Research report

Parental control and the dopamine D2 receptor gene (DRD2) interaction on emotional eating in adolescence

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The present study addresses the emergence of emotional eating in adolescence in relation to maternal or paternal psychological control. A reduction of food intake is considered the biological natural response to distress, therefore we tested whether the atypical stress response of emotional eating develops in interaction with genetic vulnerability. Carrying the A1 allele of the dopamine D2 receptor (DRD2) gene Taq1A polymorphism (rs1800497) is associated with reduced dopamine D2 receptor availability in the brain. We hypothesized that carrying this allele would confer risk for the development of emotional eating, particularly so in adolescents with adverse rearing experiences. Participants were 279 Dutch adolescents (average age of 13.4) that participated in a prospective study with a four-year follow-up. We found a moderator effect of DRD2 genotype on the relation between both maternal and paternal psychological control and increases in emotional eating in both sexes. Adolescents showed only an increase in emotional eating in relation to high psychological control if they carried at least one DRD2 A1 allele. This study is the first to show that the relationship between adverse rearing experiences and emotional eating might be dependent on genetic make-up.

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Introduction

Distress induced eating – emotional overeating (i.e. eating in response to negative emotions such as depressive feelings) – is highly prevalent in adults who are binge eaters or obese (Bohon, Stice, & Spoor, 2009; van Strien, Engels, van Leeuwe, & Snoek, 2005; van Strien, Herman, & Verheijden, 2009) but has only a low prevalence in young children (Sleddens, Kremers, & Thijss, 2008; van Strien & Oosterveld, 2008; Wardle, Carnell, Haworth, & Plomin, 2008). This would suggest that most young children show the biologically natural response to distress (loss of appetite), since the normal distress response is associated with physiological reactions that are designed to prepare the individual for a fight or flight reaction, thereby suppressing feelings of hunger and satiety (Gold & Chrousos, 2002). It has been suggested that the biologically unnatural response to distress of emotional eating is acquired as a possible result of inadequate parenting (Bruch, 1973; Snoek, Engels, Janssens, & van Strien, 2007). The present study addresses the emergence of emotional eating in early adolescence in relation to the inadequate parenting practice of psychological control.

Adverse rearing experiences early in life may indeed have lasting effects on stress-responsive neurobiological systems, particularly when they pertain a perturbed parent–infant relationship (Cicchetti & Rogosch, 2001; Yehuda et al., 2000). This may include a hypoactivation, rather than hyperactivation of the hypothalamic–pituitary–adrenal axis (HPA) axis, with reverse neurovegetative symptoms: increased food intake (hyperphagia) and weight gain, instead of hypophagia and weight loss (Gold & Chrousos, 2002). These alternations generally do not become manifest before puberty (Silveira, Portella, Goldani, & Barbieri, 2007).

Parental psychological control can be conceived as an adverse rearing experience, because this manipulative and suppressive type of parental control includes guilt induction and withdrawal of love, thereby intruding into the psychological and emotional development of the child (Barber, 1996). There is ample evidence that this parenting practice is associated with delinquency (Hoeve et al., 2009) and internalizing problems in adolescents (Petit, Laird, Dodge, Bates, & Criss, 2001; Shek & Lee, 2005). Further, paternal psychological control was shown to be associated with emotional symptoms, difficulty in emotional regulation and eating disorder symptoms (McEwen & Flouri, 2009). Additionally, in a study on young adolescents, both maternal and paternal psychological control were indeed related to the adverse stress response of emotional eating (Snoek et al., 2007), though, as with other
adverse experiences (Miller, Chen, & Zhou, 2007), effects were only small. Evidently not all people with adverse rearing experiences develop emotional eating patterns, suggesting a possible role for genetics.

There is some evidence that the dopamine system, especially the dopamine D2 receptor (DRD2) gene may be involved in the pathogenesis of emotional eating (Davis et al., 2008; Volkow et al., 2003). Scholars disagree, however, on whether emotional eating reflects a deficit of reward (Volkow et al., 2003) or a heightened sensitivity to reward (Davis, Strachan, & Berkson, 2004). In a study on brain dopamine measures, emotional eating was negatively associated with baseline dopamine D2 receptor availability in the dorsal striatum (Volkow et al., 2003). This finding was taken to support the view that hypo-dopaminergic functioning underlies the development of emotional eating which would explain why emotional eaters are more receptive to the reinforcing value of food (Volkow et al., 2003), and use food as ‘self-medication’ to blunt effects of negative emotions. Reduced brain dopamine of emotional eaters has, however, also been explained as possible outcome of an adaptive downregulation of the dopaminergic system: a reflection of a neuroadaptation secondary to over-stimulation with food as result of a heightened sensitivity to reward (Bohon et al., 2009; Davis et al., 2004).

In the present study, we will prospectively examine the moderator effect of the DRD2 genotype in relation to maternal versus paternal psychological control on the emergence of emotional eating in adolescence. Since emotional eating has a female preponderance (van Strien, 2002), we were also interested in a possible moderator effect of sex.

Using a longitudinal four-year follow-up design we examine the change in emotional eating from early to middle adolescence. We hypothesized that DRD2 genotype, like psychological control, has a main effect on emotional eating at follow-up (T2). Additionally we hypothesized that DRD2 genotype acts as a moderator, in that the relationship between psychological control and emotional eating would be stronger if adolescents carried at least one A1 allele. We also hypothesized that the moderator effect of DRD2 genotype on the relation between psychological control and emotional eating is stronger in girls than in boys.

To get an indication of the direction of causality between reward sensitivity and emotional eating and to reduce the risk that a history of overeating would explain any prospective results, we controlled in our prospective analyses not only for initial emotional eating, but also for initial BMI. Additionally, to rule out possible effects of modeling and personality, we controlled for parental emotional eating and parental overweight and personality and depressive feelings of the child. In earlier research, emotional eating and overweight of parents and children were found to be interrelated (Snoek et al., 2007; Wardle et al., 2008). Further, emotional eating was found to be associated with personality and depressive feelings (Heaven, Mulligan, Merrillees, Woods, & Fairoz, 2001; Ouwens, van Strien, & van Leeuwe, 2009), which are in turn related to genetics (Krueger, South, Johnson, & Iacono, 2008). In additional analyses, we controlled for possible confounding effects of the other eating styles (external eating; eating in response to external food-related cues such as sight and smell of attractive food) and restrained eating; eating less than desired to maintain or loose body weight), and other styles of parenting (support and behavioral control).

Methods

Participants and procedure

Participants were 428 Dutch adolescents with an average age of 13.4 years (SD = 0.6) at baseline measurement. The adolescents participated with their parents and older sibling in the longitudinal Family and Health study, which was designed in 2002 to measure various socialization processes underlying health-related adolescent behaviors (van der Vorst, Engels, Meeus, Dekovic, & van Leeuwe, 2005; van der Zwaluw et al., 2009). Approximately 5000 families, consisting of both parents and at least two adolescents, were approached through municipalities in The Netherlands, to participate in the Family and Health study. A total of 885 families agreed to participate. We excluded families in which the family members were not biologically related, had physical or mental disabilities, or in which the children were twins. A further selection was made to accomplish an equal distribution of sibling dyads (girl–girl, boy–boy, boy–girl). In this manner, a total of 428 families were included at T1. Attrition was low, with 347 families (81%) participating in the four-year follow-up (T2).

At both assessment dates the participating families were visited by trained interviewers, who made sure that the questionnaires were filled out separately and individually. In return of their completion of the questionnaires, the families received a voucher of 30 Euros. In the follow-up (T2), DNA samples were collected by means of saliva. A total of 309 adolescents could be genotyped after written informed consent by the parents and the adolescents.

Attrition analyses were conducted to examine whether adolescents who were genotyped (participants) differed from the adolescents who were not genotyped (drop-outs; n = 119). t-Tests showed no significant differences (p > .05) in emotional eating, BMI, sex or age between participating and dropout adolescents. Participating subjects did have a slightly higher level of education at T1 than those who had not been genotyped (t(420) = 2.01, p = .04). For the present study, complete data were available for 279 sibling adolescents. Approval on data collection was obtained from the Central Committee on Research Involving Human Subjects in the Netherlands.

Measures

Emotional eating, external eating and restrained eating

Emotional, external and restrained eating were assessed with the Dutch Eating Behaviour Questionnaire (DEBQ) (van Strien, 2002; van Strien, Frijters, Bergers, & Defares, 1986). This questionnaire has 33 items, 13 on emotional eating (e.g., “Do you have a desire to eat when you are irritated?”), 10 on external eating (e.g., “If food smells and looks good, do you eat more than usual?”) and 10 on restrained eating (e.g., “Do you try to eat less at mealtimes than you would like to eat?”). All items have to be rated on a 5-point scale with response categories that range from 1 ‘never’ to 5 ‘very often.’ The DEBQ is easy to fill out by adolescents and has been used in ample studies involving adolescents (Lluch, Herbeth, Mejean, & Siest, 2000; Snoek et al., 2007). The DEBQ scales have high reliability, good validity for food consumption and high convergent and discriminative validity (van Strien, 2002). Also in the present study, the DEBQ-scales had high Cronbach’s alphas at T1. For the adolescents, Cronbach’s alpha at T1 were for emotional, external and restrained eating, α = .92, α = .85 and α = .92, respectively. Cronbach’s alpha at T2 was for emotional eating α = .95. For maternal and paternal emotional eating (reported by the parents themselves) Cronbach’s alpha’s were α = .95 and α = .94, respectively.

Psychological control, behavioral control and support

Perceived parenting was reported by adolescents on both parents separately at T1. Psychological control was measured with an instrument of Steinberg, Lamborn, Darling, and Mounts (1994) (Dutch translation; Beyers & Goossens, 1999). On a 5-point scale ranging from 1 ‘completely not true’ to 5 ‘completely true’, adolescents had to report on 8-items on coercive, non-democratic
discipline and psychological manipulative strategies in order to control the child’s behavior (e.g., ‘My father (mother) makes me feel guilty when I fail at school’). Cronbach’s alpha for maternal and paternal psychological control were α = .65 and α = .70, respectively. Behavioral control was assessed with an instrument developed by Kerr and Stattin (2000) (Dutch translation; Engels, Finkenauer, Meeus, & Dekovic, 2001). On a 5-point scale ranging from 1 ‘never’ to 5 ‘always’, adolescents had to answer on 5 items on control of whereabouts and activities (e.g., ‘Before you leave on a Saturday evening does your father (mother) want to know with whom and/or where you are?’). Alpha’s for maternal and paternal support were α = .71 and α = .87, respectively. Parental support was measured with the Relational Support Inventory (Scholte, van Lieshout, & van Aken, 2001). On a 5-point scale ranging from 1 ‘completely untrue’ to 5 ‘completely true’, adolescents had to report on 12 items on parental support (e.g., ‘My father (mother) supports me in the things I do’). Alpha’s for maternal and paternal support were α = .76 and α = .81, respectively.

The three instruments on parenting have been intensively employed in Dutch studies on adolescent substance use (van Zundert, van Der Vorst, Vermulst, & Engels, 2006) and eating behaviors (Snoek et al., 2007).

Adolescent personality

The Quick Big Five (Vermulst & Gerris, 2005) was used at T1 to assess personality traits. On a 7-point scale ranging from 1 ‘absolutely disagree’ to 7 ‘absolutely agree’, adolescents had to assess to what degree they possessed 30 personality traits, reflecting 5 personality dimensions (Big Five). In the present study, alphas for the different personality scales at T1 were: α = .77 for extraversion; α = .84 for conscientiousness; α = .79 for agreeableness, α = 0.75 for emotional stability and α = 0.65 for openness.

Depressive feelings

Depressive feelings were assesses with the Depressive Mood List (Kandel & Davies, 1982; Dékovic, 1996). Respondents reported the frequency of experienced negative feelings over the last 12 months, on a 7-point scale. Items were: “How often did you feel unhappy, sad or depressed”, and “How often did you worry too much about things.” In line with Kandel and Davies, the scale was transformed to a 3-point scale (1 = never/almost never, 2 = sometimes, 3 = often/always). The Depressive Mood List is used extensively in adolescent surveys (for a review, see Compas, Ey, & Grant, 1993) and had adequate reliability in the present study. Cronbach’s alphas at T1 was .77.

Adolescent BMI

Adolescent BMI (body mass index weight in kilograms/height in meters squared) was calculated based on self-reported height and weight at T1. In all analyses BMI’s rather than BMI z-scores were used as the youngest adolescents had limited age-ranges and we controlled in the analyses for sex. In a separate validity study on this sample we compared self-reported values with objectively measured data and found no evidence of higher underreporting of weight in certain weight groups (e.g., overweight adolescents) (Snoek, van Strien, Janssens, & Engels, 2008).

Parental overweight level

Parental overweight level (based on self-reported weight and height) was defined as follows: no overweight: BMI < 25 and overweight: BMI ≥ 25.

DRD2 genotyping

The DRD2 Taq1 A C > T polymorphism (rs1800497) was genotyped using Taqman analysis (assay ID: Taqman assay: C_7486766_10; reporter 1: VIC-A-allele, reverse assay; Applied Biosystems, Nieuwerkerk a/d ijssel, The Netherlands). Conformed by van der Zwaluw et al. (2009), genotyping was carried out in a volume of 10 µl containing 10 ng of genomic DNA, 5 µl of Taqman Mastermix (2 ×; Applied Biosystems), 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 5 duplicate DNA samples per 96–well plate, which were 100% consistent. In addition, 4 blanks were included in each plate, which were required to be negative. 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 V 3.3 (Raymond & Rousset, 1995). No deviations from HWE were detected (p = .96). Because a reduced D2 receptor availability in the brain is associated with the possession of at least one A1 allele (Jonsson et al., 1999), DRD2 genotype was dummy-coded into 0 (A2A2) and 1 (A1A2 and A1A1).

Statistical analyses

Descriptive analyses and Pearson correlation analyses were conducted on adolescent emotional eating (T1 and T2), DRD2 genotype, and psychological control of mother and father. To examine whether there was a moderator effect of DRD2 genotype on the relation between adolescent psychological control and the change in emotional eating over four years, we tested 5 regression models for psychological control for mother (or father) using SPSS 15.0. All variables were centered before computing interaction terms to avoid multicollinearity (Aiken & West, 1991). In all analyses we controlled for sex, level of education, BMI, personality and depressive feelings of the adolescent, parental emotional eating, and parental level of overweight (initial model). In additional analyses, we controlled for possible confounding effects of the other eating styles (external and restrained eating), and other parenting practices (support and behavioral control) and a full model in which all the confounders were included.

As recommended by Holmbeck (2002), post-hoc analyses with the initial model were run to determine the nature of this significant interaction. Prior to these analyses we computed conditional moderator variables where the DRD2 A2A2 genotype was assigned a value of 0 in one analysis and the A2A1 and A1A1 group was assigned a value of 0 in the other analysis. Each of these conditional variables was then multiplied by the (centered) maternal psychological control variable to create interaction terms. With these variables, we conducted two regression analyses, each of which included the main effect for maternal (or paternal) psychological control, one of the conditional DRD2 genotype groups (A2A2 or A2A1/A1A1) and the interaction of maternal (or paternal) psychological control and the DRD2 genotype group, thereby producing the slope for A2A2 vs. A2A1/ A1A1 genotypes.

Results

Descriptives and correlations

DRD2 genotype frequencies for the adolescents were for A2A2, A2A1 and A1A1, n = 185 (66.3%), n = 86 (30.8%) and n = 8 (2.9%), respectively. The mean and standard deviation of emotional eating at T1 was 2.26 (SD = 0.7). Those for maternal and paternal psychological control were 2.22 (SD = 0.5) and 2.17 (SD = 0.5), respectively. Table 1 shows the correlations between emotional
eating at T1 and T2, maternal and paternal psychological control and DRD2 genotype. Most notably were the low but significant correlations between emotional eating at T1 and maternal and paternal psychological control (\( r < .19, p < .01 \)) and that DRD2 genotype was neither significantly related to emotional eating (\( -.01 < r < .02, p > .05 \)) nor to maternal or paternal psychological control (\( -.01 < r < .02, p > .05 \)).

Regression analyses

Maternal psychological control

In the initial model the associations between parental psychological control and DRD2 genotype on one hand and emotional eating on the other hand, were controlled for sex, educational level, BMI and personality and depressive feelings of adolescent at T1 as well as parental emotional eating and overweight level.

The three-way interaction of sex, DRD2 genotype and maternal psychological control on emotional eating at T2 was not significant (\( B = .11, p = .74 \)), but there was a significant two-way interaction of DRD2 genotype and maternal psychological control (\( B = .378, p < .05 \)). This two-way interaction remained significant after the non-significant three-way and all non-significant two-way interaction terms were dropped and a reduced model was run (\( B = .35, p < .05 \)) (see Table 2). The DRD2 genotype–maternal psychological control interaction on emotional eating at T2 remained significant after controlling for the other adolescent eating styles (external and restrained eating), parental behavioral control, parental support, as well as in the full model which contained all possible confounders (see Table 2).

Results of the regression for A2A2 DRD2 genotype indicated that degree of maternal psychological control was not significantly associated with increase in emotional eating, (\( B = -.11, p = .30 \)). In contrast, results of the regression for A2A1/A1A1 DRD2 genotypes indicated that maternal psychological control was borderline significantly related with increase in emotional eating at T2 (\( B = .24; p = .06 \)). Regression lines depicting levels of maternal psychological control for the A2A2 and A2A1/A1A1 DRD2 genotypes are plotted in Fig. 1.

Paternal psychological control

Highly similar results were obtained with paternal psychological control. The three-way interaction of sex, DRD2 genotype and paternal psychological control on emotional eating at T2 in the initial model was not significant (\( B = -.03, p = .93 \)), but there was a significant two-way interaction of DRD2 genotype and paternal psychological control (\( B = .35, p < .05 \)). This two-way interaction remained significant after the non-significant three-way and all non-significant two-way interaction terms were dropped and a reduced model was run (\( B = .35, p < .05 \)) (see Table 3). The DRD2 genotype–paternal psychological control interaction on increase in emotional eating at T2 remained significant when we also controlled for the other eating styles (external and restrained eating), parental behavioral control, parental support, as well as in the full model which contained all possible confounders (Table 2).

In a similar manner as above, post-hoc analyses were conducted to determine the nature of these significant interactions. As with maternal psychological control the slopes for the A2A2 and A2A1/A1A1 genotypes ran in different directions. Results of the regression for A2A2 DRD2 genotype indicated that degree of paternal psychological control was not significantly associated with increase in emotional eating, (\( B = -.13, p = .17 \)). In contrast, results of the regression for A2A1/A1A1 DRD2 genotypes indicated

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**Table 1**

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**Table 2**

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Note: M = mother, F = father, Beh. control = behavioral control.

1. DRD2 genotype: 0 = A2A2; 1 = A1A2/A2A1.
2. Interaction between DRD2 genotype and maternal psychological control, see also Fig. 1.

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Fig. 1. Two-way interaction between DRD2 genotype (A1A2/A1A1 vs A2A2) and maternal psychological control on emotional eating at T2. The group A1A/A1A1 is the group at risk because the possession of at least one A1 allele is associated with reduced D2 receptor availability in the brain.
that paternal psychological control was borderline significantly related with increase in emotional eating at T2 ($B = .21; p = .08$). Regression lines depicting levels of paternal psychological control for the A2A2 and A2A1/A1A1 DRD2 genotypes are plotted in Fig. 2.

Discussion

The current study examined in a longitudinal full-family design, the possible interaction effects of sex, paternal psychological control and a common polymorphism involving reduced dopamine D2 receptor availability in the brain (the A1 allele of the DRD2 gene Taq1A polymorphism) on emergence of emotional eating in a sample of mid-adolescents. As we expected, adolescents who reported paternal psychological control showed more increase in emotional eating if they carried at least one DRD2 A1 allele. This interaction effect was found for both maternal and parental psychological control. We found no support for our expectation that this interaction effect would be moderated by sex.

We did not find direct effects of the DRD2 A1 allele or of parental psychological control on increase in emotional eating. Instead, in line with other studies that examined interactions between genetic polymorphisms and adverse environments (Barr et al., 2004; Caspi et al., 2003; Krueger et al., 2008; van der Zwaluw et al., 2009), we found support for a gene–environment (GxE) interaction. Only in combination with the risk genotype (the DRD2 A1 allele) the adverse environment (high parental psychological control) was found to be associated with increases in emotional eating.

This prospective study with a four-year follow-up also permits tentative conclusions regarding the direction of causality between reward sensitivity and emotional eating. The present results are more in line with the view that a primary dopaminergic functioning underlies the development of emotional eating (see also Volkow et al., 2003). By controlling for initial emotional, external and restrained eating, and initial BMI in our prospective analyses we could reduce the risk that a history of overeating would explain any prospective effects. Even so, we found an increase in emotional eating in relation to adverse parenting, but only in adolescents carrying at least one DRD2 A1 allele, which is known to result in reduced dopamine D2 receptor availability in the brain (see also Stice, Spoor, Bohon, & Small, 2008).

It should be noted that the interaction effect of DRD2 and parental psychological control on emotional eating at T2 remained significant when also parental behavioral support and parental behavioral control were entered in the analyses. On close inspection, parental psychological control was in the present study negatively associated with parental support ($- .33 < r < - .43$, $p < .001$) and only lowly associated with parental behavioral control ($- .10 < r < - .01$). So it seems, in line with cumulative risk theory, that high parental psychological control was in most cases not buffered by potential protective effects of parental support and parental control (Finkenauner, Engels, & Baumeister, 2005). Risk and protective factors in the family environment are acknowledged to play an important role in emotion regulation (Morris, Silk, Steinberg, Myers, & Robinson, 2007), in turn an important factor in development of emotional eating (Ouwens, van Strien, van Leeuwe, & van der Staak, 2009; van Strien & Ouwens, 2007). For future studies it would be of interest to also assess emotion regulation abilities (see McEwen & Flouri, 2009; Spoor, Bekker, van Strien, & van Heck, 2007) in addition to other adverse aspects of the family environment (sexual and physical abuse) in relation to genotypes (Caspi et al., 2003; Cicchetti & Rogosch, 2001).

Our study is the first to show a gene × adverse parenting interaction on emergence of emotional eating in adolescents, using a longitudinal sample with high retention rates. It goes without saying that the present findings need replication on other large, longitudinal population-based samples.

Conclusion

Our findings further clarified the emergence of emotional eating in adolescence. DRD2 genotype moderated the relation between adverse parenting and increase in emotional eating, in that adolescents showed only an increase in emotional eating if they carried at least one DRD2 A1 allele. This moderator effect of

Table 3

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Note: M = mother, F = father, Beh. control = behavioral control.

1 DRD2 genotype: 0 = A2A2; 1 = A1A2/A1A1.
2 Interaction between DRD2 genotype and Paternal psychological control, see also Fig. 2.

*p < .05.
**p < .01.
***p < .001.
1 p < .10.
**References**


