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Emotional eating in adolescents: A gene (*SLC6A4/5-HTT*) – Depressive feelings interaction analysis

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ABSTRACT

Eating in response to distress – i.e. emotional eating – is highly prevalent in (female) adults with binge eating, but has only a very low prevalence in young children. The present study addresses the emergence of emotional eating in adolescence in relation to depressive feelings. Because a reduction of food intake is considered the biologically natural response to distress, we tested whether the a-typical stress-response of emotional eating develops in interaction with genetic vulnerability. We hypothesized that the short allele of the 5-HTTLPR polymorphism in the serotonin transporter gene, which is associated with lower serotonin activity, would moderate the relation between depressive feelings and the increase in emotional eating, particularly in females. A sample of Dutch families with two adolescents was included in a longitudinal study with a four-year follow-up. A moderator effect of 5-HTTLPR genotype on the relation between depressive feelings and the increase in emotional eating was found in both sexes in the youngest siblings ($n = 286$). In the older siblings ($n = 298$), this specific moderator effect was only found in the girls. Younger adolescents and older adolescent girls showed a higher increase in emotional eating if they carried the 5-HTTLPR short allele. This is the first study that found support for a gene × depressive feelings interaction on emergence of emotional eating in (female) adolescents

1. Introduction

Emotional eating, that is eating in response to negative emotions such as tension, irritability and depression, is closely associated with binge eating in adult (clinical) samples (Racine et al., 2009; Van Strien et al., 2005). Though emotional eating is highly prevalent in adults, particularly in (female) adults who are overweight or obese (Van Strien et al., 2009, 2010), it has a very low prevalence in young children (Carper et al., 2000; Van Strien and Oosterveld, 2008; Wardle et al., 2001). This would suggest that emotional eating emerges in the transition between childhood and adulthood: the period of adolescence. In the present study we are interested in the emergence of emotional eating in adolescence in relation to depressive feelings.

Adolescence is notorious for its association with depressive feelings (Patton and Viner, 2007), particularly so in females (Cyranowski et al., 2000; Wade et al., 2002). However, eating in response to depressive feelings, so called emotional eating, is an a-typical response (Gold and Chrousos, 2002; Krauchi et al., 1997; Lam and Levitan, 2000; Ouwens et al., 2009). Major depression (melancholia), which shares many features with the stress response (Gold and Chrousos, 2002), is normally associated with a hyperactive hypothalamic–pituitary–adrenal axis (HPA axis) with physiological reactions that are designed to prepare the individual for a fight or flight reaction. These reactions inhibit gastric motility and promote the release of sugar into the blood-stream, thereby suppressing feelings of hunger (Cannon, 1915; Schachter et al., 1968). Because a reduction of food intake during distress is considered to be the evolutionally adaptive and biologically natural response, it has been postulated that the unnatural response of emotional eating is acquired, perhaps as result of adverse rearing experiences early in life (Bruch, 1973).

There is indeed evidence, starting with Spitz' seminal observations in small infants in understaffed orphanages (Emde et al., 1965) followed up by (controlled) studies on animals and humans (Barr et al., 2004; Cicchetti and Rogisch, 2001; Papaioannou et al., 2002; Yehuda et al., 2000) that adverse rearing experiences early in life may have lasting effects on stress-responsive neurobiological systems, particularly when they pertain a perturbed mother–infant relationship. This may include a hypoactivation, rather than hyperactivation of the HPA axis, with neurovegetative symptoms

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that are the reverse of those associated with typical melancholic depression (APA, 1994; Krauchi et al., 1997): increased food intake (hyperphagia) and weight gain, instead of hypophagia and weight loss (APA, 1994; Gold and Chrousos, 2002). However, not everyone who experiences adversity early in life develops the a-typical response to later distress (Cicchetti and Rogisch, 2001), suggesting the possible role of genetics. There is accumulating evidence that the a-typical stress response develops in interaction with genetic vulnerability (Caspi et al., 2003; Gold and Chrousos, 2002).

Also, Seasonal Affective Disorder (SAD) is associated with a-typical stress responses such as emotional eating (APA, 1994; Krauchi et al., 1997). There is some evidence that the serotonin transporter gene (*SLC6A4/5-HTT)* may be involved in the pathogenesis of a-typical depressions such as SAD (Rosenthal et al., 1998; Willeit et al., 2003). A polymorphism in the promoter region of *5-HTT* (5-HTTLPR) causes differential 5-HTT expression, with the short (S) allele resulting in a loss of HTT activity compared to the long (L) allele (Hariri and Holmes, 2005). Low levels of serotonin activity have been associated with a more reactive arousal system, including increased appetite and body weight (Bortolino et al., 2005; Leibovitz and Alexander, 1998; Sookoian et al., 2007, 2008), though relations with binge eating have been less consistent (Racine et al., 2009).

The S allele has also been linked to SAD (Rosenthal et al., 1998; Willeit et al., 2003). Because of the possible role of serotonin in the regulation of food intake and because food intake shows opposite directions in melancholia and a-typical depression, Willeit and colleagues (2003) analyzed data for differences in 5-HTTLPR genotype distribution between melancholia and a-typical depression. Carriers of the S allele were found to be significantly more likely to suffer from a-typical depressions such as SAD.

In the present study, we examine the moderation effect of 5-HTTLPR genotype on the relation between depressive feelings and the emergence in adolescence of emotional eating – emotional eating was, in an earlier study, indeed found to be associated with a-typical depression (SAD, winter-type) (Krauchi et al., 1997). Since depressive feelings, SAD and emotional eating all have a female preponderance (Cyranowski et al., 2000; Jang et al., 1997; Van Strien, 2002; Wade et al., 2002), we were also interested in a possible moderator effect of gender.

Using a longitudinal four-year follow-up design we examined the change in emotional eating over four years by controlling for baseline levels of emotional eating. We hypothesized that 5-HTTLPR genotype would act as moderator in that the relationship between depressive feelings and the increase in emotional eating would become stronger if adolescents carried the 5-HTTLPR short allele. We also hypothesized that the moderator effect of 5-HTTLPR genotype on the relation between depressive feelings and the increase in emotional eating would be stronger in girls than in boys. In initial analyses we only controlled for initial emotional eating, sex, level of education and BMI of the adolescents. In several additional analyses we investigated the robustness of the obtained results. First, we controlled for the 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 lose body weight. Secondly, we controlled for the possible confounding effect of adolescent personality, because emotional eating is related to personality (Heaven et al., 2001), which is in turn related to genetics (Krueger et al., 2008). Thirdly, we controlled for parental emotional eating and finally for parental overweight, because in previous research, adolescents' emotional eating and parental overweight were found to be related (Snoek et al., 2007a; Wardle et al., 2008).

2. Methods

2.1. Participants and procedure

Participants were 428 Dutch 2-child families with an average age of 13.4 years (SD = .6) for the youngest adolescent and an average age of 15.2 (SD = .5) for the oldest adolescent at baseline measurement (T1). The adolescents participated 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 et al., 2005). Via municipalities in The Netherlands, approximately 5000 families, consisting of both parents and two adolescents, were approached to participate in the Family and Health study. A total of 885 families agreed to participate. Those families in which the family members were not biologically related, had physical or mental disabilities, or in which the children were twins, were excluded. In addition, to accomplish an equal distribution of sibling dyads (girl–girl, girl–boy, boy–boy, boy–girl) a further selection was made. As such, a total of 428 families were included at T1. Attrition was low, with 347 families (81%) participating in the four-year follow-up (T2).

Families participated by filling in extensive questionnaires. At both assessment dates they were visited by trained interviewers, who made sure that the questionnaires were filled out separately and individually. When all family members had completed the questionnaires, the family received a voucher of 30 Euros. In the follow-up (T2) DNA samples were collected by means of saliva. A total of 612 adolescents could be genotyped after the parents and the adolescents had given written informed consent to be genotyped. Attrition analyses were conducted to examine whether adolescents who were genotyped (participants) differed from the adolescents who where not genotyped (drop-outs; $n = 344$). *T*-tests showed no significant differences ($p > .05$) in emotional eating, depressive feelings, BMI or gender between participating and drop-out younger or older adolescents. Older adolescents did have a higher level of education at T1 than those not included in the study ($t(425) = -2{,}26$, $p = .02$), but in the younger sibling this difference was borderline significant ($t(420) = -1.86$, $p = .06$). For the present study, complete data were available for 286 younger and 298 older sibling adolescents.

Since the two siblings are part of the same family, their data are dependent, or nested. Statistically it is possible to examine nested data with multilevel (regression) analysis. However, the adolescents differ 2 years in age, which puts them into quite different phases of adolescence. As such, from a theoretical point of view it is objectionable to combine both groups. Therefore, we performed separate analyses for the younger and older siblings, instead of collapsing them into one group.

Approval on data collection was obtained from the Central Committee on Research Involving Human Subjects in the Netherlands.

2.2. Measures

2.2.1. Emotional eating, external eating and restrained eating

Emotional, external and restrained eating were assessed with the Dutch Eating Behavior Questionnaire (DEBQ) (Van Strien, 2002; Van Strien et al., 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