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Dopaminergic Polymorphisms and Educational Achievement: Results From a Longitudinal Sample of Americans

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Although educational attainment has been found to be moderately heritable, research has yet to explore candidate genes for it. Drawing on data from the National Longitudinal Study of Adolescent Health, in the current study, we examined the association between polymorphisms in three dopaminergic genes (DAT1, DRD2, and DRD4), a dopamine index, and educational attainment. Statistically significant effects were found for DAT1, DRD2, DRD4, and the dopamine index for highest level of education. This study is the first to our knowledge that links measured genes to educational attainment.

Keywords: academics, adolescence, dopamine, education, genetics

Educational attainment is an important correlate of a broad range of social outcomes. Individuals with relatively higher levels of education have been found to earn more money over their life (Ganderton & Santos, 1995; Herrnstein & Murray, 1994; Steinberg, Brown, & Dornbusch, 1996), report higher levels of satisfaction with life (Meeks & Murrell, 2001), and enjoy greater long-term stability in their social class standing (Johnson, Brett, & Deary, 2010a, 2010b). Conversely, individuals with comparatively less educational attainment, especially individuals who do not graduate from high school, have been found to be at increased risk for engaging in crime and disrepute (Elliott et al., 2006; Maguin & Loeber, 1996; Schulenberg, Bachman, O'Malley, & Johnston, 1994) and to be at increased risk for prolonged welfare use and unemployment (U.S. Bureau of Justice Statistics, 2010). As a gradual process that places increasing cognitive and personal demands on individuals, educational attainment reflects an important gating process that differentiates individuals on the basis of a range of factors, including random life events, intelligence, motivation, the ability to delay gratification, and social support (Haworth & Plomin, 2010; Plomin & Spinath, 2004).

A body of research exists that implicates genetic influences on academic achievement and attainment with studies indicating that genetic factors account for approximately half of the variance in these educational measures (Baker, Treloar, Teynolds, Heath, & Martin, 1996; Gill, Jardine, & Martin, 1985; Johnson, Deary, & Iacono, 2009; Johnson, McGue, & Iacono, 2006; Van Den Oord & Rowe, 1997; Wainright, Wright, Luciano, Geffen, & Martin, 2005). Although the available evidence tends to suggest that genes influence, at least partially, educational attainment, to date, no candidate genes have been identified. The genes that would be most likely to be related to educational attainment would be those that have previously been found to be associated with known correlates to educational attainment. Some of the genes of the dopaminergic system meet this criterion because they have been found to be associated with a wide array of factors (Wahlstrom, Collins, White, & Luciana, 2010), some of which are known to correlate with educational outcomes.

In the current study, we focused on polymorphisms found in three dopaminergic genes: one in the dopamine transporter gene (DAT1), one in the dopamine D2 receptor gene (DRD2), and one in the dopamine D4 receptor gene (DRD4). Together, these three dopaminergic genes represent prime candidate genes for educa-

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tional outcomes because of their functional effects as well as their associations with known correlates to educational attainment. From a functional standpoint, the DAT1 gene codes for the production of the dopamine transporter that is involved in the reuptake of dopamine (Vandenbergh et al., 1992). A polymorphism in this gene (SLC6A3) has been shown to be functional, with some research suggesting that subjects who are homozygous for the 10R allele, in comparison with 9R allele carriers, may have reduced dopamine transporter binding (Jacobsen et al., 2000). Second, there is also reason to suspect that a point mutation in the DRD2/ANKK1 gene may be related to educational attainment. Research has revealed some functional differences between the A1 genotype and the A2 genotype, where A1 carriers have fewer D2 dopamine receptors, reduced glucose metabolism in the brain, and blunted dopaminergic activity in the central nervous system (Berman & Noble, 1995; Noble, Blum, Ritchie, Montgomery, & Sheridan, 1991; Noble, Gottschalk, Fallon, Ritchie, & Wu, 1997). Last, a polymorphism in the DRD4 gene has also been shown to have functional consequences, wherein some evidence indicates that the 7R allele may code for a postsynaptic receptor that is relatively subsensitive to dopamine (Asghari et al., 1995). Behaviorally, this gene is involved in regulating attention and facilitating motivation (Schmidt, Fox, Perez-Edgar, Hu, & Hamer, 2001).

Any potential associations that these dopaminergic genes have with educational attainment would not be direct but rather indirect, operating through a range of interpersonal characteristics and environments previously identified as influencing educational attainment. Exactly which factors might mediate the association between dopaminergic genes and educational attainment is not well-known, but, drawing from the literature, it is possible to identify at least four main groups of potential mediators. First, there is some empirical evidence to suggest that dopaminergic system genes, including some of the polymorphisms described previously, are associated with cognitive abilities and intelligence (Berman & Noble, 1995; Frank, Moustafa, Haughey, Curran, & Hutchison, 2007). Given that cognitive skills are known predictors of educational attainment, it is quite possible that dopaminergic genes would be related to educational attainment via the effects they have on intelligence and other cognitive measures. Second, a number of studies have revealed that dopaminergic genes are associated with violence (Palermo, 2010), which has been shown to be related to lower educational levels. Third, there is some evidence to suggest that exposure to risky peer groups is influenced, in part, by dopaminergic polymorphisms (Yun, Cheong, & Walsh, 2011). At the same time, adolescents who are embedded in antisocial peer networks are at risk for low educational attainment. Last, poverty—a strong predictor of educational attainment—is known to be transmitted across generational lines, and there is some evidence that certain genes may explain part of the reason for chronic poverty (Rowe & Rodgers, 1997). Given that dopaminergic genes have been found to be related to intelligence, violence, and other maladaptive outcomes, it stands to reason that the effect that these genes have on educational attainment may be mediated, partially, by poverty. It is also important to note that demographic variables may potentially confound associations between genetic polymorphisms and phenotypes. To help minimize this possibility, in the current study, we also take into account the effects of race (to avoid population stratification effects), age (to capture potential genetic effects that vary across age), and sex (to capture potential genetic effects that vary between males and females).

Against this backdrop, there are three goals of the current study. First, we examine whether each of the individual dopaminergic polymorphisms is associated with educational attainment. However, given that educational attainment is a multifactorial, polygenic phenotype, we anticipate that the associations between the individual dopaminergic polymorphisms and educational attainment will be quite small. Previous research examining multifactorial phenotypes, however, has revealed that genetic effects on complex phenotypes tend to be stronger and more consistent when the individual polymorphisms are combined together to create a genetic predisposition or profile (Beaver, 2008a; Belsky & Beaver, 2011; Harlaar et al., 2005). As a result, the second goal of the study is to examine the link between a dopamine index and educational attainment. The third goal of the study is to examine the potential mediating mechanisms that might account for the link between dopaminergic polymorphisms and educational outcomes. To do so, we examine the mediating roles of poverty, IQ, violence, and risky peers.

Method

Subjects

Data for this study were drawn from the National Longitudinal Study of Adolescent Health (Add Health). Details of the Add Health data and sampling design have been discussed elsewhere (Harris, Halpern, Smolen, & Haberstick, 2006; Resnick et al., 1997; Udry, Bearman, & Harris, n.d.). Briefly, the Add Health is a four-wave, prospective study of a nationally representative sample of American youths who were enrolled in middle or high school in 1994–1995 (Udry, 2003). The initial wave of data was collected between 1994 and 1995 when approximately 90,000 students were administered a self-report survey (i.e., the Wave 1 in-school survey). Follow-up interviews were conducted with a subsample of 20,745 youths and their primary caregivers (usually their mother) at their home (i.e., the Wave 1 in-home survey). A second round of surveys was administered in 1996 with 14,738 respondents. Nearly six years later, the third wave of data was collected from 15,197 respondents. The fourth and final wave of data was collected between 2007 and 2008, when most of the respondents were between the ages of 24 and 32 years old. A total of 15,701 adolescents were successfully interviewed at Wave 4 (Harris et al., 2003).

During Wave 3 interviews, a subset of respondents were asked to participate in the DNA sample of the Add Health study by submitting samples of their buccal cells for genotyping. Not all respondents were eligible to participate in the DNA sample; only respondents who had a sibling who also was included in the Add Health study were asked to participate. In total, more than 2,500 subjects were genotyped, making the Add Health study one of the largest samples in the world that includes genotypic data (Harris et al., 2006). After removing cases using listwise deletion techniques, the final analytical sample used in all of the analyses included 1,674 respondents.

Genotyping

Genotyping of the Add Health subjects was conducted in a coordinated effort between the Institute for Behavioral Genetics in Boulder, Colorado, and Add Health. In the current study, we examine three dopaminergic polymorphisms that were genotyped in the Add Health study. First, subjects were genotyped for a 40 base pair variable number of tandem repeats (VNTR) that has been found in the 3′ untranslated region of the dopamine transporter gene (SLC6A3). The number of repeats (R) for this polymorphism ranges between 3 and 11 copies. This VNTR was amplified by using the primer sequences forward, 5′-TGTGGTGTAGGGAACGGCCTGAG-3′ (fluorescently labeled), and reverse, 5′-CTTCCTGGAGGTCACGGCTCAAGG-3′, which produced PCR products of 320 (6R allele), 360 (7R allele), 400 (8R allele), 440 (9R allele), 480 (10R allele), and 520 (11R allele) base pairs. We followed prior researchers analyzing the Add Health data and only included subjects who possessed the 9R allele or the 10R allele; all other alleles lengths were removed from the final analytical sample (Hopfer et al., 2005). The distribution of alleles was as follows: 38.2% of the sample was homozygous or heterozygous for the 9R allele, whereas 61.8% of the sample was homozygous for the 10R allele.

The second polymorphism included in the current analysis is the DRD2/ANKK1 TaqIA polymorphism. This polymorphism was assayed by using the Applied Biosystem's Taqman Assays by Design for SNP Genotyping Service (Haberstick & Smolen, 2004). The DRD2/ANKK1 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 indicated the presence of the TaqIA-1 allele and the C-probe signal indicated the presence of the TaqIA-2 allele. Fifty-six percent of the sample was homozygous for the A-2 allele, and 44% of the sample was heterozygous or homozygous for the A-1 allele.

The third gene that was included in the analysis was DRD4. DRD4 has a polymorphism that is the result of a 48 base pair VNTR located at 11p15.5 on exon III. This polymorphism was amplified by using the primer sequences forward, 5′-AGGACCCTCATGGCCTTG-3′ (fluorescently labeled), and reverse, 5′-GCGACTACGTGGTCTACTCG-3′, which produced PC products of 379 (2R), 427 (3R), 475 (4R), 523 (5R), 571 (6R), 619 (7R), 667 (8R), 715 (9R), and 763 (10R) base pairs. The two most common alleles were the 4R and the 7R. Following extant research (Hopfer et al., 2005), we pooled together the 2R allele, the 3R allele, the 4R allele, the 5R allele, and the 6R allele, and we pooled together the 7R allele, the 8R allele, the 9R allele, and the 10R allele. Sixty-one percent of the sample was homozygous for the $<$7R allele, whereas 39% of the sample was heterozygous or homozygous for the $\geq$7R allele.

Hardy–Weinberg equilibrium (HWE) tests were calculated for all three dopaminergic genes for the final analytical sample. The results revealed that HWE was fulfilled for DAT1, $\chi^2 = .236$, $p = .626$, and for DRD4, $\chi^2 = .305$, $p = .581$, but not for DRD2, $\chi^2 = 7.10$, $p = .008$.

Last, we extended prior research by creating a dopaminergic index to examine simultaneously the cumulative effects of the dopaminergic polymorphisms on educational attainment (Beaver, 2008a; Conner, Hellemann, Ritchie, & Noble, 2010; Harlaar et al., 2005). This index was created by summing together the number of risk alleles that each respondent possessed for DAT1, DRD2, and DRD4. Specifically, the 10R allele of DAT1 was identified as the risk allele (where respondents who were homozygous for the 10R allele were assigned a value of 1), the A1 allele of DRD2 was identified as the risk allele (where respondents who were homozygous or heterozygous for the A1 allele were assigned a value of 1), and the 7R allele of DRD4 was identified as the risk allele (where respondents who were homozygous or heterozygous for alleles with seven or more repeats were assigned a value of 1). The resulting value on this index indicates the number of risk alleles that each respondent possesses using the coding scheme described above. Table 1 includes a bivariate correlation matrix for all of the dopaminergic measures as well as the other variables and scales that are used in the analyses.

Measures

Educational attainment. During Wave 4 interviews, respondents were asked to indicate the highest level of education that they had completed. Responses to this item were coded from 1

Table 1
Correlation Matrix for Selected Add Health Study Variables (N = 1,674)

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Education level	—													
2. DAT1	−.05*	—												
3. DRD2	−.10*	.01	—											
4. DRD4	−.07*	−.01	−.00	—										
5. Dopamine index	−.12*	.57*	.59*	.57*	—									
6. Poverty	−.20*	.00	.03	.04	.04	—								
7. IQ	.37*	−.07*	−.11*	−.04	−.13*	−.19*	—							
8. Violence	−.19*	−.02	.05*	−.00	.02	.01	−.07*	—						
9. Risky peers	−.21*	−.00	.02	.03	.03	.03	−.05*	.32*	—					
10. Caucasian	.03	−.11*	−.18*	.02	−.16*	−.07*	.30*	−.02	.06*	—				
11. African American	−.03	.09*	.11*	.06*	.15*	.08*	−.27*	.05*	−.08*	−.71*	—			
12. Other minority	−.01	.05*	.13*	−.10*	.04	.01	−.12*	−.03	.01	−.58*	−.16*	—		
13. Age	.03	.02	.01	−.02	.01	−.03	−.02	−.03	.27*	−.02	−.05*	.08*	—	
14. Sex	−.10*	−.01	.01	−.03	−.02	−.02	.05	.19*	.03	.03	−.02	−.02	.01	—

* $p < .05$, two-tailed.

(indicating eighth grade or less) through 13 (indicating postbaccalaureate professional degree [e.g., law or medical degree]). Because the coding scheme is somewhat ordinal, we also used different coding schemes for this variable (e.g., collapsing different categories together), and the results of the models remained the same. As a result, we only present the results for the models using the original coding scheme.

Mediation variables. If the dopaminergic measures are related to educational outcomes, then it is important to also examine some potentially mediating variables. On the basis of previous research, we identified four variables that could mediate the association between dopaminergic polymorphisms and educational outcomes: poverty, intelligence (IQ), violence, and exposure to delinquent peers. Poverty was measured through a single item asked of the subject's primary caregiver at Wave 1. Specifically, they were asked whether they were currently receiving public assistance, such a welfare (0 = no, 1 = yes). Intelligence was measured through a modified version of the Peabody Picture Vocabulary Test (PPVT) that was administered to subjects during Wave 1 in-home interviews. Violence was measured with a previously created seven-item scale (Beaver, 2008a) that asked the subjects at Wave 1 to indicate the frequency in the past year of engaging in violent behaviors, such as shooting or stabbing someone, pulling a knife or gun on someone, and taking part in a group fight ($\alpha = .72$). Last, a three-item risky peers measure was created on the basis of previous protocols (Bellair, Roscigno, & McNulty, 2003). During Wave 1 interviews, subjects were asked to indicate how many of their three closest friends smoke at least one cigarette each day, smoke pot at least once a month, and drink alcohol at least once a month ($\alpha = .76$).

Control variables. To help reduce the possibility of confounding, we included three control variables in the analyses. First, to avoid population stratification effects, we entered a race variable into all of the statistical equations. Race was self-reported and we trichotomized race, such that 0 = Caucasian, 1 = African American, and 2 = other minority. Second, age was included in all of the statistical models as a continuous variable measured in years. Third, sex was included as a dichotomous dummy variable (0 = female and 1 = male).

Analytic Approach

The analysis for this article proceeded in a series of linked steps. First, the three genetic polymorphisms were used as predictors of highest level of education in an ordinary least squares (OLS) regression equation and, in a separate OLS model, the dopamine index was used as a predictor variable of the highest level of education variable. For both of these models, two different equations were estimated: a baseline model and a mediation model. The baseline model estimated the effects of the dopaminergic measures controlling only for race, age, and sex. Race was controlled for by introducing dummy variables for Caucasian and African American; other minority was excluded and used as the reference category. The mediation model expanded the baseline model by introducing the mediation variables of poverty, IQ, violence, and risky peers. A comparison of the two models will reveal whether any statistically significant associations between the dopaminergic measures and the highest level of education are mediated through common correlates to educational attainment. In addition, Huber–White standard errors were estimated for all of the models to correct for the lack of independence in some of the observations (i.e., more than one sibling from the same household). Following prior research, one twin from each monozygotic twin pair was randomly removed to produce more conservative and unbiased tests of statistical significance (Haberstick et al., 2005).

Results

The analysis began by examining the associations between the dopaminergic measures and the highest level of education by estimating OLS models. The results of these models are presented in Table 2. As shown in the baseline model, all three of the

Table 2
Association Between Dopaminergic Genes and Highest Level of Education (N = 1,674)

	Baseline model			Mediation model			Baseline model			Mediation model		
Measure or variable	*b*	*SE*	β	*b*	*SE*	β	*b*	*SE*	β	*b*	*SE*	β
Genetic measure												
DAT1	−.22	.11	−.05*	−.17	.10	−.04						
DRD2	−.41	.11	−.09*	−.26	.10	−.06*						
DRD4	−.31	.11	−.07*	−.21	.10	−.05*						
Dopamine index							−.31	.06	−.12*	−.21	.06	−.08*
Mediation measure												
Poverty				−1.10	.17	−.13*				−1.10	.17	−.13*
IQ				.05	.00	.35*				.05	.00	.35*
Violence				−.09	.02	−.09*				−.09	.02	−.09*
Risky peers				−.14	.02	−.17*				−.14	.02	−.17*
Control variables												
Caucasian	.03	.18	.01	−.32	.16	−.07	.04	.18	.01	−.31	.16	−.07
African American	−.06	.21	−.01	.17	.19	.03	−.05	.21	−.02	.18	.19	.03
Age	.04	.03	.03	.09	.03	.07*	.04	.03	.03	.09	.03	.07*
Sex	−.43	.11	−.10*	−.40	.10	−.09*	−.43	.11	−.10*	−.40	.10	−.09*

Note. All models are estimated using Huber–White standard errors.
* $p < .05$, two-tailed.

dopaminergic polymorphisms maintained a negative association with the highest level of education achieved. In the mediation model, DAT1 dropped from statistical significance, but DRD2 and DRD4 remained significant predictors of the highest level of education. Sobel tests were calculated to further examine the potential mediating effects of the mediation variables (Sobel, 1982). The results of these tests revealed that IQ partially mediated the effects of DAT1 and DRD2; however, none of the other variables mediated the associations between the dopaminergic genes and highest level of education. The last two models in Table 2 present the findings for the dopamine index. In the baseline model, the dopamine index was negatively related to the highest level of education, and this significant effect persisted in the mediation model, too. Again, the results of the Sobel tests revealed that only IQ partially mediated the association between the dopamine index and the highest level of education.

Discussion

The possibility that academically oriented phenotypes are under considerable genetic influence has evolved from taboo to common acceptance. Although much research has explored the genetic underpinnings of intelligence, virtually none has examined candidate genes that contribute to more complex, behaviorally oriented phenotypes such as educational attainment in community samples. Given that dopaminergic polymorphisms have been linked to a range of known correlates for educational attainment, the current study examined the potential association between three dopaminergic polymorphisms and highest level of education.

Analysis of the Add Health data revealed some evidence indicating that dopaminergic risk alleles were associated with the highest level of education achieved. The question of how these polymorphisms ultimately affect educational attainment, however, remains unresolved. Clearly, there are not single genes that have a one-to-one correspondence with educational attainment. Rather, mediating mechanisms and endophenotypes act as intermediary processes linking gene products to educational outcomes. On the basis of prior research, we anticipated that the link between dopaminergic polymorphisms and educational attainment could be mediated by environmental factors, such as poverty and exposure to delinquent peers, and/or individual-level phenotypes, such as violence and intelligence. The mediation models that introduced measures for poverty, intelligence, violence, and delinquent peers did not provide much empirical support for this proposition. Across the models, the inclusion of the mediation variables reduced the effects of the dopaminergic measures, but the associations between DRD2 and DRD4 and educational achievement remained statistically significant. Sobel tests revealed that only one variable—IQ—mediated the effects of DAT1 and DRD2 on highest level of education achieved. None of the other mediation variables were implicated in the pathway between the genes and educational levels. That a mere three polymorphisms were related to educational attainment even after controlling for some key mediating factors speaks to their potential importance in understanding the developmental pathways to educational outcomes. It is important to point out, however, that although the three genes had statistically significant effects on education levels, each of these three genes had relatively small effects, accounting for only a small proportion of variance. These small effects are anticipated, as educational achievement is a complex polygenic phenotype that is influenced by hundreds or thousands of genes, with each gene having only a small effect.

Moreover, an important next step in unpacking the mechanisms by which dopaminergic genes are related to educational attainment is to explore potential gene–environment interactions. Prior research has revealed, for instance, that certain environmental factors moderate genetic effects on a range of antisocial behaviors and psychopathologies (Caspi et al., 2002, 2003; Rutter, 2006). It is quite likely, therefore, that certain environments may dampen or exacerbate the effects of the dopaminergic genes on educational attainment. Although we explored the possibility that the mediation measures were actually moderators of genetic effects, the results of these models did not provide any clear evidence indicating the presence of a gene–environment interaction. Future research should examine this possibility in detail.

The results of this study should be interpreted cautiously because of a number of limitations. First, the DNA sample analyzed in the current study is not necessarily nationally representative, which could limit the generalizability of the results. Prior research has examined whether the sibling pairs sample (which the DNA sample is embedded within) and the nationally representative sample are different from each other on key demographic variables and behavioral characteristics (Beaver, 2008b; Jacobson & Rowe, 1998). These studies did not reveal any significant differences between the samples, providing some suggestive evidence that the results of the current study might be generalizable to the larger population of Americans. Second, the analyses estimated a series of multivariate models, leaving open the possibility that the results were a methodological artifact owing to multiple tests. Third, the dopamine index was created by summing together individual alleles for three dopaminergic genes. It is quite possible that the effects of the dopaminergic genes would not have a cumulative additive effect but rather may work in more complex ways, such as through interactions.

In closing, dopamine genes have been extensively studied in clinical samples with a focus on their associations with educational and cognitive deficits and psychopathology (Gunter, Vaughn, & Philibert, 2010). The current study suggests that dopaminergic genes are similarly important in nonclinical, community samples and that relatively small genetic effects can produce large implications for educational attainment. However, given the problems with replicating the results of molecular genetic association studies, caution should be exercised in accepting these results until future studies reveal similar findings.

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