1, DRD2, and DRD4 genes and measures of academic performance drawn from two time periods during adolescence. Additionally, since academic performance is a multifactorial phenotype, and since the dopaminergic genes should have only small effects on this phenotype, the current study examined the cumulative effect of these genes. To do so, a global dopaminergic index was employed that was simultaneously able to examine the effects of all three genetic polymorphisms on educational success. All of the analyses are carried out by analyzing data drawn from a large sample of American youths.

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4. Method

4.1. Participants

Data for this study come from the National Longitudinal Study of Adolescent Health (Add Health; Udry, 2003). The Add Health is a longitudinal, prospective, and nationally representative sample of American youths enrolled in middle or high school. Initial data collection efforts began during the 1994–1995 school year when more than 90,000 adolescents completed a self-report survey at school, known as the wave 1 in-school component of the Add Health study. The wave 1 in-school surveys asked youths about a range of topics, including their family relationships, their peer networks, and their demographic characteristics. In order to gain more in-depth information about the youths, including information about sensitive topics, a subset of adolescents was then chosen to be reinterviewed in their homes along with their primary caregivers (usually their mother). These wave 1 in-home interviews included questions about the adolescent's performance at school, their involvement in delinquency, and their romantic relationships, among others. Altogether, 20,745 adolescents and approximately 17,700 of their primary caregivers participated in the wave 1 in-home interview (Harris et al., 2003).

The second round of data was collected about one to two years after the first wave of data was gathered. Most of the respondents were still adolescents and thus the survey instruments used at wave 2 were very similar to the ones used at wave 1. For example, adolescents were asked questions about their performance at school, their social relationships, and their involvement in delinquent behaviors. In total, 14,738 youths of the original in-home sample were reinterviewed at wave 1. The primary caregivers were not interviewed at wave 2. The third wave of data was collected between 2001 and 2002 when most of the respondents were young adults. Consequentially, the questions included on the survey instruments were amended to reflect more age-appropriate questions. Respondents, for example, were asked questions about their employment history, their marital status, and their financial well-being. Overall, 15,197 participants were included in the wave 3 component of the Add Health study (Harris et al., 2003).

At wave 3, a subsample of Add Health participants was asked to submit buccal cells to be genotyped. Only respondents who had a sibling who also was part of the Add Health study were eligible for inclusion in the DNA subsample. The DNA subsample was thus drawn from the sibling-pair sample. The average ages of the sibling-pair sample at the three waves were 16, 17, and 22 years old. Overall, approximately 52% of the sample (at wave 3) was male and approximately 68% of the sample was Caucasian. A total of more than 2500 subjects were genotyped for polymorphisms involved in neurotransmission. More detailed information about the DNA sample has been published elsewhere (Harris, Halpern, Smolen, & Haberstick, 2006) and, importantly, the genotypic data of the Add Health study have been analyzed previously in a wide range of studies (e.g., DeLisi et al., 2009; Guo et al., 2007; Haberstick et al., 2005; Hopfer et al., 2005).

4.2. Measures

4.2.1. Genotyping

Genotyping was conducted at wave 3 and was carried out by a joint effort between the Institute for Behavioral Genetics in Boulder, Colorado and Add Health. Three dopaminergic polymorphisms were chosen to be genotyped by Add Health researchers and were available for analysis. As a result, we included these three polymorphisms in the current analyses. First, a 40 base pair variable number of tandem repeats (VNTR) has been found in the 3′ untranslated region of the DAT1 gene (SLC6A3), with the number of repeats ranging between 3 and 11 copies. This VNTR was amplified by using the following primer sequences: forward, 5′-TGTGGTGTAGGGAACGGCCTGAG-3′ (fluorescently labeled), and reverse, 5′-CTTCCTGGAGGT-CACGGCTCAAGG-3′. This genotyping method produced PCR products of 320 (6-repeat allele), 360 (7-repeat allele), 400 (8-repeat allele), 440 (9-repeat allele), 480 (10-repeat allele), and 520 (11-repeat allele) base pairs.

The 9R allele and the 10R allele are the two most common alleles across racial groups (Doucette-Stamm, Blakely, Tian, Mockus, & Mao, 1995). As a result, and following the lead of other researchers analyzing the Add Health data, respondents who possessed alleles other than the 9R allele or the 10R allele were deleted from the final analytical sample (Hopfer et al., 2005). With this nomenclature in place, 4.8% of the sample was homozygous for the 9R allele, 33.8% of the sample was heterozygous for the 10R allele, and 61.4% of the sample was homozygous for the 10R allele. The 10R allele was coded as the risk allele. All of the genetic polymorphisms were coded dominantly, where participants who were homozygous or heterozygous for the risk allele were assigned a value of "1;" if they were homozygous for the other allele (i.e., the non-risk allele), then they were assigned a value of "0."

The dopamine D2 receptor gene (DRD2) has a TaqIA endonuclease site which is found in the 3′ untranslated region of the gene about 2500 base pairs downstream from the gene's coding region. This polymorphism was genotyped by using the Applied Biosystem's "Taqman© Assays by Design™ for SNP Genotyping Service" (Haberstick & Smolen, 2004). The TaqIA polymorphism was genotyped by using the following primers and probes: forward primer, 5′-GTGCACTCACTCCATCCT-3′, reverse primer, 5′-GCAACACAGCCATCCTCAAAG-3′, probe 1, 5′-VIC-CCTGCCT***T***GACCAGC-NFQMGB-3′, and probe 2, 5′-FAM-CTGCCT***C***GACCAGC-NFQMGB-3′ (Haberstick & Smolen, 2004). The T-probe signal corresponded to the TaqIA-1 allele and the C-probe corresponded to the TaqIA-2 allele. In the final analytical sample, 54.8% of the sample was homozygous for the A-2 allele, 37.3% of the sample was heterozygous for the A-1 allele, and 7.9% of the sample was homozygous for the A-1 allele. The A-1 allele was coded as the risk allele.

The dopamine D4 receptor gene (DRD4) has a 48 base pair VNTR located at 11p15.5 on exon III. DRD4 was amplified by using the following primer sequences: forward, 5′-AGGACCCT-CATGGCCTTG-3′ (fluorescently labeled), and reverse, 5′-GCGACTACGTGGTCTACTCG-3′. This assay produced PCR products of 379, 427, 475, 523, 571, 619, 667, 715, 763, and 811 base pairs. The two most frequently occurring alleles were the 4-repeat and the 7-repeat. In line with prior research (Hopfer et al., 2005), we grouped together the 379 (2R), 427 (3R), 475 (4R), 523 (5R), and 571 (6R) bp alleles and we grouped together the 619 (7R), 667 (8R), 715 (9R), and 763 (10R) bp alleles. Using this nomenclature, 62.3% of the sample was homozygous for the 4R allele, 32.7% of the sample was heterozygous for the 7R allele, and 5.0% of the sample was homozygous for the 7R allele. The 7R allele was coded as the risk allele.

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Last, we created a cumulative genetic index to examine whether the effects of the dopaminergic genes on academic performance had cumulative, rather than independent, effects. To do so, the dominant scores for DAT1, DRD2, and DRD4 were summed together to create the cumulative genetic index. The value on this index represented the total number of risk alleles, when measured homozygously, that each respondent possessed. Scores on this index ranged from 0 to 3, with a score of 0 indicating the participant was not homozygous for any putative risk alleles and a score of 3 indicating that the participant was homozygous for all three putative risk alleles. Overall, 1.4% of the sample received a score of 0 on the index, 34.8% of the sample received a score of 1 on the index, 48.0% of the sample received a score of 2 on the index, and 15.7% of the sample received a score of 3 on the index. This modeling strategy for creating a cumulative genetic index is similar to ones that have been used previously (Beaver, 2008a; Conner, Hellemann, Ritchie, & Noble, 2010; Harlaar et al., 2005).

4.2.2. Academic performance

To measure academic performance, five measures were created at wave 1 and the same five measures were created at wave 2. Specifically, during wave 1 interviews, adolescents were asked to report their grade (from their most recent grading period) in English or language arts, mathematics, history, and science. Responses for each of these questions were recoded to conform to the following response set: 1 = D, 2 = C, 3 = B, and 4 = A. Additionally, a grade point average (GPA) measure was created by summing the values for these four academic performance measures and then dividing by 4. The resulting value provided the GPA of the student at wave 1 during their most recent grading period ($\alpha = .75$). The same measures were available at wave 2, with individual grades being assessed for English or language arts, mathematics, history, and science. A wave 2 composite GPA measure was also created using an identical procedure that was used to create the wave 1 GPA measure ($\alpha = .74$). The stability coefficient between wave 1 and wave 2 for English was $r = .469$ ($P < .05$), for mathematics was $r = .442$ ($P < .05$), for history was $r = .473$ ($P < .05$), for science was $r = .416$, ($P < .05$) and for the composite GPA was $r = .696$ ($P < .05$). Table 1 provides descriptive statistics for all of the academic performance measures.

Table 1
Descriptive statistics for course grades and cumulative GPA at wave 1 and wave 2.

Variable	Mean	Median	Mode	SD	Min.–Max.
Wave 1					
English	2.88	3.00	3.00	0.95	1–4
Math	2.72	3.00	3.00	1.03	1–4
History	2.94	3.00	4.00	1.01	1–4
Science	2.87	3.00	4.00	1.02	1–4
GPA	2.88	3.00	3.25	0.75	1–4
Wave 2					
English	2.89	3.00	3.00	0.93	1–4
Math	2.77	3.00	3.00	1.02	1–4
History	2.94	3.00	4.00	1.00	1–4
Science	2.88	3.00	3.00	0.99	1–4
GPA	2.91	3.00	3.00	0.74	1–4

The decision to measure academic performance with individual grades for specific subjects along with a composite GPA scale was driven by two main factors. First, performance in different subjects may be the result of different etiological processes. Excelling in mathematics versus excelling in the English language, for example, is partially the result of different biological functions (Brizendine, 2006). Second, the intercorrelations among self-reported grades for the individual subjects ranged between $r = .337$ and $r = .495$ at wave 1 and between $r = .325$ and $r = .467$ at wave 2. All of these bivariate correlations reveal a significant degree of overlap, but they also reveal a substantial amount of nonshared variance. Similarly, the stability between the wave 1 composite GPA scale and the wave 2 composite GPA scale was $r = .696$. While the magnitude of the stability coefficient was strong, less than one-half of the variance in the wave 2 composite GPA scale was explained by the wave 1 composite GPA scale. Taken together, the most thorough way to examine the association between the dopaminergic genes and academic performance is to measure academic performance multiple ways. This is the approach that is employed in the current study.

4.2.3. Control variables

Two control variables were included in the analyses to help take into account confounding. First, gender was included as a dichotomous dummy variable, where 0 = female and 1 = male. Second, to take into account potential population stratification effects, race was also included as a dichotomous dummy variable, where 0 = Caucasian and 1 = minority. Overall, 52% of the sample was female and 67% reported their race as Caucasian.

4.3. Analytical strategy

The analysis was conducted in several steps. First, as an initial examination of the potential association between the dopaminergic polymorphisms and academic achievement, the means and standard deviations (SDs) were presented by genotypes (as measured dominantly). Second, the dopaminergic genetic polymorphisms were entered into a series of ordinary least squares (OLS) regression equations using the individual grades in English, math, history, and science measured at wave 1 as the dependent variables. Prior to estimating these models, we examined whether the association between the dopaminergic genes and the various measures of school performance appeared to be linear and additive. The results of these analyses indicated that these associations could be captured by an additive, linear model. We tested for non-linear interaction effects and the results of these models did not reveal any evidence of a non-linear association between the dopaminergic genes and academic achievement. Third, these same OLS equations were replicated with the exception that the wave 2 grades were employed as the dependent variables. Fourth, the total GPA at wave 1 was entered into an OLS equation as the outcome measure and the genetic index was introduced as a predictor variable. These models were estimated for the full sample and separately for males and females to explore potential gender differences. Fifth, these models were replicated using the wave 2 total GPA measure as the outcome measure. All statistical models were estimated by removing missing data via listwise deletion techniques.

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Table 2
Means and standard deviations by dopaminergic genotypes.

	DAT1		DRD2		DRD4	
	No 10R	≥1 10R	No A1	≥1 A1	No 7R	≥1 7R
Wave 1						
English						
Mean	3.06	2.88	2.93	2.82	2.93	2.80
SD	0.95	0.95	0.93	0.97	0.93	0.97
Math						
Mean	2.85	2.71	2.77	2.67	2.78	2.62
SD	1.01	1.03	1.03	1.03	1.01	1.05
History						
Mean	3.07	2.93	3.02	2.84	2.97	2.89
SD	1.02	1.01	0.98	1.03	1.00	1.02
Science						
Mean	2.90	2.86	2.93	2.78	2.90	2.82
SD	1.08	1.01	0.99	1.04	1.02	1.01
GPA						
Mean	3.03	2.87	2.95	2.80	2.92	2.81
SD	0.78	0.75	0.74	0.76	0.74	0.76
Wave 2						
English						
Mean	2.95	2.88	2.93	2.84	2.88	2.90
SD	0.93	0.93	0.92	0.94	0.93	0.92
Math						
Mean	2.78	2.77	2.81	2.71	2.80	2.72
SD	1.06	1.02	1.04	1.01	1.01	1.04
History						
Mean	3.02	2.94	2.97	2.91	2.98	2.88
SD	1.02	1.00	1.00	0.99	0.98	1.03
Science						
Mean	2.80	2.88	2.91	2.84	2.92	2.80
SD	1.09	0.99	1.01	0.97	0.99	0.99
GPA						
Mean	2.92	2.91	2.95	2.87	2.95	2.85
SD	0.83	0.74	0.76	0.73	0.73	0.76

Because some of the observations were not independently selected (e.g., more than one sibling per household was included in the sample), the assumption of independence that is necessary when estimating OLS models was not preserved. The violation of non-independence can produce biased tests of statistical significance for the regression coefficients. Two approaches were utilized to correct for non-independence. First, one monozygotic (MZ) twin from each MZ twin pair was randomly selected and removed from the analysis in situations where two monozygotic (MZ) twins from the same household were included in the sample (Haberstick et al., 2005). Second, all tests of statistical significance were calculated by using Huber/White standard errors which correct for the clustering of observations.

The final analytical sample size varied between waves and across the different measures of academic performance for two main reasons. First, students who graduated from high school between waves 1 and 2, or who were no longer attending school at wave 2, were excluded from the wave 2 statistical models (owing to missing data). Second, within each wave, not all students were uniformly enrolled in English, mathematics, history, and science. In other words, some students may not have been enrolled in a mathematics course during their most recent grading period. As a result, their grade in this subject was coded as missing and they were excluded from that particular statistical model. All of the tables/figures provide the exact sample size that was used in the calculation of each statistical model.

5. Results

Table 2 presents the means and SDs for the academic achievement measures across genotypes for the three dopaminergic polymorphisms. As this table shows, there appears to be an association between the genotypes and academic achievement, where respondents who possess at least one of the risk alleles tend to have lower grades when compared to respondents who did not possess a risk allele. These results provide initial evidence of an association between dopaminergic polymorphisms and academic achievement.

The next models estimated the effects that the three dopaminergic genes had on grades in English, math, history, and science gathered during wave 1 interviews (shown in Table 3). As can be seen, DAT1 had a marginally significant and negative effect on English, but was unrelated to grades in the three other academic subjects. DRD2 had a statistically significant and negative association with English grades, math grades, history grades, and science grades. Similar results were reported for DRD4 although the effects were attenuated. DRD4 was significantly associated with grades in English and math and marginally associated with grades in history and science. Importantly, the effect of race was statistically significant across all of the models, meaning that minorities self-reported lower grades when compared to Caucasians.

The results of the OLS models using the wave 2 grades appear in Table 4. In these models, DAT1 was unrelated to all of the grades, while DRD2 was marginally associated with English grades, but unrelated to grades in all of the other three subjects. Finally, DRD4 maintained a statistically significant and positive association with grades in history and science, but did not have a statistically significant relationship with grades in English or

Table 3
The association between dopaminergic polymorphisms and academic achievement in four specific subjects at wave 1.

	English			Math			History			Science		
	b	*Beta*	*P*	*b*	*Beta*	*P*	*b*	*Beta*	*P*	*b*	*Beta*	*P*
Genetic polymorphism												
DAT1	−.18	−.04	.051	−.14	−.03	.179	−.11	−.02	.303	−.03	−.01	.773
DRD2	−.09	−.05	.020	−.11	−.05	.018	−.13	−.07	.004	−.14	−.07	.003
DRD4	−.15	−.08	<.001	−.17	−.08	<.001	−.09	−.04	.052	−.09	−.04	.053
Control variables												
Gender	−.37	−.20	<.001	−.10	−.05	.023	−.24	−.12	<.001	−.28	−.14	<.001
Race	−.11	−.05	.010	−.14	−.06	.005	−.22	−.10	<.001	−.10	−.05	.035
N	2181			2096			1977			1995		

Note: All models were estimated using Huber/White standard errors.

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Table 4
The association between dopaminergic polymorphisms and academic achievement in four specific subjects at wave 2.

	English			Math			History			Science		
	b	*Beta*	*P*	*b*	*Beta*	*P*	*b*	*Beta*	*P*	*b*	*Beta*	*P*
Genetic polymorphism												
DAT1	−.04	−.01	.665	.03	.01	.805	−.08	−.02	.464	.10	.02	.407
DRD2	−.07	−.04	.096	−.07	−.04	.139	−.04	−.02	.457	−.05	−.02	.359
DRD4	.03	.01	.558	−.07	−.03	.163	−.11	−.05	.035	−.12	−.06	.016
Control variables												
Gender	−.35	−.19	<.001	−.17	−.08	<.001	−.20	−.10	<.001	−.13	−.07	.009
Race	−.14	−.07	.003	−.23	−.11	<.001	−.10	−.05	<.072	−.23	−.11	<.001
N	1834			1682			1608			1563		

Note: All models were estimated using Huber/White standard errors.

Table 5
The association between the genetic index and grade point average at wave 1 for the full sample and by gender.

	Full sample			Male subsample			Female subsample		
	b	*Beta*	*P*	*b*	*Beta*	*P*	*b*	*Beta*	*P*
Genetic index	−.12	−.12	<.001	−.11	−.10	.004	−.14	−.14	<.001
Gender	−.25	−.17	<.001						
Race	−.17	−.11	<.001	−.15	−.09	.006	−.20	−.13	<.001
N	1748			838			910		

Note: All models were estimated using Huber/White standard errors.

math. Once again, the effect of race was statistically significant in all of the models.

Next, the cumulative genetic index was used to predict total GPA measured at wave 1. These models were estimated for the full sample and separately by gender, the results of which are presented in Table 5. For the full sample, the genetic index had a statistically significant and negative effect on overall GPA. Race also was significantly related to overall GPA. Very similar results were generated when analyzing both males and females separately. A difference-in-coefficients z-test (Paternoster, Brame, Mazerolle, & Piquero, 1998) confirmed that the effect sizes for the genetic risk index were not significantly different between males and females. To facilitate interpretation of these effects, the total GPA was plotted as a function of scores on the genetic index. As Fig. 1 shows, there was a steep decrease in GPAs for the full sample, for males, and for females as the number of risk alleles increased. For the full sample, the average GPA for subjects who scored "0" on the genetic index was 3.25, while the average GPA for subjects who scored a "3" on the genetic index was 2.85. For females who scored "0" on the genetic index, their average GPA was 3.10, while the average GPA for females who scored a "3" on the genetic index was 2.73. Finally, the average GPA for males who scored "0" on the genetic index was 2.94, while the average GPA of a male who scored a "3" on the genetic index was 2.62.

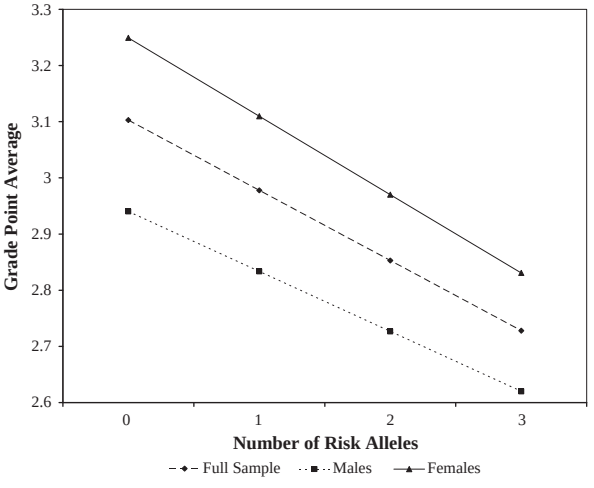

Fig. 1. The association between the number of risk alleles and grade point average for the full sample and by gender at wave 1.

Last, models presented in Table 5 and Fig. 1 were replicated by using GPAs that were garnered from wave 2 interviews. Table 6 shows the results of these models. The far left-hand column depicts the findings in respect to the full sample and shows that the genetic index maintained a statistically significant and negative association with total GPA at wave 2. A very similar pattern of results emerged for the male sample, where the genetic index had a significant negative effect on GPA at wave 2. For females, however, there was not an association between the genetic index and GPA at wave 2. Importantly, the results of a difference-in-coefficients test (Paternoster et al., 1998) revealed that there was not a statistically significant difference in effect sizes for the genetic risk index between males and females. Across all of the models, race was significantly related to GPA. The association between GPA and scores on the genetic index was once again plotted and the results presented in Fig. 2. For the full sample, the average GPA for subjects who scored "0" on the genetic index was 3.04, while the average GPA for subjects who scored a "3" on the genetic index was 2.82. For females who scored "0" on the genetic index, their average GPA was 3.10, while the average GPA for females who scored a "3" on the genetic index was 2.96. Note, however, that the association between GPA and the genetic index was non-significant for females. Finally, the average GPA for males who scored "0" on the genetic index was 2.97, while the average GPA of a male who scored a "3" on the genetic index was 2.67.

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Table 6
The association between the genetic index and grade point average at wave 2 for the full sample and by gender.

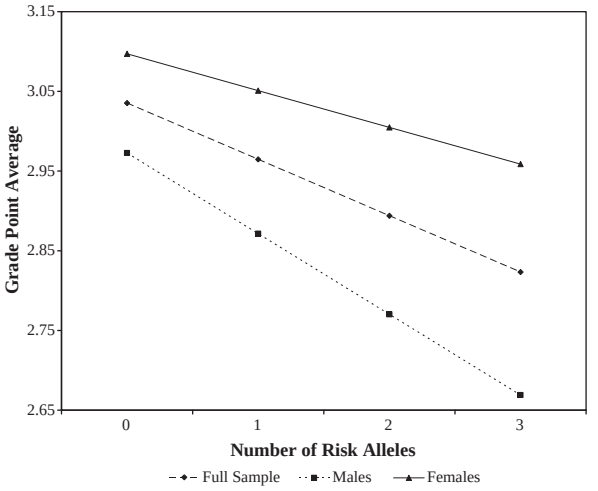
	Full sample			Male subsample			Female subsample		
	b	*Beta*	*P*	*b*	*Beta*	*P*	*b*	*Beta*	*P*
Genetic index	−.07	−.07	.010	−.10	−.10	.015	−.04	−.05	.210
Gender	−.22	−.15	<.001						
Race	−.17	−.11	<.001	−.16	−.10	.012	−.19	−.12	<.001
N		1298			676			622	

Note: All models were estimated using Huber/White standard errors.

6. Discussion

The past ten years has witnessed an explosion of molecular genetic research attempting to link certain genotypes to virtually every measurable phenotype. This body of research has produced a considerable amount of knowledge about the specific genes and gene systems that are related to a wide range of behavioral, personality, and cognitive phenotypes (Plomin, Kennedy, & Craig, 2006; Rutter, 2006). Surprisingly, however, less research has directly examined the specific genetic polymorphisms that might contribute to variation in academic achievement and performance (Plomin, 2003). The current study was designed to address this limitation and to examine whether polymorphisms in three dopaminergic genes—DAT1, DRD2, and DRD4—were related to middle and high school grades in English, math, history, and science. To our knowledge, this is the first study to examine the association between dopaminergic polymorphisms and academic achievement by analyzing genotypic data drawn from the National Longitudinal Study of Adolescent Health. Analysis of these data revealed three main findings.

First, the three dopaminergic genes were significantly related to grades in English, math, history, and science. These associations, however, were somewhat variable. DAT1, for example, only had a marginal effect on English grades at wave 1, but was unrelated to all of the other academic grades at wave 1 and at wave 2. In contrast, DRD2 was associated with grades in all of the subjects at wave 1, while DRD4 was related to grades in all four subjects at wave 1 and history and science at wave 2. The reasons why these genes had different effects on different subjects at different times are not immediately obvious. The differential genetic effects, however, likely are produced, in part, by the fact that performance in certain academic subjects depends on the use of specific regions of the brain. Performance in English courses requires the use of areas of the brain that are related to verbal abilities, while performance in science and math courses requires visuospatial skills (Brizendine, 2006). Given that the dopaminergic genes analyzed in this study tend to have different effects on these brain functions (cf. Ball et al., 1998; Berman & Noble, 1995; Moises et al., 2001; Petrill et al., 1997) necessarily means that the genetic effects should vary across academic subjects. Indeed, Haworth, Kovas, Dale, and Plomin (2008) recently found differential heritability estimates for *g*, English, math, and science which comports with the current findings that measured genes had differential effects across disciplines.



Note: the slope for females is not statistically significant

Fig. 2. The association between the number of risk alleles and grade point average for the full sample and by gender at wave 2.

The second main finding to emerge from the current study was that the genetic index was related to cumulative GPA at two points in adolescence. Genetic indexes have gained traction in recent years as one way to explore the genetic underpinnings to multifactorial phenotypes that are produced by gene systems (Beaver, 2008a; Beaver, Sak, Vaske, & Nilsson, 2010; Conner et al., 2010; Harlaar et al., 2005; Sonuga-Barke, Oades, Psychogiou, Chen et al., 2009). This is especially true for academic performance, which is likely partially a function of genes from the dopaminergic system. Importantly, the use of a genetic index is particularly well-suited to examine academic performance because some dopaminergic genes may be related to some endophenotypes, but others are not. For example, DAT1 has been linked to antisocial behaviors (Guo et al., 2007), but not cognitive abilities. Other genes from the dopaminergic system, including DRD2, have been linked to cognitive abilities (Berman & Noble, 1995). As a result, to systematically evaluate the extent to which dopaminergic genes relate to academic achievement, it is in many ways far more useful to combine all of these genes together into one cumulative index. By doing so, it is possible to begin to map out the ways in which systems of genes do or do not relate to complex phenotypes, such as academic performance.

The genetic index had relatively modest effects on GPA, but bear in mind that only three genes were examined. It is likely that had more dopaminergic genes been included in the genetic index, the association would have increased in magnitude. Even with only three genes, there was a non-trivial effect. For example, for males there was an 11% reduction in GPA when comparing those who scored "0" on the genetic index with those that scored a "3." This difference could translate into a

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student being accepted into a college versus being denied admission. Of course, much more research is needed to explore this possibility, but for now the current study draws attention to the utility of creating genetic indexes when examining multifactorial, polygenic phenotypes.

Third, there was some evidence of a gendered effect, wherein the genetic index was associated with GPA for males at both waves of data collection, but was only related to GPA for females at wave 1. The current study was unable to investigate the possible reasons for this difference. While speculative, this gender difference could be a methodological artifact or it could accurately capture the differential effects that dopaminergic genes have on GPA later in high school. Future research would benefit by exploring this issue in greater detail.

The findings generated from the current study should be interpreted with caution in light of a number of limitations. Of particular concern was that the measure of GPA was generated from self-reports, not from official school transcripts. As a result, the academic school performance measure may not accurately capture the GPA of all students. However, the only way that this measurement strategy would bias the results would be if GPA reporting were a function of the genotypes examined in this study. We were unable to locate any study showing that item-response functioning is tied to genes from the dopaminergic system. To the extent that dopaminergic genes do not systematically bias youths to misrepresent their GPAs, the use of self-reports should not bias the results reported in the current study.

Additionally, although the Add Health sample is a nationally representative sample of youths, the DNA subsample of the Add Health is not necessarily nationally representative. The possibility exists, therefore, that the results reported here would not be generalizable to adolescents in the United States or in other countries. It is important to note, however, that research has examined whether the sibling-pair data (from which the DNA sample is drawn) differs significantly from the nationally representative Add Health sample. The results of these studies have failed to detect any significant differences on key demographic variables and on phenotypic measures (Beaver, 2008b; Jacobson & Rowe, 1998). Last, genetic research has underscored the importance of the environment in moderating the effects of specific genes (Devlin et al., 1997; Moffitt, 2005; Plomin & Petrill, 1997; Rutter, 2006). Because the current study was exploratory and was designed to examine the main effects of genes, potential gene–environment interactions were not examined. Future research, however, should begin to map out the various environments, such as those found in the family or in the school, that might magnify or blunt the genetic effects that were reported in this study.

Increasing academic performance and achievement are critically important to increasing subsequent prosocial outcomes and reducing negative ones. In order to do so, however, a concrete understanding of the factors that are associated with educational performance is of utmost importance. For the most part, very little is known about the genes that might contribute to academic achievement. The current study shed some light on the genetic contributors to academic performance and hopefully will spawn additional researchers to examine the complex ways in which genes and the environment work in concert to produce variation in educational performance.

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