Genetic and attachment influences on adolescents’ regulation of autonomy and aggressiveness

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Background: Adolescence is a time when intense emotions are elicited within the parent–adolescent relationship, often when autonomy subjectively is endangered. As emotion dysregulation is one of the risk processes for the development of psychopathology, adolescence may be perceived as a highly sensitive period for maladjustment. Inter-individual differences in emotionality and emotion regulation have been shown to be influenced or moderated by molecular genetic differences in the serotonin transporter gene (5-HTT) and by attachment patterns. We investigated whether both the 5-HTT and attachment are associated with emotionality and emotion regulation in an observed adolescent–mother interaction and the personality traits aggressiveness and anxiety in adolescence. Methods: Ninety-one adolescents at age 12 were observed in interaction with their mothers during a standardized emotion-eliciting social task to assess emotionality and emotion regulation in relation to autonomy. Adolescents’ aggressiveness and anxiety were assessed by mother report. Concurrent attachment quality was determined by an attachment interview. DNA samples were collected in order to assess the 5-HTTLPR, a repeat polymorphism in the promoter region of the serotonin transporter gene. Results: While the short allele of the serotonin transporter gene was associated with a higher overall rate of autonomy behaviors, attachment security was related to more agreeable and less hostile autonomy. A significant interaction revealed a moderating effect of attachment security. Carriers of the short version of the 5-HTTLPR showed more agreeable autonomy when they had a secure attachment behavior strategy but showed more hostile autonomy when they were insecurely attached. Carriers of the short version of the 5-HTTLPR and insecurely attached adolescents were rated as more aggressive. Conclusions: The study suggests a gene–attachment interaction in adolescents where the adolescent’s attachment status moderates a genetically based higher negative reactivity in response to threats to autonomy in social interactions. Keywords: Serotonin-transporter, emotion regulation, attachment, autonomy, adolescence.

Emotion regulation and emotion dysregulation have repeatedly been shown to be related to psycho-pathological symptoms or deviant pathways in childhood and adolescence and can be interpreted as a mediator between experiences within the family context and psychopathology (Cole, Michel, & Teti, 1994; Denham, 1998; Maughan & Cicchetti, 2002; Morris, Silk, Steinberg, Myers, & Robinson, 2007). Emotion regulation encompasses changes in the quality, intensity, duration, and latency of the emotional reaction and expression in the service of adaptation (Thompson, 1994). In contrast, emotion dysregulation describes the inability to change the intensity of emotional experiences or reactions, often disrupting social relations (Cole et al., 1994). Adolescence is a developmental period of intense emotionality which is based on major changes in hormonal, physical, social, and cognitive domains (Larson, Moneta, Richards, & Wilson, 2002; Silk, Steinberg, & Morris, 2003) and elicited by stage-salient issues, specifically autonomy development (Allen, Hauser, Bell, and O’Connor, 1994). Despite an improvement in emotion regulation capacities during adolescence, expressed in a general increase of active coping strategies and secondary control strategies (Seiffge-Krenke, 1995), the developmental changes constantly confront adolescents with new challenges to their emotion regulation abilities. For example, social and academic evaluations become more prominent as elicitors of fears in adolescence as compared to childhood (Westenberg, Drewes, Goedhart, Siebelink, & Trefers, 2004).

Many intense emotions are elicited and regulated within the parent–adolescent relationship (Collins & Laursen, 2006). While the number of conflicts within the family increases, there is little change in individual dyadic time spent with each parent (Larson, Richards, Moneta, Holmbeck, & Duckett, 1996). The frequency of parent–adolescent conflict is highest in early adolescence, and conflicts seem to be most intense in middle adolescence (Laursen, Coy, & Collins, 1998). Often, such disputes are elicited by challenges to the adolescent’s autonomy (Allen & Hauser, 1996). This indicates that emotion regulation in the context of autonomy is an important topic already at the beginning of adolescence. Despite this normative trend in the frequency and intensity of parent–adolescent conflicts, there are major...
inter-individual differences in the way these intense emotions are regulated, how such disputes are resolved, and in what developmental consequences these differences in the regulation of conflicts and autonomy within the family result (Allen, Hauser, Bell, & O’Connor, 1994).

Emotion dysregulation in parent–adolescent conflicts has been linked to externalizing and internalizing behaviors in adolescents. While internalizing behaviors were more closely connected to a lack of autonomy, externalizing behaviors were more closely related to autonomy without relatedness (Allen, Porter, & McFarland, 2007), as expressed in aversive behaviors of both parent and adolescent (Dadds, Sanders, Morrison, & Rebegetz, 1992). Moreover, maintaining relatedness when exhibiting autonomous behavior was positively associated with self-esteem and ego development (Allen et al., 1994). These findings suggest that conflictual adolescent–parent interactions have a positive effect on adjustment, as long as they occur in the broader context of a supportive relationship. When autonomy was displayed (by the adolescent) or challenged (by the parent) in an agreeable, non-hostile way, increases in self-esteem and ego development were higher over a two-year range in mid-adolescence (Allen et al., 1994). Based on these findings, we conclude that, especially for adolescents, autonomy-related emotion regulation is important for concurrent and later developmental adjustment. Differences in emotion regulation in general and specifically in parent–adolescent relationships might be explained by genetic and environmental factors and their interaction (Thompson, 1994; Morris et al., 2007; Moffitt, Caspi, & Rutter, 2006).

**Genetic effects on emotionality and emotion regulation**

The study of dispositional or molecular-genetic effects on emotionality and emotion regulation revealed some candidate genes, one of which is the serotonin transporter 5-HTT (Canli & Lesch, 2007). Serotonin (5-HT) can be seen as a key modulator of emotional reactivity and behavior. The 5-HTT polymorphism leads to differences in serotonin 5-HT neurotransmission, with the short (S) allele variant displaying significantly less 5-HTT binding in the brain than the homozygous long (L) variant (Murphy & Lesch, 2008). As a result of the relative loss of 5-HTT gene function in S allele carriers, impaired functional integration of cortico-limbic connectivity and compromised inhibitory regulation via the prefrontal cortex lead to hyper reactivity of the amygdala to emotionally provocative stimuli (Hariri & Holmes, 2006). S allele carriers show a higher emotional reactivity (i.e., a lower threshold to emotional stimuli) as well as a higher baseline activity of the amygdala (Canli & Lesch, 2007). Beside associations of the short allelic variant of the 5-HTTLPR genotype with high neuroticism, anxiety, and low agreeable-ness (Canli & Lesch, 2007), significant associations with aggressiveness also have been reported (Henning, Reuter, Netter, Burk, & Landt, 2005). After reviewing the literature, Carver and Miller (2006) came to suggest that the short allele variant might be more closely related to impulsivity and aggressiveness, compared to anxiety.

In summary, S allele carriers feature an ‘emotional phenotype’ characterized by heightened emotional reactivity and an impaired capacity to regulate emotions, imposing an increased risk of developing mental disorders. Yet, this genetic disposition may only have an increased maladaptive outcome if individuals were exposed to life stress or adverse child environments (Caspi et al., 2003; Reif et al., 2007), thus providing a model explaining individual vulnerability in the face of adversities for S allele carriers but also the moderating effect of social support.

**Family effects on emotionality and emotion regulation**

Family influences on the development of emotionality and emotion regulation can be seen in parenting practices, emotional family climate, and different emotional learning experiences (Morris et al., 2007). Klimès-Dougan et al. (2007) found that non-supportive parental emotion socialization practices were associated with adolescents’ emotional and behavioral problems. Parents of adolescents with externalizing problems were found to be more dismissive towards their offspring’s emotional displays, thus predisposing their children for acting out problems.

Attachment can be seen as specifically influential for the effectiveness of emotion regulation (Cassidy, 1994; Mikulincer & Shaver, 2005), because attachment patterns already in infancy represent specific ways of interactive emotion regulation with the caregiver (Zimmermann, 1999). In infancy, attachment differences are assessed in emotion-inducing situations (i.e., the Strange Situation). While the secure attachment pattern is characterized by effective social emotion regulation, the insecure patterns are inefficient in their regulatory approach, whether social (ambivalent attachment) or individual (avoidant attachment). Several studies have shown that secure attachment in infancy predicts lower rates of aggressiveness and more social competence in later development (Sroufe, Egeland, & Carlson, 1999; Suess, Grossmann, & Sroufe, 1992). In adolescence, secure attachment is associated with effective social emotion regulation and a balance of autonomy and relatedness in interaction with parents and peers during conflict discussions as well as in task contexts (Kobak, Cole, Ferenz-Gillies, Fleming, & Gamble, 1993; Allen & Hauser, 1996; Zimmermann, Maier, Winter, & Grossmann, 2001; Becker-Stoll, Fremmer-Bombik, Wartner, Zimmermann, & Grossmann, 2008).
Moderating the genetic risk

The investigation of gene–environment interaction is a promising approach to explain developmental processes in psychopathology (Moffitt et al., 2006), because gene–environment interaction may explain the high variability in findings of studies only focusing on genetic or environmental main effect models. Reviewing environmental effects, Rothbaum and Weisz (1994) reported small but significant associations between parenting and externalizing or internalizing behavior. Child effects on parent–child conflicts and psychopathology have been shown as well (O’Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998). A twin study by Burt, McGue, Krueger, and Iacono (2005) showed that, although the stability of conflicts and externalizing behavior in adolescence seems to be genetically based, a transactional model of reciprocal effects of parent–child conflicts and dispositions best explained the development of externalizing behavior. Eisenberg and Morris (2002) suggested that children with high emotionality in particular need effective emotion regulation capacities in order to prevent the development of externalizing behavior. Parental sensitivity, one determinant of attachment security, may be an environmental context promoting the development of such effective emotion regulation. Bakermans-Kranenburg and van IJzendoorn (2006) demonstrated that sensitive parenting can moderate the genetic risk for externalizing behavior of the DRD4 polymorphism. In addition, Spangler, Johann, Ronai, and Zimmermann (2009) found that carriers of the short 5-HTTLPR allele had a heighten-ed risk of developing a disorganized attachment pattern. This effect, however, was moderated by maternal responsiveness which seems to function as a social buffer against this genetic risk. Thus, transactional developmental models and effects of gene–environment interactions on emotion regulation may help to explain developmental processes in psychopathology.

The main objective of the present study was to investigate the interplay between attachment quality and allelic variations of the serotonin transporter gene on emotionality and emotion regulation a) in an age-appropriate emotion-eliciting task concerning adolescents’ autonomy and b) in the assessment of aggressiveness and anxiety as personality traits that are characterized by emotion dysregulation. We expected that carriers of the short allele variant of the 5-HTTLPR would show a higher emotional reactivity and impaired emotion regulation when confronted with an emotion-eliciting task and should show increased levels of aggressiveness and anxiety. In addition, we expected an effect of adolescents’ attachment security on adaptive emotion regulation, specifically a moderating effect on the genetic risk for emotion regulation but not for emotionality, and on lower levels of aggressiveness and anxiety.

Method

Participants

The current study was conducted as part of the 12-year longitudinal follow-up assessment of the Regensburg Longitudinal Study IV, a sample of originally 106 healthy German, Caucasian, low-risk infants (53 girls/53 boys), first assessed at age 1. At the 12-year assessment, a total of 96 participants and their mothers could be seen again. The complete data set for this report, however, was only available for 91 adolescents (45 males, 46 females). The families represent a fairly wide range of socioeconomic status, including 42% upper middle class, 24% middle class, and 27% lower class, as assessed by the father’s education, occupation, and family income.

Procedure

Adolescents and their mothers were invited to the university. After obtaining informed consent from the parents, cheek-cells for gene analyses were collected. Mothers and adolescents then were interviewed separately about their attachment experiences. Afterwards, they participated in a standardized interaction task, the dyadic ‘talk show’ situation, designed to induce negative emotions by creating a social evaluative context, which is a major elicitor of fear in adolescence. Participants were informed that the mother had to interview the adolescent in front of a public audience. In preparation for this talk show, mother and adolescent had 10 minutes in which to cooperatively write down six personal topics which would characterize the adolescent best during the following public presentation. Both mothers and adolescents could make suggestions and accept or contradict them. Afterwards, the adolescent was interviewed by the mother in front of a small audience and a video camera. The notion of the adolescent being under social evaluation was emphasized in the instructions to ensure the emotional impact of the situation. The 10 minutes of preparation were videotaped and coded for adolescent autonomy behaviors as indicators of emotional reactivity and emotion regulation, and for emotion expression. All analyses were carried out by independent and reliable coders using a standardized observational system.

Measures

Emotion expression. Adolescents’ negative emotions were coded by means of a second-by-second event-based coding system from their facial expressions of fear, uneasiness, discomfort, anger, and sadness. Inter-rater reliability was good (kappa .75). All emotional expressions were summed up to a total frequency score of negative emotional expression.

Adolescents’ observed autonomy. Autonomy in this study is understood as self-regulation by explicitly defining one’s own goals and trying to reach them. In the interaction task, all events were coded as autonomy behavior in which the adolescent disagreed with his or her mother’s suggestions or decisions of what to ask during the subsequent public presentation in the talk
show. More specifically, autonomy behaviors included explicitly dismissing the mother’s question, arguing against it, or nonverbally but unequivocally expressing dissent. Autonomy behavior also was coded when the adolescent insisted on his or her own suggestions or negotiated differing viewpoints.

The adolescents’ reactions were defined as agreeable autonomy when the disagreements were solved and followed by further positive communication with the mother while trying to argue for their point of view rather than trying to force their will upon her. Disagreements with the mother were coded as hostile autonomy when they were followed or accompanied by verbal or physical attacks of the mother or by an explicit refusal to cooperate for at least 3 seconds. Hostile autonomy was also coded when the adolescent responded in an emotionally charged manner towards the mother as a reaction to an earlier disagreement with her. Examples of such attacks are active or reactive verbal threat, threatening gestures, physical attacks, or ridiculing utterances. Elicitors could be the mother’s tone of voice, her gestures, or actions. Inter-rater reliability (kappa) was .72 and .84 for agreeable autonomy and hostile autonomy, respectively.

Mothers’ observed intrusiveness. Maternal intrusive behavior was defined as inhibiting or undermining the adolescent’s autonomy and is behavior incongruent with the adolescent’s intentions or mood. Typical intrusive behaviors include insisting on questions not wanted from the adolescent, not accepting the adolescent’s suggestions, interfering with the adolescent’s task activities, or physical aggression. Maternal intrusiveness was rated on a 9-point behavioral rating scale ranging from non-intrusive (1) to highly intrusive (9) by different raters and independently of the adolescent coding. The inter-rater agreement resulted in a kappa of .92.

Attachment. Attachment was assessed by the Late Childhood Attachment Interview (LCAI; Zimmermann & Scheuerer-Englisch, 2000), a semi-structured interview that probes the individual’s descriptions of the current relationship to both parents in attachment-relevant situations and the attachment behavior towards the parents. The interviews were rated from videotapes in regard to attachment representations for both parents and to attachment behavior strategy on 5-point scales. Coherence was coded with a categorical event-based system. The attachment behavior strategy scale from this system was utilized in the current study. Higher scores on this scale reflect seeking caregiver proximity, support, or comfort when experiencing negative emotions that the adolescent cannot regulate without help. Low scores reflect avoidance of the caregivers, retreat, or pretending that no help is needed. The interviews were rated by an independent coder not informed about other data of this study. Reliability (kappa), established on 20 interviews from a different sample, was .93.

The validity of the LCAI was demonstrated in previous studies that showed significant associations of the attachment behavior strategy with the Strange Situation attachment patterns in infancy, parent–child interaction in toddlerhood, concurrent parenting, and later attachment representations assessed with the Adult Attachment Interview (Zimmermann et al., 2000; Grossmann et al., 2002; Grossmann, Grossmann, Winter, & Zimmermann, 2002).

Adolescent’s aggressiveness and anxiety. Aggressiveness and anxiety were assessed using the California Child-Q-sort (CCQ; Block & Block, 1980), which consists of 100 items with different personality characteristics. Mothers were asked to describe their adolescent children by means of the CCQ. The individual Q-sorts were correlated with prototypical Q-sorts describing aggressiveness and anxiety at that age (provided by experienced developmental psychologists and child psychiatrists). The correlations between each child’s Q-sort and the prototypical Q-sorts were used as raw scores for further statistical analysis (Block & Block, 1980). In a pilot study with children from a child-psychiatry clinic, maternal Q-sort ratings were significantly associated with the Child Behavior Checklist (CBCL) scores provided by clinicians. For easier understanding of the results, the scores were transformed linearly by an addition of 1, resulting in a possible range from 0 to 2.

Molecular-genetic analyses

Genotyping for the 5-HTTLPR polymorphism was performed at the Institute of Medical Chemistry, Molecular Biology and Pathobiocchemistry, Semmelweis University (Budapest, Hungary), by scientists blind to the psychological data. Genomic DNA was isolated from buccal swabs using published procedures (Freeman et al., 1997).

The 5-HTTLPR variable number of tandem repeats (VNTR) polymorphism was investigated by employing two flanking primers for the polymerase chain reaction (sense primer: 5’ GGC GTT GCC GCT CTG AAT GC 3’, antisense primer: 5’ GAG GGA CTG AGC TGG ACA ACC AC 3’); thermocycling was initiated at 95°C for 10 min to activate HotStar DNA polymerase (Qiagen) followed by 35 cycles of 1 min denaturation at 95°C, 1 min of annealing at 65°C and 1 min extension at 72°C. In the VNTR polymorphism 50% of dGTP were replaced with dITP avoiding allelic drop-out in heterozygotes. The length of the generated PCR-amplions directly shows the repeat number (Ronai et al., 2001).

Statistical analyses

Two-factorial genotype × attachment analyses of variance were applied to test the hypotheses of this study. For use as independent factors, the 5-HTTLPR polymorphism and the attachment behavior strategy were dichotomized. Regarding the 5-HTTLPR, subjects were grouped into carriers and non-carriers of a short allele (ss, sl vs. ll). Regarding attachment, the subjects were grouped according to their scores on the attachment strategy scale into subjects with insecure (score ≤ 3; avoidant or ambivalent attachment behaviors) vs. secure (score > 3; seeking proximity) attachment strategies. To control for the influence of maternal behavior during the interaction situation, maternal intrusiveness was used as a covariate in the ANOVAs.

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Results

Preliminary analyses

The frequencies of short and long alleles of 5-HTTLPR were 41.7% and 58.3%, respectively, which are comparable to European populations (Gelernter, Kranzler, & Cubells, 1997). The genotype frequencies were 35 (37%), 42 (44%), and 19 (20%) for the ll, ls, and ss genotypes, respectively. The 5-HTTLPR genotype distribution was in the Hardy–Weinberg equilibrium ($\chi^2 (2, N = 96) = .96, N.S.$).

Neither maternal intrusiveness ($t(90) = -1.6, N.S.$) nor adolescents’ attachment behavior strategies ($t(90) = .46, N.S.$) significantly differed regarding the adolescents’ 5-HTTLPR genotype status (ll vs. ss/sl). This indicates that neither was influenced by the serotonin transporter gene polymorphism. In addition, the adolescents’ gender was independent from the 5-HTTLPR genotype status ($\chi^2 (1, N = 96) = .63, N.S.$).

Girls showed a significantly higher number ($t(90) = 2.3, p = .02$) of agreeable autonomy behaviors ($M = 3.8$) than boys ($M = 2.1$) and significantly higher scores ($t(90) = 2.0, p = .046$) for attachment behavior ($M = 3.4$) than boys ($M = 2.8$). However, there were no significant gender differences for hostile autonomy, negative emotional expression during the task, aggressiveness, anxiety, or maternal intrusiveness. The numbers of both agreeable and hostile autonomy behaviors were not significantly associated with aggressiveness or anxiety. Maternal intrusiveness was significantly associated with adolescents’ hostile autonomy ($r(91) = .40, p < .01$), however not with agreeable autonomy, aggressiveness, or anxiety. In further statistical analyses of the talk show behavior, maternal intrusiveness and sex were included as covariates.

The talk show interaction task successfully elicited negative emotions in the adolescents, as presented in Table 1.

Gene–attachment interactions

First, we tested a possible genotype and attachment quality influence on emotional reactivity during the emotion-eliciting interaction task. A 5-HTTLPR (ll vs. sl/ss) x attachment strategy (insecure vs. secure) ANOVA with negative emotional expression as dependent variable and maternal intrusiveness as a covariate did not result in any significant effects. Thus, emotionality, indexed by the number of negative emotional expressions, seems not to be associated with the quality of the attachment strategy or the variations in the serotonin transporter gene.

Next, the hypothesis of a possible genotype x attachment interaction in explaining the adolescents’ autonomy behavior was examined. A 5-HTTLPR x attachment MANOVA with agreeable autonomy and hostile autonomy as dependent variables and maternal intrusiveness and sex as covariates revealed significant main effects for the 5-HTT polymorphism ($F(2, 84) = 3.9, p = .025$) and for attachment quality ($F(2, 84) = 4.4, p = .015$), and a significant multivariate interaction between attachment quality and the 5-HTTLPR ($F(2, 84) = 5.9, p = .004$). Post hoc univariate analyses showed a significant main effect for attachment quality ($F(1, 85) = 5.1, p = .026$) and a significant attachment x genotype interaction ($F(1, 85) = 7.7, p = .007$) for agreeable autonomy, and significant main effects for attachment quality ($F(1, 85) = 4.6, p = .035$) and the 5-HTTLPR polymorphism ($F(1, 85) = 4.2, p = .04$), and a significant attachment x genotype interaction ($F(1, 85) = 5.1, p = .026$) for hostile autonomy. Post hoc t-tests revealed that carriers of the short variant of the 5-HTTLPR with a secure attachment quality at age 12 showed significantly more agreeable autonomy behavior ($t(25.9) = -3.1, p = .005$) and significantly less hostile autonomy behavior ($t(43.8) = 3.9, p < .0001$) in the talk show than carriers of the short variant of the 5-HTTLPR with an insecure attachment quality, as presented in Table 1. We additionally conducted non-parametric analyses to control for possible effects of skewed distributions apparent in the high standard deviation of autonomy behaviors in the carriers of the short variant of the 5-HTTLPR. The Mann–Whitney U-test showed that carriers of the short variant of the 5-HTTLPR with a secure attachment quality at age 12 showed a significantly higher ($U = 36.5, p = .016$) mean rank in agreeable autonomy (Mean Rank = 34.7) compared to the insecure group (Mean

Table 1 Means (and standard deviations) of adolescents’ emotionality, observed autonomy behaviors, aggressiveness, and anxiety at age 12 as a function of serotonin transporter polymorphism (HTTLPR) and attachment status

<table>
<thead>
<tr>
<th></th>
<th>Long HTTLPR</th>
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<th>Short HTTLPR</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Insecure attachment (n = 22)</td>
<td>Secure attachment (n = 13)</td>
<td>Insecure attachment (n = 33)</td>
<td>Secure attachment (n = 23)</td>
</tr>
<tr>
<td>Emotionality</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative emotions</td>
<td>11.47 (7.8)</td>
<td>12.07 (5.6)</td>
<td>12.45 (9.2)</td>
<td>10.78 (5.7)</td>
</tr>
<tr>
<td>Autonomy behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Agreeable autonomy</td>
<td>2.31 (2.2)</td>
<td>2.53 (2.3)</td>
<td>1.84 (1.9)</td>
<td>5.52 (5.4)</td>
</tr>
<tr>
<td>Hostile autonomy</td>
<td>2.04 (3.6)</td>
<td>1.76 (3.5)</td>
<td>9.24 (9.8)</td>
<td>2.13 (3.3)</td>
</tr>
<tr>
<td>Personality</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aggressiveness</td>
<td>.76 (.22)</td>
<td>.60 (.15)</td>
<td>.82 (.24)</td>
<td>.74 (.22)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>.77 (.17)</td>
<td>.73 (.17)</td>
<td>.75 (.21)</td>
<td>.74 (.18)</td>
</tr>
</tbody>
</table>

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Rank = 24.2) and a significantly lower (U = 201.5, p = .002) mean rank in hostile autonomy behavior (Mean Rank = 20.8) compared to the insecure group (Mean Rank = 33.9).

The third hypothesis looked for effects of serotonin genotype and attachment at the level of personality, specifically at the level of aggressiveness and anxiety. Table 1 presents the means for the different groups. A 5-HTTLPR × attachment ANOVA with aggressiveness as dependent variable revealed a significant main effect for the 5-HTTLPR polymorphism (F(1, 90) = 4.4, p = .038) and for attachment quality (F(1,90) = 6.2, p = .014) on aggressiveness. For anxiety, no significant effects were found. Adolescents carrying two long alleles were rated as less aggressive (M = .70) than those carrying at least one short allele (M = .79), and those with secure attachment behavior strategies were rated as significantly less aggressive (M = .69) compared to the insecure attachment group (M = .79).

Discussion

The primary goals of this study were to examine the effects of molecular-genetic polymorphisms of the serotonin transporter gene and attachment in adolescence on emotionality and emotion regulation in an emotion-eliciting social context and on aggressiveness and anxiety as personality traits.

The literature on the effects of the VNTR polymorphism in the promoter region of the serotonin transporter gene suggests mainly an effect on emotional reactivity and, perhaps as a consequence, on emotion regulation (Canli & Lesch, 2007). We assessed emotion regulation patterns during a social interaction task with the mother, designed to elicit negative emotion in the adolescents. The findings showed that this situation validly induced negative emotions in the adolescents, independently of their genetic characteristics, and thus provided an appropriate situation to study emotion regulation (Cole et al., 2004). In the present study, we did not find a general, but a specific, genetic effect on emotionality. Whereas there was no significant genetic association with the general expression of negative emotions during this task, the study revealed a heightened emotional reactivity specifically to restrictions of autonomy for short 5-HTTLPR carriers in adolescence, an age period in which autonomy restrictions elicit negative emotions very intensely. Carriers of the short allele were significantly more sensitive to restrictions of their autonomy, so that maternal directing behavior led more often to impulsive contradictions and assertive reactions in the adolescents. However, while the general negative emotional reactivity to restrictions and threats to autonomy in adolescence might have a genetic basis, the quality of emotion regulation during the interaction was influenced by differences in attachment security. The higher negative emotionality of the S allele carriers was expressed in a cooperative way towards the mother when the adolescent’s attachment pattern was secure. However, when the attachment pattern was insecure, the negative emotionality remained unregulated and was expressed as aggressive assertiveness, in hostile verbal and physical attacks that further endangered the relationship. As adolescence is characterized by increased daily conflicts with parents, mainly about parental restrictions of everyday life (Laursen et al., 1998), this emotion dysregulation of the insecure attachment group might describe a potential risk mechanism for the development of psychopathology and of externalizing behavior in particular because chronic impulsive disputes can undermine the effectiveness of parenting (Dadds et al., 1992). Allen and colleagues (1998) found that high maternal control in an adolescent risk group was effective in reducing externalizing behavior only when the adolescent’s attachment representation was secure. Primate studies show similar effects. Suomi (2006) reported that adolescent rhesus monkeys carrying the short variant of the 5-HTTLPR and lacking care and control by their mothers displayed an increased rate of aggression, in contrast to mother-reared primates. In a similar vein, violent behavior was best predicted by the interaction of adverse life experiences and the presence of the short promoter allele of the SHRT gene (Reif et al., 2007).

Thus, at least in adolescence, the 5-HTTLPR does not seem to affect emotionality as observable in a more general heightened frequency of emotional expression but particularly in impulsive assertive reactions to stage-salient emotional elicitors, such as restrictions of autonomy. However, this genetic risk may not normatively develop into maladaptive emotion dysregulation as a risk factor for psychopathology (Cole et al., 1994) when adolescents have developed a secure attachment behavior strategy.

Effective emotion regulation abilities can be especially protective for children with high negative emotionality to avoid the development of externalizing behavior (Eisenberg & Morris, 2002). Parenting in adolescence – may it be cooperative and guiding behavior during an emotionally arousing task, as in this study, or controlling behavior, as in the study by Allen and colleagues (1998) – seems to be more effective when the adolescents have a secure attachment organization so that they can effectively regulate negative emotions within relationships. Allen and Hauser (1996) concluded from their research that the development of autonomy inevitably leads to conflicts between parents and adolescents and that these conflicts promote ego development if they are resolved in a supportive relationship. Similarly, Klimes-Dougan et al. (2007) emphasize the importance of supportive parenting for adolescents’ emotion regulation.

Thus, for adolescent carriers of the short serotonin transporter allele, secure attachment strategies
might be protective against later maladjustment. According to our findings, the genetically influenced tendency to impulsively react to restrictions of autonomy in adolescence does not seem to be a risk factor for maladjustment in itself, because this disposition can be regulated cooperatively within a secure attachment relationship.

A more general assessment of the personality traits aggressiveness and anxiety revealed that differences in attachment quality as well as in the 5-HTTLPR contributed to aggressiveness, but not to anxiety. Regarding the genetic effect, this is in line with other studies showing similar associations with aggression for school-age children (Haberstick, Smolen, & Hewitt, 2006) and adults, specifically for hostile aggressiveness (Hennig et al., 2005). This supports the conclusion by Carver and Miller (2006) that the link between serotonin functioning and anxiety is questionable. There seems to be more reliable empirical evidence for a link to impulsivity and aggressiveness. The presented results lead us to suggest that in future studies the elicitors of hostile aggressiveness should be taken into account.

The attachment effect on aggressiveness adds further empirical evidence to earlier studies in infancy (Suess et al., 1992; Sroufe, Egeland, and Carlson, 1999) and adolescence (Allen et al., 2007). Attachment behavior patterns are characterized by effective or ineffective emotion regulation within specific close attachment relationships. Attachment theory proclaims internal working models of attachment as control mechanisms assumed to influence perception, interpretation, and emotion regulation during social interactions (Zimmermann, 1999). This approach can be used to explain the negative association between secure attachment organization and aggressiveness. There is empirical evidence showing that insecure attachment patterns are associated with a hostile interpretation bias in social conflict or rejection situations and with less adaptive emotion regulation (Suess et al., 1992; Zimmermann, 1999), important process variables for the explanation of aggressive behavior (Lemerise & Arsenio, 2000). However, this needs further investigation.

The present study has some limitations that need to be considered before the results might be generalized.

With respect to standards of molecular-genetic studies (Murphy & Lesch, 2008), the sample size of this study was relatively small, which decreases statistical power. In addition, we did not investigate the effects of cumulative environmental risks (Caspi et al., 2003) or the interaction with other candidate genes or the L(G) variant of the 5-HTTLPR which might be functionally similar to the short (S) variant of the 5-HTTLPR. Moreover, other social interaction situations eliciting negative emotions and other interaction partners (e.g., fathers, peers) should be considered. Thus, replications are required. The different results for observed hostile behavior in comparison to aggressiveness as personality traits still need to be addressed.

Notwithstanding these limitations, we believe this study offers a valuable approach to study genetic and attachment influences on emotion dysregulation as a psychopathological risk in adolescence within a transactional model of development. The genetic risk for impulsive reactivity may develop into a cooperative or a maladaptive and hostile style of emotion regulation, depending on attachment security which is based on sensitive caregiving experiences.

Acknowledgements

This study was supported by the Koehlerstiftung and the German Research Foundation (SP 312/16-1 & ZI 511/13-1). We thank Stephanie Ast-Scheitenberger, Melanie Pillhofer and Julia Popp (Erlangen) for the maternal intrusiveness coding, Fatma Celik and Nadine Lüpschen (Dortmund) for the coding of the talk show, and Zsolt Ronai, Budapest University, for conducting the gene analysis. Finally we express our gratitude to the families for their enduring participation.

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Key points

- The short variant of the serotonin transporter polymorphism has been associated with a wide spectrum of emotion dysregulation and psychopathology.
- Insecure attachment is a risk factor for psychopathology because it is associated with emotion dysregulation. Attachment patterns are activated specifically when negative emotions are aroused.
- The findings suggest a gene-attachment interaction for emotion regulation: the genetic predisposition for adolescents to react impulsively when their autonomy is threatened is moderated by concurrent attachment security, thus being expressed as socially appropriate assertiveness.
- Clinical implications: genetic predispositions for emotion dysregulation may be influenced socially in their phenotypical behavior outcome. This offers a starting point for attachment-based interventions.
References


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Manuscript accepted 29 July 2009