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Genetic Correlates of Adult Attachment Style

Omri Gillath
University of Kansas

Phillip R. Shaver
Jong-Min Baek
David S. Chun
University of California, Davis

Attachment theory attempts to explain effects of social experiences, not genes, on personality development. Most studies of the development of attachment insecurities support this emphasis on social experiences rather than genes, although there are exceptions. In the present study, the authors examine associations between attachment insecurities and particular genetic polymorphisms related to emotions and social behavior. They find that (a) anxious attachment is associated with a polymorphism of the DRD2 dopamine receptor gene, (b) avoidant attachment is associated with a polymorphism of the 5HT2A serotonin receptor gene, and (c) the rs53576 A polymorphism of the OXTR oxytocin receptor gene is not associated with attachment insecurities. These findings suggest that attachment insecurities are partially explained by particular genes, although there is still a great deal of individual difference variance that remains to be explained by other genes or social experiences.

Keywords: *attachment style; genes; dopamine; serotonin; oxytocin; polymorphism*

Bowlby (1982), the creator of attachment theory, described child–caregiver attachment as an innately prepared process, which, given an adequate rearing environment, leads to a stable sense of attachment security. This sense of security is an important foundation for later social relationships and effective affect regulation (for reviews, see Grossmann, Grossmann, & Waters, 2005; Mikulincer & Shaver, 2007). According to Bowlby, the rearing environment experienced by a child (e.g., a sensitive, responsive parenting style vs. a rejecting or neglectful one) is the major factor that determines a person's later relationship patterns and attachment style (also see Fraley, 2002). This theoretical framework implies that a child can develop any attachment style (e.g., secure or insecure, anxious or avoidant), depending on social experiences. That is, over time, social interactions shape a stable set of internalized working models (cognitive–affective schemas) and affect–regulation skills that, together, constitute the person's dispositional attachment style. Viewed from the perspective of behavioral genetics, attachment theory focuses primarily on unique and shared environmental determinants of personality development rather than on genetic determinants (Fearon et al., 2006).

In the past decade, however, researchers have begun to report data, mainly from twin studies but also from animal models, indicating that genes play a role in the development of separation anxiety, proximity seeking, and attachment style (e.g., Parent et al., 2005). These data, while providing information about the possible contribution of genes to adult attachment style, rarely reveal which genes are involved, especially in the case of normally developing adults (as distinct from those with a disorganized attachment style, which seems to be more affected by genes and more conducive to psychopathology; e.g., Carlson, 1998; van IJzendoorn & Bakermans-Kranenburg, 2006). The present study focuses on identifying specific candidate genes associated with adult attachment security and insecurity in a normal college sample.

Individual differences in attachment style can be assessed along two dimensions of insecurity, usually labeled *attachment-related anxiety* and *avoidance* (e.g., Bartholomew & Horowitz, 1991; Brennan, Clark, & Shaver, 1998; Fraley & Shaver, 2000). Attachment

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anxiety is characterized by fear of rejection and abandonment and by doubts about one's desirability as a relationship partner. Avoidant attachment, in contrast, includes emotional distancing from relationship partners as well as an exaggerated sense of independence and self-reliance motivated by discomfort with interpersonal closeness and interdependence. Scoring low on both dimensions indicates attachment security, which is associated with feelings of trust, comfort with closeness, and relative ease in initiating and maintaining committed, satisfying, long-lasting relationships.

The two attachment dimensions (anxiety and avoidance) have been examined in hundreds of studies (see Mikulincer & Shaver, 2007, for a review), demonstrating that they are associated with the ways in which people experience and behave in romantic relationships and regulate emotions during periods of stress. The major dimensions of attachment insecurity have well-documented cognitive, physiological, and neurological correlates (e.g., Diamond, 2001; Gillath, Bunge, Shaver, Wendelken, & Mikulincer, 2005), which resemble the biological correlates of certain temperaments (e.g., Crawford et al., 2007; Davidson, 2004).

This resemblance is not surprising, because the two attachment-style dimensions—anxiety and avoidance—have been shown in previous studies (e.g., Noftle & Shaver, 2006) to be correlated with broader personality traits, such as those included in the well-accepted Five-Factor Model of Personality (McCrae & Costa, 1996). Therefore, in many studies investigating attachment style correlates or effects, researchers have checked to see whether attachment-related outcomes can be explained by broader personality traits, such as general anxiety or neuroticism (e.g., Gillath et al., 2005; Mikulincer, Gillath, & Shaver, 2002). As expected, based on attachment theory, even when attachment anxiety—and sometimes avoidance as well—are found to correlate with more general traits, the hypothesized correlates and effects (both behavioral and neurological) of attachment insecurities remain statistically significant even after scores on measures of general personality traits have been statistically controlled.

Specifically, anxious attachment is often correlated with the broad trait of neuroticism, and avoidant attachment is sometimes correlated with the broad trait of agreeableness (e.g., Noftle & Shaver, 2006), although the correlations are never high enough to suggest that the attachment-specific and more general traits are completely redundant. These correlations are potentially important for behavior genetics, however, because twin studies show that the five major personality traits, including neuroticism and agreeableness, are influenced by genes (e.g., Jang et al., 2006; Livesley & Jang, 2005; Yamagata et al., 2006).

Although there are hundreds of studies of the nature and correlates of attachment insecurities, there are relatively few dealing with possible genetic influences. These few differ in at least two respects: (a) the way in which researchers measure attachment style, via self-report instruments, such as the Experiences in Close Relationships Inventory (ECR; Brennan et al., 1998); behavioral observations, such as the Strange Situation procedure (Ainsworth, Blehar, Waters, & Wall, 1978); or clinical interviews, such as the Adult Attachment Interview (George, Kaplan, & Main, 1985); and (b) the way in which genetic influences are measured (e.g., in behavior genetic studies of twins or by assessing polymorphisms of candidate genes).

Most behavior genetic studies of infant attachment style (e.g., Bokhorst et al., 2003; O'Connor & Croft, 2001; Roisman, & Fraley, 2006) have found that additive genetic effects do not account for individual differences in attachment, whereas shared and nonshared environment do account for them at least partially. Still, some investigators have obtained evidence for genetic correlates. For example, Finkel, Wille, and Matheny (1998) found that twin concordance on what the authors defined as attachment security was almost twice as high among monozygotic twins as among dizygotic twins. This study has been criticized by attachment researchers, however, because Finkel et al. (1998) did not use the standard measure of infant attachment, Ainsworth's Strange Situation (Ainsworth et al., 1978).

There is also some evidence for genetic contributions to attachment style in adulthood, based mainly on self-report measures (e.g., Brussoni, Jang, Livesley, & MacBeth, 2000; Crawford et al., 2007; Donnellan, Burt, Levendosky, & Klump, 2008; although Torgersen, Grova, & Sommerstad, 2007, used the Adult Attachment Interview). The self-report studies provide consistent evidence for genetic influences on attachment anxiety, which is known to be associated with neuroticism, a personality trait influenced by genes; but so far, they do not provide consistent evidence of genetic influences on avoidant attachment.

It seems likely, as Fonagy (2001) has suggested, that attachment patterns are shaped by a combination of genetic factors and social experiences. To understand this combination, it is important to identify candidate genes that predispose a person to a certain style of attachment. The predisposed style could then either emerge in development or be modified by social experiences (see Kagan, 2003, for a review). In other words, carrying specific genes or having a specific genetic polymorphism at a particular gene locus might be associated with developing a particular attachment style in a particular kind of social environment.

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Lakatos et al. (2002) offered an example of this kind of research. They examined polymorphisms of the dopamine DRD4 receptor gene in relation to disorganized attachment in infancy, a pattern of behavior that is characterized theoretically as a failure or inability to adopt one of the more organized insecure attachment strategies styles (anxious or avoidant). Lakatos and colleagues found that 1-year-olds were at a greater risk of disorganized attachment if they carried the DRD4 exon III 48 base-pair repeat polymorphism (7-repeat allele). In contrast, Bakermans-Kranenburg, van IJzendoorn, Bokhorst, and Schuengel (2004) failed to replicate this genetic main effect in a larger sample. Still, van IJzendoorn and Bakermans-Kranenburg (2006) did find that the DRD4 gene played a moderating role in the development of disorganized attachment, such that maternal unresolved loss or trauma was associated with infant disorganization 18.8 times as often among children with the DRD4 7-repeat allele. In other words, they found evidence of a gene–environment interaction (also see Gervai et al., 2005).

These studies suggest that further research on associations between candidate gene polymorphisms, on one hand, and attachment patterns, on the other, could reveal other polymorphisms related to attachment style. Because we, in our broader research program, are interested in adult attachment styles measured by self-report questionnaires (see Torgersen et al.'s [2007] discussion of the main alternative, the Adult Attachment Interview), we undertook the present study of associations between certain gene polymorphisms and attachment style assessed with the ECR inventory (Brennan et al., 1998), the most frequently used self-report measure of attachment style.

Possible Genetic Predictors of Attachment-Related Anxiety and Avoidance

At least three genes studied to date seem to play a role in social relationships: those associated with dopamine, serotonin, and oxytocin receptors (see reviews by Carter, 1998, 2006; Young & Wang, 2004).[1] Dopamine receptor genes and the dopaminergic system are thought to play a role not only in attachment (e.g., Gingrich, Liu, Cascio, Wang, & Insel, 2000; Insel, 2003; Lakatos et al., 2002) but also in social behavior more generally and specifically in social anxiety disorder (e.g., SAD; Kaminer & Stein, 2005; Stein, Westenberg, & Liebowitz, 2002). For instance, animal studies show that social status in monkeys is related to striatal dopamine D2 receptor binding, such that lower social status is associated with decreased D2 receptor binding (Grant et al., 1998).

Similarly, in humans, decreased striatal dopamine D2 binding is associated with dysfunctional social behavior (e.g., Pallanti, Quercioli, Rossi, & Pazzagli, 1999). Moreover, striatal dopamine D2 receptor density is associated with neuroticism, which is correlated with attachment anxiety (e.g., Hunnerkopf, Strobel, Gutknecht, Brocke, & Lesch, 2007; Lee et al., 2005). Insel (2003) summarized evidence from multiple studies (mainly with mice and monogamous prairie voles) by claiming that mesocorticolimbic dopamine plays a role in both infant–mother and adult pair bonding.

Reduced density of D2 dopamine receptors has been associated with the presence of the A1 allele of the dopamine D2 receptor gene (DRD2). For example, Noble (2001) found that the A1 allele was related to reduced density of D2 dopamine receptors in all areas of the striatum, reaching statistical significance in the ventral caudate and putamen. Specifically, there was a 30% to 40% reduction in D2 receptor density in the striatums of individuals with the DRD2 A1 allele compared to those who did not have it (i.e., were homozygous for the A2 allele; also see Thompson et al., 1997). This reduction in density is associated with impaired social functioning and heightened anxiety (Lawford, Young, Noble, Kann, & Ritchie, 2006) and is thought to be especially characteristic of A1 homozygotes (Blum, Braverman, Dinardo, Wood, & Sheridan, 1994; Finckh et al., 1997; Lucht et al., 2001).

Based on these findings, it seems likely that attachment anxiety or fearful avoidance (the combination of anxiety and avoidance; Bartholomew & Horowitz, 1991) is associated with lower levels of dopamine D2 receptors. Specifically, higher scores on attachment anxiety, and possibly also on avoidance when combined with attachment anxiety, are likely to be associated with the presence of one or two copies of the A1 allele on the DRD2 receptor gene.

The serotonin receptor gene is another candidate gene that may relate to attachment (e.g., Beech & Mitchell, 2005; Hennighausen & Lyons-Ruth, 2006). Serotonin, along with dopamine and oxytocin, seems to mediate the affective aspect of attachment. More broadly, deficits in the serotonergic system are involved in various affective disorders such as depression (e.g., Gross et al., 2002) and play a mediating role in the social behavior of animals. Reduced serotonin levels lead to avoidant social behavior in primates, whereas increased serotonin levels result in enhanced sociality (Raleigh, Brammer, & McGuire, 1983). This association between social behavior and serotonin appears to be bidirectional, such that changes in social status (as when dominant animals are removed from their social group) result in decreased serotonin levels (Raleigh, McGuire, Brammer, & Yuwiler, 1984).

In humans, serotonin has been hypothesized to play a role in social affiliation. Increased serotonergic activity is positively associated with affiliation and negatively associated with aversive affective experiences (Knutson

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et al., 1998). Additional evidence for the role of serotonin comes from the psychiatric treatment of affective disorders, where selective serotonin reuptake inhibitors are used to treat social anxiety and social withdrawal (e.g., van der Linden, Stein, & van Balkom, 2000).

The effects of serotonin on social behavior and social anxiety are thought to occur either through variations in the 5-HT transporter gene (5-HTT), which affects social affiliation, or through variations in the gene code for tryptophan hydroxylase (TPH), which affects anxiety (Davidson, Putnam, & Larson, 2000). In the present study, we focused on the T102C polymorphism of the serotonin receptor 5HT2A gene, which might reduce social involvement and thereby contribute to an avoidant attachment style.

Reduced serotonergic activity has been associated with the presence of the T102C polymorphism of the HTR2A gene (allele T, for short). Specifically, the presence of the T102C polymorphism has been associated with reduced serotonin transporter binding in the prefrontal cortex and thus with reduced serotonergic activity (Turecki, 2001; Turecki et al., 1999), which is negatively associated with social affiliation. The presence of allele T is also associated with symptoms of mood disorders (depression, anxiety) and lower scores on personality traits typically correlated inversely with avoidant attachment (i.e., agreeableness; Greenberg et al., 2000; Ni et al., 2006). We therefore predicted that higher scores on avoidant attachment would be associated with reduced levels of serotonin and more specifically with the presence of the T102C polymorphism.

Recently, animal studies have identified oxytocin as a neuropeptide involved in attachment (e.g., Bartz & Hollander, 2006; Carter & Cushing, 2004; Kirsch et al., 2005; Leckman, Hrdy, Eric, & Carter, 2006; Lim & Young, 2006). Studies of the regulation of affiliative behavior in mice suggest that oxytocin may be involved in human clinical disorders marked by social deficits and insecure attachment (e.g., Bartz & Hollander, 2006). Gonzaga and colleagues (Gonzaga, Turner, Keltner, Campos, & Altemus, 2006) found, in a study related to human attachment, that nonverbal displays of romantic love were associated with the release of oxytocin into the blood stream. Similarly, Tops, van Peer, Korf, Wijers, and Tucker (2007; also Marazziti et al., 2006) found attachment to be associated with oxytocin levels in the blood and to moderate the negative association between oxytocin and state anxiety. These studies, although based mainly on peripheral levels of oxytocin, suggest that the oxytocin receptor gene is another potential candidate for explaining aspects of attachment style.

Further support comes from animal studies in which the oxytocin receptor (OXTR) was found to regulate aspects of social behavior (Takayanagi et al., 2005) and anxiety (Amico, Mantella, Vollmer, & Li, 2004). Specifically, reduced oxytocin, brought about by "knocking out" the OXTR gene, was associated with impaired social, sexual, and maternal behavior (e.g., Takayanagi et al., 2005). However, there is little data concerning variations in OXTR in humans. The limited human data on OXTR and social behavior are based on studies of people with autism. The hypothesis guiding these studies is that disruptions in the oxytocin system contribute to social deficits associated with autism (Hammock & Young, 2006; Ylisaukko-Oja et al., 2005). Wu et al. (2005) found that a specific polymorphism—rs53576 A—is related to impaired social behavior associated with autism. Based on that finding, we hypothesized that rs53576 A would be associated with attachment insecurity, although we did not have sufficient previous data to suggest which attachment dimension it would be associated with.

To summarize, our hypotheses regarding specific genetic polymorphisms were as follows: (a) Polymorphisms of the DRD2 gene—specifically, having two copies of the A1 allele—will be associated with attachment anxiety or fearful avoidance (a combination of anxiety and avoidance); (b) polymorphisms of the 5HT2A serotonin receptor gene, and specifically the presence of the T102C polymorphism, will be correlated with avoidant attachment; and (c) the rs53576 A polymorphism of the OXTR oxytocin receptor gene will be associated with attachment anxiety and/or avoidance.

METHOD

Participants

One hundred forty-seven undergraduates (40 men, 107 women) participated in the study for course credit.[2] Their ages ranged from 18 to 29 years, with a median of 19 years; 44% were Asian or Asian American, 31% were Caucasian, 12% were Hispanic, 3% were African American, and 10% were Other. (Ethnicity was not associated with any of the genetic polymorphisms and is not further considered in this article.)

Materials and Procedure

Participants completed the ECR inventory (Brennan et al., 1998), a two-dimensional, 36-item measure of attachment anxiety and avoidance. They rated the extent to which each item was descriptive of their experiences in close relationships, using a 7-point scale ranging from 1 (*not at all*) to 7 (*very much*). Eighteen items tapped attachment anxiety (e.g., "I worry about being abandoned") and 18 tapped avoidant attachment (e.g., "I find it difficult to allow myself to depend on close relationship partners"). The reliability and validity of the scales have been repeatedly demonstrated (e.g., Brennan et al., 1998; Mikulincer & Shaver, 2007,

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chap. 4). In the present study, Cronbach alphas were high for both anxiety and avoidance (.92 and .87, respectively), and the correlation between the two scales was low and nonsignificant, as intended (r = .10, *ns*). Higher scores on one or both dimensions indicate attachment insecurity; low scores on both dimensions indicate security.

Participants also completed the Big Five Inventory (BFI; Benet-Martinez & John, 1998) to rule out alternative explanations of the findings. The BFI is a 44-item instrument that assesses the five major personality traits (Neuroticism, Extraversion, Openness, Agreeableness, and Conscientiousness). Items are rated on a 5-point Likert-type scale ranging from 1 (*disagree strongly*) to 5 (*agree strongly*). BFI scales include 8 to 10 items each, have moderate to high internal consistency reliability (in the present study, alphas ranged from .73 to .84), and converge well with other measures of the Big Five traits (John & Srivastava, 1999).

Upon completion of these scales, participants answered demographic questions and provided a saliva sample for the genetic analyses. They were then debriefed and thanked for participating.

Genetic collection and analyses. The saliva samples were processed at the Genomics Facility of the University of California, Davis. The DNA samples were obtained using the MasterAmp Buccal Swab kit (Epicentre Inc.). DNA was extracted according to the protocol provided by the company. A Polymerase Chain Reaction (PCR) was used to amplify the DNA sequences of interest. We then examined associations between the attachment dimensions and the three genetic polymorphisms related to dopamine, serotonin, and oxytocin receptors.

DRD2 gene. PCR primers used to test for the presence of the A1 allele of the dopamine D2 receptor (DRD2) gene were based on previous studies (Kaiser, Tremblay, Klufmöller, Roots, & Brockmöller, 2002). The primer sequences are 5'-CCGTCGACGGCTGGCCAAGTTGTCTA-3' and 5'-CCGTCGACCCTTCCTGAGTGTCATCA-3'. These primers amplify a 294 base-pair region of the DRD2 gene that contains a *Taq*1 recognition site. The allele was detected by digesting the PCR products with *Taq*1 restriction enzyme followed by separation with agarose gel electrophoresis.

Serotonin receptor. The T102C polymorphism of the serotonin receptor 2A gene (HTR2A) was genotyped under conditions previously reported (Quist et al., 2000), using forward 5' TCTGCTACAAGTTCTGGCTT-3' and reverse 5'-CTGCAGCTTTTTCTCTAGGG-3' primers. The 342 base-pair product was digested by *Msp*I and separated in agarose gel. Allele C was not digested, and allele T was digested into two fragments of 216 and 126 base pairs.

Oxytocin receptor. PCR primers for the single nucleotide polymorphism (rs53576; G/A) of the oxytocin receptor gene (OXTR) were used, as described by Wu et al. (2005). The sequences were 5'-GCCCACCATGCTCTCCACATC-3' and 5'-GCTGGACTCAGGAGGAATAGGGAC-3'. The 340 base-pair PCR products were digested with *Bam*HI and separated on agarose gel to identify the targeted polymorphism.

RESULTS

Sixteen of the 147 participants had two copies of the DRD2 A1 dopamine receptor allele (a condition we refer to as A1A1), 61 had one copy of it (A1A2), and 70 had no copies of it (A2A2). Forty-six of the 147 participants had a pair of HTR2A T serotonin T102C alleles (here labeled TT), 59 had one T allele and one C allele (labeled TC), and 42 had a pair of C alleles (labeled CC). On the oxytocin receptor gene (OXTR), 47 of the 147 participants had a pair of rs53576 A alleles (which we refer to as the AA group), 60 had one A allele and one G allele (labeled AG), and 40 had a pair of G alleles (labeled GG).

To examine whether differences in these alleles were related to attachment anxiety or avoidance, we conducted two univariate analyses of variance (ANOVAs), one for each attachment dimension. The three sets of allele categories (which we call dopamine, serotonin, and oxytocin, for short) served as the independent variables.

The analysis for attachment anxiety yielded a significant main effect of dopamine on attachment anxiety, $F(2, 108) = 3.23$, $p < .05$. No other main effects or interactions were significant. When this effect was examined with a simple one-way ANOVA, recapturing the degrees of freedom used in the first analysis to test the effects of the serotonin and oxytocin gene polymorphisms, it was again statistically significant, $F(2,108) = 3.78$, $p < .05$. Scheffé post hoc tests indicated that participants with two A1 alleles, as hypothesized, scored higher on attachment anxiety (M = 4.37, SD = 0.66) than participants with only one A1 allele (M = 3.67, SD = 0.77) or none (M = 3.50, SD = 0.84; both ps < .05). (The latter two groups did not differ significantly from each other.)

Because attachment anxiety is correlated with the Big Five personality trait of neuroticism (the correlation was r = .25, $p < .001$, in the present study; see full correlation matrix in Table 1 and means and SDs in Table 2), we ran the analysis again, this time as an analysis of covariance predicting attachment anxiety from both the dopamine variable and neuroticism. Although neuroticism was significantly related to attachment anxiety,

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TABLE 1: Zero-Order Correlations Among Attachment Scores and Big Five Trait Scores

	Avoidance	*Anxiety*	*Agreeableness*	*Conscientiousness*	*Openness*	*Neuroticism*	*Extroversion*
Avoidance		.09	–.05	–.11	.10	–.01	–.10
Anxiety			–.03	–.07	.06	.25**	–.16
Agreeableness				.44**	–.08	–.16	.17
Conscientiousness					.06	–.02	.26**
Openness						–.11	.18*
Neuroticism							–.03
Extroversion							

TABLE 2: Means and Standard Deviations of Attachment Variables and Big Five Trait Dimensions

	Avoidance	*Anxiety*	*Agreeableness*	*Conscientiousness*	*Openness*	*Neuroticism*	*Extroversion*
M	3.02	3.70	3.79	3.53	3.35	3.15	3.28
SD	0.89	0.82	0.60	0.67	0.65	0.74	0.69

$F(1,126) = 6.20$, $p < .05$, the dopamine allele pattern also continued to yield a significant effect, $F(2,126) = 5.50$, $p < .01$. Thus, the effect of the A1A1 polymorphism of the dopamine receptor gene on attachment anxiety was not redundant with genetic influences on the correlated variable, neuroticism.[3]

The initial three-way ANOVA for avoidant attachment revealed only a significant main effect of serotonin, $F(2, 49) = 3.15$, $p < .05$. The main effect of serotonin was explored further in a simple one-way ANOVA followed by Scheffé post hoc tests. The main effect was again significant, $F(2,70) = 3.24$, $p < .05$, and the Scheffé tests revealed that participants with a pair of T alleles scored higher on avoidant attachment ($M = 3.30$, $SD = 0.91$) than those with the CC combination ($M = 2.81$, $SD = 0.95$), but not those with the TC combination ($M = 2.94$, $SD = 0.79$). The latter two means did not differ significantly from each other.

Because avoidant attachment is often negatively related to the Big Five dimension of agreeableness (although the correlation was only $r = –.05$, *ns*, in the present study), we once again analyzed the effect of the serotonin allele pattern on avoidant attachment while controlling statistically for agreeableness. Agreeableness was not at all associated with avoidance, as already mentioned, $F(1,126) = .36$, *ns*; and the dopamine allele variable continued to have a significant effect, $F(2,126) = 4.55$, $p < .05$. Thus, the apparent effect of the TT pattern of the serotonin receptor gene on avoidant attachment is not redundant with genetic influences on agreeableness.[4]

DISCUSSION

The results provide preliminary support for connections between polymorphisms of certain neurotransmitter and neuromodulator receptor genes and the two major dimensions of attachment insecurity: anxiety and avoidance. As expected, attachment anxiety was associated with the presence of two copies of the A1 allele of the DRD2 dopamine receptor gene, such that participants with two A1 alleles scored higher on attachment anxiety than those with either one copy or none. Also as expected, the TT pattern of alleles on the serotonin HTR2A receptor gene was associated with avoidant attachment, such that participants with two copies of the T allele scored higher on avoidance than those with only one or none. Variants of the OXTR oxytocin receptor gene were not related to either attachment anxiety or avoidance.

Genetic polymorphisms that had previously been associated with general anxiety were also related to attachment anxiety, and genetic polymorphisms associated with social withdrawal were related to avoidant attachment. Thus, our results fit nicely with Gingrich et al.'s (2000) findings regarding prairie voles and the importance of DRD2 receptors in the nucleus accumbens for social attachment, and with proposals by Beech and Mitchell (2005) and by Hennighausen and Lyons-Ruth (2006) about the role of genes in determining attachment patterns.

This is not to say that the attachment dimensions are simply redundant with more general personality traits, however, because the connections between genetic polymorphisms and attachment insecurities remained significant when the relevant Big Five trait scores were statistically controlled, and the trait scores themselves were not influenced by the polymorphisms we examined. The results do suggest, however, that there may be genes or genetic polymorphisms that predispose a person to one kind of attachment insecurity rather than another, a conclusion also reached by Crawford et al. (2007) and Donnellan et al. (2008), based on behavior genetic studies of twins, and by Bakermans-Kranenburg and van

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IJzendoorn (2008), who studied genetic polymorphisms related to parental responsiveness.

Our findings, while implicating genes as a factor in attachment insecurity, do not contradict the general conclusion in the attachment literature (e.g., Bokhorst et al., 2003; O'Connor & Croft, 2001; Roisman, & Fraley, 2006) that early relationships with caregivers play a major role in determining a person's attachment style. The genetic polymorphisms we studied explained less than 20% of the variance in attachment anxiety and avoidance. As suggested by Fox, Hane, and Pine (2007), the combination of a certain genotype and a certain history of experiences with major attachment figures may explain the development of particular attachment styles. Of course, we did not examine every relevant genetic locus or polymorphism, so additional variance in attachment style may be explained by future genetic studies.

The fact that there was not a unique main effect of oxytocin polymorphisms may mean that oxytocin, which has previously been linked with attachment per se (i.e., with becoming attached rather than with the quality or security of the attachment), is related only indirectly to differences in adult attachment style and has more to do with caregiving style than attachment style (Bakermans-Kranenburg & van IJzendoorn, 2008). But it may also mean that OXTR is not the best candidate for relating to attachment style. Future studies should explore other oxytocin receptor polymorphisms such as rs2254298 and BTA13.

Our findings support Torgersen et al.'s (2007) speculation that genetic factors may influence attachment patterns in adulthood more than in infancy, whereas shared environmental factors may have more importance in infancy. Longitudinal studies have shown that genetic factors can affect a particular personality trait differently at different ages (Plomin, DeFries, McClearn, & McGuffin, 2000). It is also possible that twin studies of infants contain considerable error. Each twin has to be tested in laboratory interactions with his or her mother, and the mother is not naïve to the procedure the second time around. Also, the infant who is not being tested first has to be separated from the mother while she is observed interacting with the other infant. These procedural complexities may cause twin siblings to seem more different in attachment style than they really are.

While preliminary and in need of replication, our findings suggest that we will understand the roots of adult attachment styles more thoroughly if we take genetic polymorphisms into account. Given nonoptimal parenting, a person's form of attachment insecurity may be influenced by genetic propensities. Future studies should examine other candidate genes and polymorphisms, such as the catechol-o-methyltransferase gene, which has been associated with approach and avoidance tendencies (Reuter, Schmitz, Corr, & Hennig, 2006); the UCNIII gene, which has been associated with the attenuation of anxious behavior (e.g., Venihaki et al., 2004); and the GABRA6 gene, which is related to mood disorders (e.g., Uhart, McCaul, Oswald, Choi, & Wand, 2004). Future studies should also examine possible interactions of various genes and polymorphisms.

Nevertheless, the present study is a valuable stepping stone on the way to a better understanding of adult attachment behavior. Attachment researchers have thus far emphasized parenting almost to the exclusion of other determinants of attachment patterns, and this may place more pressure on parents than is justified. As with other innate behavioral systems, which is what Bowlby (1982) thought the attachment behavioral system is, there is likely to be a mixture of innate and experiential forces determining how the attachment system develops. What may be needed for optimal development is not a one-size-fits-all form of parenting but forms of parenting appropriate for each child's innate propensities. Mapping those likely interactions and deciphering their implications for parenting is an important task for the future.

NOTES

1. Although we focused on these three polymorphisms of dopamine, serotonin, and oxytocin receptor genes, we obviously do not rule out the possibility that other allelic variations in these genes, or allelic variations in other genes, such as the X-linked monoamine oxidase A (MAOA) gene, COMT, GABRA6 gene, urocortin III, or other genes and polymorphisms mentioned at the end of this article might also affect the development of attachment style. This should be explored in future studies.

2. We collected data from more participants than 147, but 15 saliva samples could not be processed fully because of low quality.

3. We also ran two similar univariate analyses of variances predicting neuroticism and agreeableness (see the Results section) from dopamine, serotonin, and oxytocin allele patterns. No main effects or interactions were found in these analyses, suggesting that the specific genetic polymorphisms we studied are more predictive of attachment dimensions than of general personality traits.

4. In fact, as explained in Note 3, the gene polymorphisms we studied were not significantly associated with agreeableness.

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