The dopamine D2 receptor gene, perceived parental support, and adolescent loneliness: longitudinal evidence for gene–environment interactions

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Background: Loneliness is a common problem in adolescence. Earlier research focused on genes within the serotonin and oxytocin systems, but no studies have examined the role of dopamine-related genes in loneliness. In the present study, we focused on the dopamine D2 receptor gene (DRD2). Methods: Associations among the DRD2, sex, parental support, and loneliness were examined in a longitudinal study spanning five annual waves (N = 307). Results: Using Latent Growth Curve Modeling, DRD2 genotype was not directly related to loneliness. Interactions were found between parental support and DRD2 genotype, showing that adolescents with the A2A2 genotype who perceived little support from their parents had the highest baseline levels of loneliness. Adolescents with an A1 allele were not susceptible to the rewarding effect of parental support. Conclusions: The present study is the first to examine the role of the DRD2 genotype in loneliness. Our results contribute to a further understanding of the environmental and genetic basis of loneliness in adolescence. Keywords: loneliness, dopamine D2 receptor gene, DRD2, parental support, gene–environment interaction, adolescence.

Introduction

Loneliness is a common experience that can be present in all phases of life, particularly in adolescence (Heinrich & Gullone, 2006). This experience is typically defined as the negative emotional response to a discrepancy between the desired and achieved quality and quantity of one’s social network (Perlman & Peplau, 1981). People can feel lonely because they have fewer friends compared with others or they can have a sufficient number of friends but feel dissatisfied with the overall quality of their relationships with these friends. Chronic loneliness has been related to Diagnostic and Statistical Manual of Mental Disorders (DSM)-based major depressive disorder, anxiety disorders, and schizophrenia (Heinrich & Gullone, 2006) and to physical health problems (e.g., cardiovascular disease and poorer immune functioning; Cacioppo et al., 2002). Therefore, it is important to examine its antecedents. The aim of the present study is to examine the predictors of loneliness in adolescence, with a focus on genetic factors (DRD2 genotype) and parental support.

Behavioral genetic studies have shown that loneliness is moderately heritable, with heritability estimates ranging between 48% and 55% (Boomsma, Willemsen, Dolan, Hawkley, & Cacioppo, 2005). Till date, however, only two molecular genetic studies have examined associations between loneliness and specific genes (Lucht et al., 2009; Van Roekel, Scholte, Verhagen, Goossens, & Engels, 2010). Lucht et al. (2009) found a relation between the oxytocin receptor gene (OXTR) and emotional loneliness in adults, whereas Van Roekel et al. (2010) found a significant association between the serotonin transporter gene (5-HTTLPR) and the development of loneliness throughout adolescence. Although these studies provide interesting insights, additional genes could be involved in loneliness as well.

A recent theory on loneliness states that lonely people do not enjoy positive events as much as nonlonely individuals do (Hawkley & Cacioppo, 2010). For example, a diary study has shown that individuals high on loneliness experienced less reward from daily social interactions compared with individuals low on loneliness (Hawkley, Preacher, & Cacioppo, 2007). Experiences of reward and enjoyment are typically associated with the dopamine system in the brain (e.g., Blum et al., 1996). A Functional Magnetic Resonance Imaging (fMRI) study on loneliness found that lonely people showed less activation in the ventral striatum in response to positive social stimuli compared with nonlonely people (Cacioppo, Norris, Decety, Monteleone, & Nusbaum, 2009). Within this system, the dopamine receptor D2, which is regulated by the DRD2 gene, is of interest (Blum et al., 1996). The A1 allele of the DRD2 gene was found to be associated with reduced dopamine D2 receptor binding in the ventral striatum, a region of the brain that is...
involved in reward mechanisms (Thompson et al., 1997). It is assumed that this reduced D2 receptor binding leads to experiencing less reward (Blum et al., 1996). This was also shown in an fMRI study (Cohen, Young, Baek, Kessler, & Ranganath, 2005) in which participants carrying at least one A1 allele showed lower responses in brain areas involved in reward mechanisms (e.g., nucleus accumbens) when presented with a reward. These findings indicate that people carrying at least one A1 allele have reduced binding of DRD2 receptors in the brain reward system and are less sensitive to rewards compared with people homozygous for the A2 allele. As lonely people showed lower levels of reward in response to positive social stimuli, this might suggest lower D2-binding capacities in certain brain areas. Therefore, we expected that the DRD2 genotype would be related to loneliness.

To our knowledge, the existing research has not examined direct effects of the DRD2 gene on loneliness; however, mixed findings were obtained regarding depression, a condition related to loneliness. Some studies (Audrain-McGovern, Lerman, Wileyto, Rodriguez, & Shields, 2004; Guo & Tillman, 2009; Lawford, Young, Noble, Kann, & Ritchie, 2006) found that A1 carriers evidenced more depressive symptoms, but this effect was not confirmed in other studies (e.g., Elovainio et al., 2007). Such discrepant findings are common in molecular genetic studies (e.g., Lin, Vance, Pericak-Vance, & Martin, 2007).

Most studies on the determinants of loneliness in adolescence have focused on environmental factors. A meta-analysis (Mahon, Yarcheski, Yarcheski, Cannella, & Hanks, 2006) revealed that the most important environmental predictors of loneliness were social support and both maternal and paternal expressive sensitivity. More specifically, several studies find that high levels of both maternal and paternal support prevent feelings of loneliness in adolescence (Franzoi & Davis, 1985; Mounts, Valentinier, Anderson, & Boswell, 2006). Several explanations are offered for this association. High levels of parental support indicate an overall closer parent-adolescent relationship in general and higher levels of communication, allowing adolescents to talk about their feelings of loneliness with their parents (Franzoi & Davis, 1985; Mounts et al., 2006). Next to direct environmental influences, direct gene effects on multifactorial forms of psychopathology, such as depression, anxiety, and loneliness (Rutter et al., 1997), are often small and hence difficult to detect. In addition, the effects of certain genes may only be expressed under particular environmental conditions, most often exposure to negative environments (Rutter, 2007). Until now, most studies have examined interactions between genes and negative environments. Studies examining interactions between stress and the DRD2 genotype have revealed inconsistent results. One study found that individuals with the (higher activity) A2A2 genotype were more affected by environmental stress, and as a consequence had higher levels of depression (Elovainio et al., 2007), whereas another study (Vaske, Beaver, Wright, Boisvert, & Makarios, 2009) found A1 carriers to be more depressed when they experienced environmental stress.

However, no studies on gene–environment interactions have reported on positive environmental influences, such as parental support. Other positive environmental variables, such as social cooperative behavior and social comparison, were also associated with activation in the ventral striatum (Fliesbach et al., 2007; Rilling et al., 2002). As A1 allele carriers have fewer D2 receptors in the ventral striatum (Thompson et al., 1997) and show lower responses to rewarding stimuli (Cohen et al., 2005), they may be less sensitive to these social types of reward as well. Therefore, we hypothesized that A1 carriers would be less susceptible to parental support, and that adolescents with the A2A2 genotype would benefit from higher levels of support.

The aim of our study was to examine the relationships of the DRD2 genotype, parental support, and DRD2 by parental support interactions with adolescent loneliness. We used a longitudinal five-wave design, which allowed us to examine relations with both the baseline level of loneliness and the development over time. As previous studies found differences between boys and girls in the level of loneliness (e.g., Koenig & Abrams, 1999; Van Roekel et al., 2010), we also examined sex differences. We did not have a specific hypothesis for the relation between DRD2 genotype and loneliness because the results from studies examining relations between DRD2 genotype and depression are inconsistent. For the gene–environment interactions, we expected that parental support would not affect A1 carriers, which would result in a nonsignificant relation between support and loneliness in this group. However, adolescents homozygous for the A2 allele would be susceptible to positive social rewards; therefore, they may benefit from higher levels of support. Hence, we expected that high levels of parental support would be related to lower levels of loneliness in adolescents with the A2A2 genotype.

Methods

Procedure

Data for the present study were derived from a longitudinal Dutch survey study called Family and Health with five annual waves which examined different socialization processes in relation to various health behaviors among adolescents and their families (Harakeh, Scholte, de Vries, & Engels, 2005). For a detailed description of the procedure, see Van der Zwaluw et al. (2008).

Participants

The present study used data from the youngest adolescents in each family. These adolescents were...
entering adolescence at T1, which allowed us to examine loneliness throughout adolescence. Our final sample consisted of 307 adolescents, of which 53.4% were girls. The mean age at T1 was 13.4 years (SD = .51). One-third (33.4%) of the adolescents attended lower education (i.e., preparatory secondary school for technical and vocational training), one-third (36.8%) intermediate general education (i.e., preparatory secondary school for college), and one-third (29.8%) attended the highest level of secondary school (i.e., preparatory secondary school for university). A small group of adolescents were not born in the Netherlands (1.2%), and of this group, 0.2% were not born in a European country.

Attrition analyses were conducted to examine whether adolescents who gave their consent for genotyping (participants; n = 307) differed from the adolescents who did not (dropouts; n = 121). t-tests showed no significant differences between participants and dropouts (p > .05) in loneliness, maternal support, or age. However, participants did experience a slightly lower level of paternal support compared with dropouts (t(426) = -2.164, p = .03). For educational level and sex, chi-squared statistics were calculated to examine differences in education and sex between dropouts and participants. Significant differences existed for educational level, indicating that participating adolescents had a higher level of education compared with dropouts (χ²[176] = 7.61, p = .02). No sex differences were found in retention (χ²[428] = 0.50, p = .48).

Measures

Loneliness. Loneliness was measured at five time points using 12 items from the peer-related subscale of the Louvain Loneliness Scale for Children and Adolescents (LLCA; Marcoen, Goossens, & Caes, 1987). Sample items were, ‘I feel abandoned by my friends’ and ‘I feel sad because I have no friends’. The items were measured on a 4-point scale ranging from never (1) to always (4). Scores ranged from 12 to 48, with higher scores indicating higher levels of loneliness. Cronbach’s alpha ranged from .91 to .93 at different time points.

Perceived parental support. Participants completed a 12-item version of the Relational Support Inventory (RSI; Scholte, van Lieshout, & van Aken, 2001) at T1. The scale measures aspects of emotional and instrumental support. Participants completed the questionnaire for fathers and mothers separately. The questionnaires for maternal and paternal support were identical. Example items are: ‘My mother/father supports me in the things I do’, and ‘My mother/father explains or shows how I can make or do something’. Each item was rated on a 5-point Likert scale ranging from very untrue (1) to very true (5). Alpha was .77 for maternal support, and .80 for paternal support.

**DRD2 genotyping.** The DRD2 Taq1 A C>T polymorphism was genotyped using Taqman analysis (assay ID: Taqman assay: C__7486676_10; reporter 1: VIC-A-allele, reverse assay; Applied Biosystems, Nieuwerkerk a/d IJssel, the Netherlands). Genotyping was carried out in a volume of 10 µl containing 10 ng of genomic DNA, 5 µl of Taqman Mastermix (2×; Applied Biosytems), 0.125 µl of the Taqman assay, and 3.875 µl of H₂O. Genotyping was performed on a 7500 Fast Real-Time PCR System and genotypes were scored using the algorithm and software supplied by the manufacturer (Applied Biosystems).

To investigate the random genotyping error rate, the lab included five duplicate DNA samples per 96-well plate, which were 100% consistent. In addition, four blanks were included in each plate, which were required to be negative. By running PEDCHECK (O’Connell & Weeks, 1998) for single-point Mendelian inconsistencies on the markers, we identified one family with potential pedigree errors. This family was removed from the analysis. Hardy–Weinberg equilibrium (HWE) proportions were estimated from parental genotype information using the Markov chain Monte Carlo approximation of the exact test implemented in the GENEPOP package V3.3 (Raymond & Rousset, 1995). No deviations from HWE were detected (p = .96). To maximize the power of the analyses, DRD2 genotype was dummy-coded into 0 (A1A2 and A1A1) and 1 (A2A2).

Statistical analyses

Latent Growth Curve Modeling (LGCM) was used to estimate both the individual level of loneliness at baseline (i.e., intercept), and the change in loneliness over time (i.e., slope; Duncan, Duncan, & Strycker, 2006). In this approach, it is not assumed that all participants start at the same level of loneliness at baseline and have the same rate of change in loneliness over time; instead, individual growth is examined for each participant. Therefore, LGCM is an excellent way to examine individual variation in the development of loneliness and to investigate whether certain predictors relate to these changes over time. Mplus (Muthén & Muthén, 1998–2007), a statistical software program designed for Structural Equation Modeling (SEM) analyses, was used for these analyses. Parameters in the models were estimated by applying a method that corrects for the non-normal distribution of the dependent variables. This method is referred to as the maximum likelihood estimator with robust standard errors or MLR. To deal with missing data, which were rare, we did not impute these data but borrowed information from the observed portion of the data. This approach (which is referred to as the full-information maximum likelihood or FIML approach) is superior to other techniques for handling missing data, such as multiple imputation, pairwise deletion, or listwise deletion (Muthén & Muthén, 1998–2007).

First, we tested the initial developmental model, estimating the intercept (or initial level) and slope (or rate of change) in loneliness regardless of genetic or environmental influences. Second, we examined the relation between the DRD2 genotype and the initial or baseline level and rate of change in loneliness (Model 2). Third, we examined the main effects of maternal and paternal support on loneliness (Models 3 and 4). Finally, we examined the interactions between parental support and the DRD2 genotype (Models 5 and 6). To avoid multicollinearity, all variables were centered before computing the interaction terms. Model fit was assessed by the following global fit indices: χ²,
Confirmatory Fit Index (CFI) (with a cut-off value of .95), and Root Mean Square Error of Approximation (RMSEA) (with a cut-off value of .06; Hu & Bentler, 1999).

Results

Descriptive statistics

Of the 307 participants, 205 (66.3%) were homozygous for the A2 allele, 96 (31.1%) had the heterozygous genotype, and 8 (2.6%) were homozygous for the A1 allele. Table 1 shows means, standard deviations, and Pearson correlations among model variables. The average level of loneliness across the five time points was relatively low in absolute terms, with means ranging from 17.67 to 18.81 (out of a maximum score of 48). These means were comparable with other studies on community samples (Marcoen & Goossens, 1993). The loneliness scores ranged from 12 to 48 at T1, 12 to 45 at T2, 12 to 38 at T3, 12 to 46 at T4, and 12 to 44 at T5. Sex was dummy-coded (0 = boys; 1 = girls) so that the average score for sex in Table 1 reflects the proportion of girls in the sample. Concurrent correlations at T1 showed a significant negative association between loneliness and parental support, as expected. Support from both father and mother were negatively related to loneliness at most waves. The correlations showed that the DRD2 genotype and sex did not relate to loneliness. No sex differences were found for any of the measures.

Model findings

First, we tested the initial developmental model (i.e., no predictors; Table 2). The intercept and slope were significant, indicating that participants scored, on average, 18.88 for loneliness at baseline and that the level of loneliness decreased significantly over time.

Second, the DRD2 genotype was included as a predictor in the model, but it was not significantly related to intercept or slope. To examine whether the intercept and slope differed for boys and girls, we included sex as a predictor. The results showed a significant relation between sex and the slope, indicating that girls remained relatively stable in loneliness over time, whereas loneliness decreased among boys. No relation was found between sex and baseline levels of loneliness. As a result of the sex differences in the slope of loneliness, we tested whether the DRD2 genotype explained this relation by adding the interaction between sex and the DRD2 genotype to the model. This interaction was not related to the intercept of loneliness or the slope. Third, we examined relations between parental support and loneliness. We examined direct relations between parental support and loneliness first while controlling for sex.

Table 1 Correlations among model variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>SD</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. DRD2 or A2</td>
<td>.34</td>
<td>.47</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Sex</td>
<td>.53</td>
<td>.50</td>
<td>.01</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>3. Loneliness (T1)</td>
<td>18.81</td>
<td>6.60</td>
<td>.01</td>
<td>-.06</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Loneliness (T2)</td>
<td>18.69</td>
<td>6.44</td>
<td>-.03</td>
<td>.08</td>
<td>.56**</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>5. Loneliness (T3)</td>
<td>18.31</td>
<td>6.48</td>
<td>-.01</td>
<td>.52**</td>
<td>.61**</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>6. Loneliness (T4)</td>
<td>18.08</td>
<td>6.40</td>
<td>.06</td>
<td>.41**</td>
<td>.48**</td>
<td>.53**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Loneliness (T5)</td>
<td>17.67</td>
<td>6.69</td>
<td>.11</td>
<td>.49**</td>
<td>.39**</td>
<td>.47**</td>
<td>.70**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Support (mother)</td>
<td>4.11</td>
<td>4.0</td>
<td>.10</td>
<td>.09</td>
<td>-.18**</td>
<td>-.07</td>
<td>-.18**</td>
<td>-.18**</td>
<td>-.15**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Support (father)</td>
<td>3.92</td>
<td>4.7</td>
<td>-.02</td>
<td>.11</td>
<td>-.20**</td>
<td>-.41</td>
<td>-.18**</td>
<td>-.08</td>
<td>-.04</td>
<td>.61**</td>
<td></td>
</tr>
</tbody>
</table>

*p = TT (A1A1) and TC (A1A2); 1 = CC (A2A2).

Table 2 Regression of initial level (intercept) and rate of change (slope) in adolescents’ loneliness on Gene × Environment interactions

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Intercept</th>
<th>Slope</th>
<th>$\chi^2$ (df)</th>
<th>CFI</th>
<th>RMSEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Initial developmental model</td>
<td>18.88 (0.35)**</td>
<td>-2.9 (0.10)*</td>
<td>37.81 (10)</td>
<td>.92</td>
<td>.095</td>
</tr>
<tr>
<td>2. DRD2</td>
<td>-0.3 (0.07)</td>
<td>.11 (0.08)</td>
<td>40.50 (13)</td>
<td>.93</td>
<td>.083</td>
</tr>
<tr>
<td>3. Sex</td>
<td>-0.4 (0.07)</td>
<td>.19 (0.08)*</td>
<td>48.13 (16)</td>
<td>.93</td>
<td>.081</td>
</tr>
<tr>
<td>4. DRD2 × Sex</td>
<td>.49 (0.28)</td>
<td>-.24 (0.32)</td>
<td>55.07 (19)</td>
<td>.92</td>
<td>.079</td>
</tr>
<tr>
<td>5. Maternal support</td>
<td>-.18 (0.07)**</td>
<td>-.05 (0.10)</td>
<td>52.79 (16)</td>
<td>.92</td>
<td>.087</td>
</tr>
<tr>
<td>6. DRD2 × Maternal support</td>
<td>.67 (0.17)***</td>
<td>-.65 (0.28)*</td>
<td>55.66 (22)</td>
<td>.93</td>
<td>.071</td>
</tr>
<tr>
<td>7. Paternal support</td>
<td>-.25 (0.07)**</td>
<td>.17 (0.10)</td>
<td>48.48 (16)</td>
<td>.92</td>
<td>.081</td>
</tr>
<tr>
<td>8. DRD2 × Paternal support</td>
<td>.62 (0.19)***</td>
<td>-.75 (0.26)**</td>
<td>51.31 (22)</td>
<td>.94</td>
<td>.066</td>
</tr>
</tbody>
</table>

In all analyses, we controlled for sex. Only new variables entered in the model are depicted in the table. DRD2, dopamine D2 receptor gene; df, degree of freedom.

*p < .05; **p < .01; ***p < .001.

Both maternal and paternal support were negatively related to the intercept but not to the slope. Low levels of support were related to high levels of loneliness at baseline. In the next step, we examined the interactions between parental support and the DRD2 genotype for maternal and paternal support separately. For both maternal and paternal support, the interaction was significantly related to the intercept of loneliness, as depicted in Figure 1. (Because the figures were similar for paternal and maternal support, only the figure for maternal support is shown.) No relation existed between parental support and baseline levels of loneliness for adolescents who carry at least one A1 allele, whereas low parental support in adolescents with the A2A2 genotype was related to higher levels of loneliness at baseline.

The interaction was also significantly related to the slope of loneliness, for both maternal and paternal support. Parental support did not relate to the slope of loneliness in adolescents carrying at least one A1 allele. However, in adolescents with the A2A2 genotype, lower levels of parental support were related to a faster decrease in loneliness over time. As seen in Figure 2 (paternal support; the results were similar for maternal support), this effect was due partly to the high levels of loneliness at baseline for adolescents who experienced low levels of support. These adolescents started with the highest levels of support at baseline, subsequently decreased at a faster rate, but still ended up with higher levels of loneliness at T5 compared with adolescents who experienced medium or high levels of support. Finally, we conducted multigroup analyses to examine sex differences. These analyses showed no differences in effects between boys and girls.

Discussion
The aim of the present study was to examine the relations between DRD2 genotype, parental support, and loneliness in adolescence. We did not find a direct relation between DRD2 genotype and the baseline and rate of change in loneliness, which is in line with our hypothesis. We did find that boys decreased in loneliness at a faster rate than girls did, but the DRD2 genotype could not explain this relation as the interaction between sex and the DRD2 genotype was not significant. One possible explanation for the nonsignificant direct effects of DRD2 genotype in our sample is that direct relations between genes and complex mental problems are often small and therefore difficult to detect (Rutter, Moffitt, & Caspi, 2006).

Both paternal and maternal support were negatively related to the baseline level of loneliness. Adolescents who experienced low levels of support had the highest levels of loneliness at baseline. This was in line with our hypothesis and previous research (e.g., Franzoi & Davis, 1985; Mounts et al., 2006). However, the results from the gene environment interactions showed that this relation only holds for adolescents carrying the A2A2 genotype, as expected. In adolescents with the A2A2 genotype, a negative relation was found, in that adolescents who experienced low levels of support showed the highest levels of loneliness at baseline. Adolescents with an A1 allele were not affected by support from their father or from their mother. This is an important finding because it implies that adolescents with the A2A2 genotype may benefit from high levels of social support. It may be that these adolescents experience adequate levels of social reward when interacting with their parents, whereas adolescents with A1 genotypes run a risk for internalizing problems because of inadequate levels of social reward experienced in general. This lack of reward experience in A1 allele carriers may be caused by the reduced concentration of D2 receptors in the brain areas associated with reward (Thompson et al., 1997).

In addition to the relations with the intercept, adolescents with the A2A2 genotype who experienced the lowest levels of support, decreased in loneliness at a faster rate compared with adolescents experiencing high levels of support. At first glance, this is in contrast to our expectations. However,
Figure 2 shows that this result is due mainly to the negative effect of parental support on the baseline level of loneliness. A possible explanation for these results may be that adolescents who receive low levels of parental support try to compensate for this by seeking more support from other people, such as peers or friends (Scholte et al., 2001). Therefore, their loneliness levels may decrease at a faster rate but remain at high levels because parental support is still an important predictor of loneliness.

Theoretical implications

The results from the present study, combined with the results from previous molecular genetic studies on loneliness (Lucht et al., 2009; Van Roekel et al., 2010), provide important insights into the biological and genetic underpinnings of loneliness. In addition to these studies, a recent theory (Hawkley & Cacioppo, 2010) states that people who score high on loneliness (a) do not enjoy positive events as much as do nonlonely individuals and (b) show a hypervigilance for social threats. These combined characteristics lead to a self-reinforcing loop of increasingly negative social expectations that ultimately give rise to the negative sequelae of sustained loneliness.

Experiences of reward and enjoyment are typically associated with the dopamine system in the brain and the DRD2 gene. As our results show, individuals who carry at least one A1 allele are not susceptible to a particular type of social reward, that is, parental support. This may indicate that they also experience less joy from positive events, which is one of the characteristics of lonely people proposed in the former theory (Hawkley & Cacioppo, 2010). In addition, research on oxytocin has shown that increased levels of this neuropeptide are related to attachment, bonding, and reward dependence (Bora, Yucel, & Allen, 2009). This may indicate that oxytocin also plays a role in the lowered social reward experiences in lonely people, which may explain why a relation was found between the oxytocin receptor gene and emotional loneliness in adults (Lucht et al., 2009). Hypervigilance to threat is often associated with the serotonin system in the brain and, in particular, with peculiarities regarding the serotonin transporter, as regulated by the serotonin transporter gene (5-HTTLPR). Individuals who carry at least one copy of the short allele of this gene show an increased risk for hypervigilance to threat (Pezawas et al., 2005). More research on the specific mechanisms underlying the relations between these three genes and loneliness is warranted.

Clinical implications

The findings on gene–environment interactions in the current study may also inform intervention efforts for lonely people. As loneliness is jointly determined by genetic and environmental influences, it is vital for counselors and therapists to take into account the degree of perceived parental support. Adolescents who enjoy high levels of support experience lower levels of loneliness, at least when their capacity to enjoy social contacts is unimpaired. This additional source of support may be put to good effect in the actual interventions developed for lonely people.

Research on the genetic underpinnings of social cognition in lonely people may have clinical implications for interventions designed to reduce loneliness. A recent review of such efforts revealed that interventions that directly tackled the way in which lonely people think about social situations were more successful compared with other types of interventions (Masi, Chen, Hawkley, & Cacioppo, in press). Pending further research on the model described previously (Hawkley & Cacioppo, 2010), interventions may try to both decrease hypervigilance to social threat and teach lonely people to enjoy social contacts with other people more fully.

Limitations and suggestions

This study has some limitations that should be addressed. First, our sample scored relatively low on loneliness. This could be due to the inclusion criteria, which required that parents be married or living together. Moreover, participating family members had to be biologically related to each other. Because of these restrictions, our sample may have comprised relatively well-functioning families. Future research should focus on testing relations between DRD2 genotype and loneliness in clinical samples or at-risk groups in which support levels may be lower (Thomson, Hanson, & McLanahan, 1994). Second, we only examined parental support as an environmental influence, whereas other variables may also be important. For example, research has shown that peer-related variables, such as peer support (Domingovitch & Bierman, 2001) and self-disclosure to peers (Franzoi & Davis, 1985), play a role in adolescent loneliness. Future studies could examine those factors in relation to the DRD2 genotype and loneliness. In addition, genetic factors may also influence parental support, as previous research on parenting has shown (e.g., Van IJzendoorn, Bakermans-Kranenburg, & Mesman, 2008). However, we did not find a correlation between parental support and the adolescents’ genotype; therefore, we may conclude that this has not influenced our analyses. Third, the participating adolescents completed both the loneliness and parental support measures. Correlations between these measures, therefore, may partially reflect shared method variance. Still, it has been suggested that the adolescents’ perception of the support they receive may be more important than the actual support that parents provide (Steinberg, Lamborn, Dornbusch, & Darling, 1992). Fourth, there are some inherent problems associated with gene–environment interaction research. Typically,
this type of interaction is difficult to replicate, and replications of an initially positive result always require larger samples than used in the original positive study (Rutter, 2006). Therefore, it is important to emphasize that replication of the findings of the present study is needed in both population-based and clinical samples.

Another suggestion for further research is to examine the effects of DRD2 genotype on responses in reward brain areas in lonely people, because the ventral striatum might be implicated in loneliness (Cacioppo et al., 2009) and this brain area is an important site of D2 receptor density (Thompson et al., 1997).

Conclusion

Although we did not find a direct effect of the DRD2 genotype on loneliness, an important finding of our study was that adolescents homozygous for the A2 allele were more susceptible to social rewards and therefore experienced less loneliness when they received high levels of parental support. It is important to stress that replication of these findings is needed in both population-based and clinical samples.

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Key points

- Previous studies on loneliness have shown that loneliness has a substantial genetic component, but only two studies have examined the specific genes involved.
- In the present study, no relation was found between DRD2 genotype and the intercept and slope of loneliness.
- Adolescents with the A2A2 genotype who perceived little support from their parents had the highest baseline levels (intercept) of loneliness, whereas adolescents with an A1 allele were not susceptible to the rewarding effect of parental support.
- For adolescents with the A2A2 genotype it was also found that lower levels of parental support were related to a faster decrease (slope) in loneliness over time. However, they still ended up with the highest levels of loneliness.

References


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