

Abstract

In view of the theory that the attachment relationship provides a foundation for the development of emotion regulation, here, we evaluated (1) whether change in attachment security from 4 to 6 years predicts change in emotion regulation from 6 to 8 years and (2) whether *5-HTTLPR* moderates this relation in a Norwegian community sample (n=678, 99.7% Caucasian). Attachment was measured with the Manchester Child Attachment Story Task, and teachers completed the Emotion Regulation Checklist. Attachment security was modestly stable, with children becoming more secure over time. Regression analyses revealed that increased attachment security from 4 to 6 forecasted increases in emotion regulation from 6 to 8 and decreased attachment security forecasted decreases in emotion regulation. This effect was strongest among the *5-HTTLPR* short-allele homozygotes and, according to competitive model fitting, in a differential-susceptibility manner.

Keywords: 5-HTTLPR, attachment, differential susceptibility, emotion regulation, serotonin transporter gene

Change in Attachment Predicts Change in Emotion Regulation

— Particularly among 5-*HTTLPR* Short-Allele Homozygotes

A child's attachment to parents has been regarded as a foundation on which emotion regulation develops. Indeed, attachment has been described in terms of dyadic regulation of emotion (Sroufe, 1996) and as a regulation theory (Schoore, 2001). As a result, attachment is presumed to have wide ranging, even if indirect, developmental consequences. After all, emotion regulation has been shown to play a pervasive role in human development (Cole, 2014), including social functioning (e.g., Blair et al., 2015; English, John, Srivastava, & Gross, 2012) and psychopathology (e.g., Bosquet & Egeland, 2006; Halligan et al., 2013). However, beyond the infancy and early-childhood years, there has been limited investigation of associations linking attachment and emotion regulation (Borelli et al., 2010; Kim & Page, 2013). This is a notable lacuna, in that research on younger children (e.g., Kochanska, Philibert, & Barry, 2009; Vondra, Shaw, Swearingen, Cohen, & Owens, 2001) may not generalize to older children or across developmental periods. This is no doubt due, at least in part, to the well-known fact that development continues well beyond the early-childhood years.

Thus, the work presented herein seeks to extend research on an important ecological transition (Bronfenbrenner, 1979), the shift from day care to school, while examining links between attachment and emotion regulation. Given the evidence that environmental effects, **including** attachment effects, may vary as a function of children's genetic make-up, we further evaluate the possibility that links between (change in) attachment and (change in) emotion regulation may be genetically moderated by a widely studied polymorphism that has been repeatedly found to operate in such a manner, namely, the serotonin-transporter gene, 5-*HTTLPR*. Indeed, in exploring this issue, we competitively evaluate diathesis-stress and

differential-susceptibility models of gene-X-environment (GXE) interactions, conceptualizing attachment as a reflection of exposure to quality of the rearing environment.

Emotion Regulation and the Forgotten Years

Emotion regulation is an important component of emotional competence (Saarni, 1999) and can be defined “as the ability to respond to the ongoing demands of experience with the range of emotions in a manner that is socially tolerable and sufficiently flexible to permit spontaneous reactions as well as the ability to delay spontaneous reactions as needed” (Cole, Michel & Teti, 1994, p. 76). Although a conceptual consensus on emotion regulation is still lacking (Cole, 2014), there is broad agreement that regulatory abilities emerge as a result of socio-emotional exchanges within the family, particularly between parents and children (Thompson, 2014). If these parent-child transactions are problematic, children may develop less efficient regulatory capacities or even emotion dysregulation (Cole et al., 1994). This is shown by, for example, strikingly low emotional intensity or avoidance of certain emotions, both of which can adversely affect children’s social relations (Cole et al., 1994).

Emotion regulation develops throughout the lifespan (Cole, 2014). In middle childhood, these capacities become more complex as evidenced by increased emotional understanding as well as the integrative processing of complex cues regarding others’ emotions (Shields & Cicchetti, 1997). Furthermore, due to the demands of schooling, middle childhood is a period of exposure to new social roles and peer hierarchies (see, e.g., Colle & Del Giudice, 2011). Children are now expected to display self-regulation in relation to their classmates, peers, teachers, and surroundings while becoming less dependent upon external regulators. In fact, the developmental and social changes—and challenges--that take place in middle childhood may be no less profound than those that characterize earlier developmental epochs (Eccles, 1999; Mah & Ford-Jones, 2012).

Despite these observations, most emotion regulation research conducted by developmentalists has focused on children prior to school entry (Adrian, Zeman & Veits, 2011). It is not surprising, then, that middle childhood, extending from approximately six to 12 years of age, has been described as “the forgotten years” (Mah & Ford-Jones, 2012). Thus, we focus on changes in attachment and emotion regulation across the transition to early middle childhood.

Attachment and Emotion Regulation

Building upon Bowlby’s (1969) hypothesis that attachment experiences organize the child’s ability to cope with future internal and external demands, attachment researchers (e.g., Sroufe, 1996) emphasize that *secure attachment* (Ainsworth, Blehar, Waters, & Wall, 1978) promotes emotion regulation. According to Thompson (2016; 2014) and Cassidy (1994), securely attached children tend to have parents who are sensitive to their children’s experience of uncertainty and distress and who are open and responsive to a wide variety of children’s emotions. Thus, in arousing situations, secure children experience parents as sources of comfort and support, which reduces emotional tension. As a result, these children develop skills and confidence in managing their own emotions (Sroufe, Egeland, Carlson & Collins, 2005). At a representational level, security-inducing care gradually promotes an internal working model (IWM) (see Bretherton, Ridgeway, & Cassidy, 1990), reflecting positive beliefs of self and others, including the beliefs that one is worthy of being cared for and that significant others represent a safe haven whenever the attachment system is activated.

In contrast, insecure children’s experiences are marked by episodes of emotional distress that is poorly handled by caregivers. The resulting IWMs may include inefficient scripts for expressing and/or managing diverse emotions in the longer term, placing these children at risk of becoming emotionally dysregulated (see Schore, 2003). Consider evidence that compared

to securely attached children, insecure children demonstrate increased anger and fear and decreased joy during the first three years of life (Kochanska, 2001).

The attachment-emotional regulation dynamic is further intensified by the fact that overwhelming emotions typically arise in the context of attachment relationships (Cassidy, 2016). For example, a child who has been left reluctantly by the attachment figure may become angry or sad. This particular attachment figure is also the one to offer support at parting and upon reunion and, in that sense, is the source of both distress and regulatory scaffolding, thereby promoting the child's self-regulation. In fact, such situations were the starting point for Bowlby's attachment theory (see Ainsworth, Blehar, Waters & Wall, 1978; 2015).

Although the links between attachment and emotion regulation during middle childhood have not been entirely ignored by developmentalists, the available work is limited (see Parrigon, Kerns, Abtahi, & Koehn, 2015 for a review) by a lack of longitudinal designs, sample diversity, and informants other than child and parent regarding children's emotion regulation. Nevertheless, available evidence indicates that greater attachment security in 8- to 12-year-olds is associated, contemporaneously, with more competent emotional coping strategies (Contreras, Kerns, Weimer, Gentzler, & Tomich, 2000; Kerns, Abraham, Schlegelmilch, & Morgan, 2007), enhanced emotion identification (Brumariu, Kerns & Seibert, 2012), increased regulation of threat-induced reactivity (Borelli et al., 2010), and more mature selection of emotion regulation strategies when confronted with hypothetical challenges (Colle & Del Giudice, 2011). However, we would be remiss not to acknowledge that Kim and Page (2013) failed to detect links between attachment and emotional regulation in this age group. The work reported herein seeks to build upon and extend prior work by employing a longitudinal design, studying a large representative Norwegian community sample, and relying on teacher reports of emotion regulation.

This study is informed by an organizational view of the role of attachment in development (Sroufe 2016; 2005; Sroufe & Waters, 1977), and thus by the way that individual patterns of early behavior organize subsequent patterns of adaptation. This process operates across contexts (Sroufe, 2016) and especially with regard to the effect of attachment on self-management or self-regulation (Sroufe, 2016, Sroufe et al., 2005). Despite the crucial factor of timing, early childhood experiences sometimes have greater impact on later development than more recent experiences (Sroufe, 2013). In line with this idea is thorough methodological evidence that maternal sensitivity in the first years predicts social competence as late as 15 years of age (Fraley, Roisman & Haltigan, 2013). Such results provide a basis for hypothesizing that earlier measured attachment should predict later measured emotion regulation. Given the long-term and sometimes slow-to-see effects of attachment on development (Sroufe, 2016), we hypothesize that the level and change in attachment security from 4 to 6 years of age will organize and influence the subsequent level and change in capacities for emotion regulation from 6 to 8 years of age. The measurement schedule of the longitudinal study on which this report is based does not afford reciprocal evaluation of the effects of emotion regulation on attachment, and it allows us to consider attachment only at ages 4 and 6 and emotion regulation at ages 6 and 8.

Change and Stability in Attachment Representations

Our effort to determine whether change in attachment predicts future change in emotion regulation is based on the view, articulated previously, that development continues beyond the preschool years. Indeed, we conceptualize the growth of security and insecurity from 4 to 6 as part of an on-going developmental trajectory, which leads us to predict that change in attachment over this period should contribute the developmental trajectory of emotion regulation.

The importance of conducting longitudinal inquiries, particularly those focused on change in attachment, is underscored by the fact that attachment security may not be as stable as long presumed, which should not be entirely surprising. After all, Bowlby (1969) acknowledged that attachment-shaped internal-working models could change, even if this was less likely the older children became. As it turns out, meta-analyses indicate that attachment security is, at best, moderately stable over time (Fraley, 2002; Pinquart, Feussner, & Ahnert, 2013). A comprehensive study recently documented weak long-term stability when investigating stability from the first 3 years of life to late adolescence (Groh et al., 2014). Stability is enhanced, however, with assessment intervals less than two years, equal methods, and measurement beyond infancy and at the representational level (Pinquart et al., 2013). Furthermore, in line with Bowlby's hypothesis of increasingly stable IWMs, stability coefficients are more substantial beyond the age of 6 (Pinquart et al., 2013).

Whether stability or instability best characterizes consistency in attachment over time, it is no doubt the case that some insecure individuals become more secure over time and that some secure individuals become more insecure over time. Such patterns of change in attachment have been related, for example, to altered caregiving behavior, negative life events, and parental marital satisfaction (see McConnell & Moss, 2011 for a review). Arguably, a child who becomes more secure over time, perhaps even changing from insecure to secure, would be expected to evince positive growth in other domains of development as well, possibly even more so than a child who remains consistently secure over time. This reasoning leads us to hypothesize that increasing security across the transition to school will forecast increased levels of emotion regulation and that increasing levels of insecurity will forecast decreased levels of emotion regulation.

Given our focus on the predictive power of change in attachment security, we are positioned to also examine the stability of attachment. Here, we extend such work by

measuring attachment at the representational level, relying on the Manchester Child Attachment Story Task (MCAST; Green, Stanley, Smith, & Goldwyn, 2000). To our knowledge, only two investigations to date have examined stability at the representational level. First, using the Attachment Story Completion Task (Bretherton et al., 1990), Stievenart, Roskam, Meunier, and Van de Moortele (2014) chronicled modest stability over a 2-year period (i.e., $r = 0.32$, intraclass correlation coefficient (ICC) = 38%) among 3- to 8-year-olds ($n = 358$). Green et al. (2000), in contrast, documented substantial stability (i.e., 76.5%) in attachment representations across a 6-month interval in a sample of 5- to 7-year olds ($n = 33$ at follow-up) using the MCAST. The latter finding needs to be interpreted in the context of a high base rate of security as well as wide confidence intervals given the limited sample size. Thus, we will investigate stability and change in secure attachment representations in children from age 4-6 years in a representative community sample; The Trondheim Early Secure Study (TESS).

Gene-by-Attachment Interaction

Even if earlier attachment influences later emotion regulation, there is reason to believe that effects of attachment, like a variety of environmental exposures and developmental experiences, could prove more operative in the case of some and less influential in the case of others (Belsky, Bakermans-Kranenburg & van Ijzendoorn, 2007; Ellis, Boyce, Belsky, Bakermans-Kranenburg & van Ijzendoorn, 2011). This raises the possibility that presumed effects of attachment may vary across children, including perhaps as a function of their genetic make-up. Indeed, to the extent that attachment reflects, at least in part, the legacy of rearing experience (see Fearon & Belsky, 2016, for an updated review), the study of attachment-*X*-gene interaction can be conceptualized, as it has been, as relevant to the broader investigation of GXE interactions. Thus, in addition to evaluating prospective linkages between changes in attachment security and emotion regulation, we address the question of

whether such associations vary as a function of children's genotypes. Here, we focus on the 5-*HTTLPR*-polymorphic region given its particular role in emotion regulation (Canli & Lesch, 2007) as well as prior work showing that this polymorphism interacts with a variety of developmental experiences and environmental exposures (Canli & Lesch, 2007; van Ijzendoorn, Belsky, & Bakermans-Kranenburg, 2012), including attachment disorganization (Kochanska et al., 2009) and maternal unresponsiveness (Davies & Cicchetti, 2014).

Whereas the traditional diathesis-stress thinking stipulates that adverse contextual conditions will negatively affect more "vulnerable" individuals than others, differential susceptibility thinking contends that such putatively vulnerable individuals are also more susceptible to supportive or positive experiences than others, making them generally more developmentally plastic, "for better and for worse" (Belsky et al., 2007; Belsky & Pluess, 2009, 2013; Ellis, Boyce, Belsky, Bakermans-Kranenburg & van IJzendoorn, 2011). Two versions of the differential susceptibility framework can be distinguished (Belsky, Pluess & Widaman, 2013); while the "strong" version stipulates that some individuals are susceptible to environmental influences and others are not, the "weak" version stipulates that some are more susceptible than others.

Given the presumption that attachment security is, at least in part, a reflection of rearing experience, such as parental sensitivity (Fearon & Belsky, 2016), we hypothesize that change in attachment security will predict changes in emotion regulation for some children more than others. What remains unclear, however, is whether the genetic moderation of the effect of change in attachment security that we anticipate will better fit the weak or strong diathesis-stress or differential-susceptibility models of the GXE interaction. As a result, we employ a model fitting approach that competitively evaluates the fit of these alternative conceptual frameworks (Belsky et al., 2013; Widaman et al., 2012). Notably, such evaluation of vulnerability vs. susceptibility models is not merely of theoretical interest. After all, support

for differential susceptibility would imply that those most vulnerable to adversity might be most likely to benefit from interventions--to promote the development of secure attachment in the first place and/or to foster security given an insecure base.

The Moderating Role of 5-*HTTLPR*

To date, some of the most promising work suggesting that the effects of attachment may be genetically moderated comes from GXE research on the serotonin transporter 5-HTT, which is encoded by the *SLC6A4* gene (Canli & Lesch, 2007). As reviewed by Canli and Lesch (2007) and Hariri and Holmes (2006), 5-HTT recycles serotonin from the synaptic cleft to the presynaptic neuron and influences the duration and intensity of serotonin signaling with post-synaptic receptors within the affective corticolimbic circuitry. Thus, 5-HTT is involved in the brain's emotional communication (Canli & Lesch, 2007; Hariri & Holmes, 2006).

Of importance to the current study is that humans vary in the *efficiency* of how *SLC6A4* codes for 5-HTT. *SLC6A4* contains a common polymorphism, 5-*HTTLPR*, which is usually reported with two allele-variations: a short ("S") and a long ("L") allele. Notably, the S allele is associated with reduced **transcription of the 5-HTT-gene promoter (Lesch et al., 1996)**. As a result, S carriers have elevated levels of extracellular serotonin, which is thought to lead to heightened emotional reactivity. For example, S carriers evince stronger amygdala reactivity (see Munafò, Brown & Hariri, 2008 for a meta-analysis) and cortisol responses to stressors (Gotlib, Joormann, Minor & Hallmayer, 2008).

Meta-analytic evidence also documents heightened emotional reactivity among S *homozygotes* in particular (Miller, Wankerl, Stalder, Kirschbaum & Alexander, 2013). This heightened reactivity highlights the potential for S homozygotes to develop differently than other children with respect to emotion regulation. Moreover, additional meta-analytic work indicates that the S allele moderates the effects of a variety of environmental exposures, at least in Caucasian children, in a manner consistent with differential-susceptibility thinking

(van Ijzendoorn et al., 2012). With regard to parameterizing heterozygotes (SL) with S or L homozygotes, the literature is equivocal (see, e.g., van Ijzendoorn, et al., 2012). For this reason, we conduct preliminary analyses to address this issue before testing competing models of GXE.

In general, prior research has raised the prospect that L carriers may be less prone to emotional dysregulation. As a result, attachment security, as an environmentally induced regulatory mechanism, could be especially important for the development of emotion regulation among more reactive S carriers. In fact, S carriers have been found to be most affected by their attachment styles with regard to self-regulation in preschool (Kochanska et al., 2009), as well as autonomy and aggression (Zimmermann, Mohr, & Spangler, 2009) and stress and depression (Starr, Hammen, Brennan, & Najman, 2013) in adolescence. Whereas the research by Kochanska et al. (2009) proved consistent with the diathesis-stress model, the work of Starr et al. (2013) and Zimmermann et al. (2009) appears more consistent with differential-susceptibility theorizing.

In summary, the purpose of this study is threefold: (1) to document stability and change in children's attachment security from 4 to 6 years of age as measured by the MCAST; (2) to evaluate whether change in attachment predicts subsequent change in children's emotion regulation from 6 to 8 years of age; and (3) to determine whether such predictions are moderated by the 5-*HTTLPR* polymorphism in a differential-susceptibility- or diathesis-stress-related manner, with S carriers proving more susceptible to environmental influences than L carriers.

Methods

Participants and recruitment

TESS is a representative cohort study with the aim of detecting risk and protective factors in child development. The Regional Committee for Medical and Health Research Ethics in

Mid-Norway approved all research procedures prior to conducting this study. The procedure and recruitment have been presented elsewhere (Wichstrøm et al., 2012); hence, only a limited outline follows. The data collection began in 2007. All children born in 2003 and 2004 in the city of Trondheim, Norway (approx. 185,000 inhabitants) and their caregivers were invited to participate in the study. The families were recruited via the municipal well-child clinics, which perform mandatory health checkups. A letter of invitation and the Strengths and Difficulties Questionnaire (SDQ) 4-16 version (Goodman, 1997) were mailed to the caregivers with their ordinary scheduled appointments for their 4-year-olds. The SDQ is a 31-item measurement of mental health problems in children from 4 to 18 years of age. Public health nurses informed the families about TESS and obtained written consent for participation. The consent rate among eligible families was 82.1%.

To increase sample variability, children with higher scores on the SDQ were oversampled. Accordingly, the SDQ scores on the problem subscales (emotional problems, conduct problems, hyperactivity/inattention, and peer relationship problems) were divided into 4 strata using the cut-off ranges of 0-4 (44.2% of the population), 5-8 (29.5% of the population), 9-11 (18.5% of the population), and 12-40 (7.8% of the population). Using a random number generator, 38.1%, 49.1%, 71.4%, and 89.2% of children in strata 1, 2, 3, and 4, respectively, were drawn to participate in a comprehensive study within the 6 weeks following the health checkup. Of the 1,250 invited families, 995 (79.5%) children ($M_{age}=4.5$, $SD=0.25$) accompanied by one caregiver attended the subsequent assessment at the university clinic. One participant had missing information on the SDQ and could not be included in the analyses. At T1, 845 (85%) caregivers were mothers, and 149 (15%) were fathers. At T2, 648 (81.5%) were mothers, and 147 (18.5%) were fathers.

Regarding attrition during the recruiting phase, the dropout rate was not different across the 4 SDQ strata ($\chi^2=5.70$, $df=3$, NS) or between genders ($\chi^2=0.23$, $df=1$, NS). A follow up

took place after two (T2) and four (T3) years; 795 children participated at T2 when they had started first grade ($M_{\text{age}}= 6.7$ years, $SD=0.17$), and 699 children participated at T3 when they were in third grade ($M_{\text{age}}= 8.8$ years, $SD =0.24$). Almost equal numbers of girls (49.5% and 51.3%) and boys (50.5% and 48.7%) participated at T2 and T3, respectively.

To address dropout beyond the recruiting phase, attrition analyses were run with all study variables. Attachment (T1, T2) did not predict attrition. Children's verbal comprehension predicted attrition from T1 to T2 (Odds Ratio (OR) =0.99, 95% $CI=0.99-.99$) and from T2 to T3 ($OR =0.99$, 95% $CI=0.98-0.99$). Emotion regulation at T1 predicted attrition from T2 to T3 ($OR=0.51$, 95% $CI=0.34-0.76$). However, when analyzing the total explained variance in attrition from T2 to T3, emotion regulation was no longer significant ($OR=0.68$, 95% $CI=0.44-1.06$), and the combined effect of predictors of attrition was modest (Cox & Snell $R^2=0.018$, Nagelkerke $R^2=0.039$). The effect of predictors from T1 to T2 was also modest (Cox & Snell $R^2=0.005$, Nagelkerke $R^2=0.009$).

With consent from the parents, the child's primary teacher completed a questionnaire concerning the child's emotion regulation at T2 and T3. There were approximately three children from each class participating in TESS, but some classes and even schools had only one participating child. Children in Norway start school when they are 6 years old, and the teacher respondent had known the child for an average of 6 months at T2 and 2.5 years at T3. The response rate was 99.1% at T2 and 86.1% at T3. The majority of teachers was female (84.6%, $n=666$ at T2; 77.7%, $n=470$ at T3).

The final sample included in the current study is based on the children who were successfully genotyped ($n=678$). The genotyped children did not diverge from those not genotyped in terms of the study variables except in the case of verbal comprehension ($OR=0.99$, $CI=0.98-0.99$). Notably, the participants in TESS were ethnically homogenous. At T2, when DNA was sampled, 99.7% of the children were Caucasian.

Measures

Attachment Security. The children's attachment representations were assessed at T1 and T2 using the MCAST (Green et al., 2000). The MCAST has been applied in a range of low- and high-risk studies, and a number of findings have underscored its reliability, internal consistency, and key components for validity (see Barone and Lionetti, 2012 for a summary).

The MCAST integrates age-relevant aspects from the Strange Situation procedure (SSP; Ainsworth et al., 1978), which measures behavior, and the Adult Attachment Interview (AAI; George, Kaplan, & Main, 1996), which measures narratives (Green et al., 2000). Therefore, the MCAST uses doll play and story stems to evoke attachment representations. Procedurally, the child is shown a non-attachment-related vignette to establish the testability of the child (i.e., a breakfast vignette), followed by four attachment-related stories designed to elicit distress and that provide the basis of scoring attachment security. The administrator establishes a story that includes a child doll and a mommy or daddy doll (depending on the gender of the parent that accompanied the child to the clinic). The child's identification with the doll figure is emphasized but made implicitly (e.g., "So this is the (name of child) doll, and this is the mommy/daddy doll"). The stories begin with everyday events followed by a distressing event: the child 1) is alone when waking up from a nightmare in the middle of the night, 2) hurts a knee while biking, with pain and bleeding, 3) experiences acute abdominal pain when watching TV alone, and 4) becomes lost while with the parent at a large shopping mall. This format is designed to activate the child's attachment system and, hence, attachment-related behaviors and thoughts, which resemble those used in the SSP or the "five adjective questions" in the AAI. As the story climaxes, the administrator asks, "What happens next?" to facilitate the completion of the child's narrative. The child is then asked about the feelings experienced by the child and the parent doll.

For the sake of clarity, the MCAST was administered in a suitable room at our university clinic, and the parent was not in the room with the child during the MCAST procedure. The entire MCAST procedure was videotaped, and reliable coders unaware of any information regarding the child and family coded each attachment vignette according to the MCAST coding manual (Green, Stanley, Goldwyn & Smith, 2007). **Different teams coded T1 and T2, and all coders were certified for research purposes in collaboration with the MCAST founders at the University of Manchester, UK.** A random 10% of the MCAST videos were re-coded by blinded coders. **As regards coders' agreement of security ratings,** the ICC reliability (see e.g. Janson & Olsson, 2004) across multiple pairs of **coders** was .81 at T1 and .86 at T2.

Given interest in testing diathesis-stress vs. differential-susceptibility models of person-X-attachment interaction, as well as to increase variation and thus statistical power (Futh, O'Connor, Matias, Green, & Scott, 2008), we employed a continuous approach in analyzing attachment security. Following a procedure described by Hygen, Guzey, Belsky, Berg-Nielsen, and Wichstrøm (2014) and also implemented elsewhere (Viddal et al., 2015; Wichstrøm, Belsky & Berg-Nielsen, 2013), the primary categorization (A, B, C, and D) of each vignette was coded as 1 (present) or 0 (absent). However, secondary classifications should also be considered when attachment is viewed continually. Given that secondary classifications are not as decisive as primary classifications; they were given a weight of 0.5 if present. The total attachment scores were computed by averaging the primary and secondary scores (range 0-1) across the four story completion vignettes. Hence, a child who was given a primary classification of B on three of the total of four vignettes in the MCAST and a secondary classification of B on one vignette would be given a B-score of 0.875 ($[1 + 1 + 1 + 0.5]$ divided by 4 vignettes to attain a mean score). Accordingly, the highest B-mean score attainable was 1.0. Conversely, a score of 0 would result if the child evinced insecure or

disorganized attachment in all four vignettes. Notably, this analytic approach does not violate the manual's principles; rather, it takes into account that children's strategies may vary within and across vignettes. Additionally, this method has previously been employed to the current sample by developers of the MCAST (see Viddal et al., 2015) and has been shown to be valid in predicting self-regulation (Viddal et al. 2015), aggression, and social skills (Hygen et al., 2014).

Due to the risk that language ability could affect attachment measurements, especially in light of evidence that securely attached children have stronger verbal skills than their insecurely attached counterparts (McElwain, Booth-LaForce, Lansford, Wu, & Dyer, 2008), the children's *receptive language ability* was measured at T1 using the Norwegian version of the Peabody Picture Vocabulary Test (PPVT-III; Dunn & Dunn, 1997) ($\alpha=0.98$) and served as a control variable in all statistical analyses.

Emotion Regulation. Emotion regulation was measured with the Emotion Regulation Checklist (ERC) (Shields & Cicchetti, 1997), which was completed by teachers at T2 and T3. The ERC consists of 24 items, for which an adult who is familiar with the 6- to 12-year-old child judges how characteristic each statement is of a particular child on a Likert scale ranging from 1 (almost always) to 4 (never). For the purposes of this study, we applied the ER subscale ($\alpha=0.78$ (T2, T3)), which comprises 8 items describing situationally appropriate affective displays, empathy, and emotional self-awareness (e.g., "can say when s/he is feeling sad, angry or mad, fearful, or afraid;" "seems sad or listless;" or "displays appropriate negative emotions in response to hostile, aggressive, or intrusive acts by peers"). The ER scale was chosen due to its particular focus on regulation and to avoid a subscale that too closely resembles the temperament trait "negative affect," which has been suggested as a plasticity factor itself (Belsky et al., 2007) and therefore could complicate the interpretation of any findings.

5-*HTTLPR* Genotyping. Genotyping was performed using two milliliters of saliva collected from children at T2 using the Oragene DNA/saliva kit (DNA Genotek, Ottawa, Ontario). DNA was later extracted and stored according to the manufacturer's protocol. The PCR of the 5-*HTTLPR* polymorphism was performed with the Ampli Taq ® 360 DNA polymerase kit (Applied Biosystems). The amplification reactions were performed with a total volume of 25 µl containing 10-100 ng genomic DNA, 1.25 units of AmpliTaq 360 DNA polymerase, 0.75 mM MgCl₂, 16% (v/v) 360 GC Enhancer, 0.5 mM dNTP, and 0.3 µM of each primer. The 5-*HTTLPR* marker was genotyped by the size separation of the PCR product on the ABI 3730 DNA Analyzer (Applied Biosystems) and was sized utilizing the GeneScan 600 LIZ Size Standard (Applied Biosystems) and the ABI PRISM Gene Mapper ® software, version 4.0 (Applied Biosystems). The 5-*HTTLPR* genotype frequencies were consistent with the Hardy-Weinberg equilibrium ($\chi^2 = 2.77$, $p = 0.10$). Of 716 saliva samples, 678 were successfully genotyped. In all, 18.4% (n=125) of the children were identified as the SS genotype, 51.5% (n=349) were identified as the SL genotype, and 30.1% (n=204) were identified as the LL genotype.

Statistical Analyses

Descriptive and Regression Analyses. The effect of the development of attachment security on the development of emotion regulation was investigated via growth curve modeling using Mplus version 7.31 (Muthén & Muthén, 1998-2012). The level (i.e., intercept, set at age 6) and change in emotion regulation from 6 to 8 years of age were regressed on the intercept (set at age 4), and change in attachment security from age 4 to 6. Additionally, change in attachment and change in emotion regulation were regressed on their respective intercepts, and verbal comprehension was adjusted for. Missing data were handled through a Full Information Maximum Likelihood (FIML) procedure (see e.g. Enders, 2001). Notably, due to the lack of a third measurement point for attachment and emotion regulation, the error

terms were set to zero, which implied that we analyzed observed (not latent) growth, which in practice is a difference score. Furthermore, by adjusting for intercepts in all analyses, regression to the mean was taken into account.

Because we oversampled for children with high SDQ scores, all analyses were performed with weights proportional to the number of children in a specific stratum divided by the number of participants in that stratum; this strategy yielded corrected population estimates. A robust maximum likelihood estimator was applied, which also provided robust standard errors; notably, this approach is robust to moderate deviations from normality.

Testing for Differential Susceptibility. Due to the expectation that short-allele carriers would prove more susceptible to environmental, and thus attachment influences, and given our focus on changes in attachment predicting changes in emotion regulation, we employed a modified version of the competitive, model-fitting approach advocated by Widaman et al. (2012) and Belsky et al. (2013) for testing differential-susceptibility vs. diathesis stress. More specifically, two modifications were applied. First, the Widaman approach concerns whether the crossover point of the regression slopes among more and less susceptible individuals deviates significantly from the minimum and maximum observed values of the exposure. Thus, there is no prior testing of whether an interaction exists at the outset. To ensure that there was indeed a GXE interaction, we examined whether the effect of intercept and change in attachment on intercept and change in emotion regulation differed across the three allelic groups of SS, SL, and LL. This examination was performed via a multi-group analysis, in which the model fit when fixing the regression coefficient as equal in two allelic groups was compared with a model with a freely estimated coefficient. The resulting difference in model fit was tested with a Wald test with 1 *df*. The literature is equivocal in regards to the placement of the SL group. Therefore, we examined whether the

prospective effects differed between the SS and SL carriers, as well as between the SL and LL carriers.

Second, in the original approach (Widaman et al., 2012), a procedure to test whether the crossover point differs from 0 is described, which is appropriate if the lines cross near the y-axis. Thus, in the present case, this would imply testing for differences in emotion regulation when children mostly change and become more insecurely attached. However, the slopes may also cross near the other end of the attachment spectrum (which runs from 0 to 1 in the present case), that is, when children predominantly become more securely attached over time. Therefore, we also tested whether the crossover point was different from 1 (moving from insecure to fully securely). For the sake of order, please note that, similar to the analyses of main effects of attachment, baseline attachment was also controlled in the GXE analyses.

Results

Descriptives

Table 1 presents the descriptive statistics and correlations between all variables included in the analyses. Attachment security was modestly stable from 4 to 6 years of age. This rank-order stability was observed in the context of increasing levels of attachment security during this period, as shown by the significant mean growth per year ($M_{growth} = 0.08$, 95% $CI = 0.06-0.09$, $p < 0.001$). The level of emotion regulation slightly increased from 6 to 8 years of age ($M_{growth} = 0.02$, 95% $CI = 0.00-0.05$, $p = 0.02$). Verbal comprehension was correlated with both attachment and emotion regulation, underscoring its potential role as a confounder in the attachment-emotion regulation relation, and the importance of adjusting for verbal comprehension in further analyses.

Level and Change in Attachment Security Predicting Level and Change in Emotion Regulation

For the sake of order, there were no differences in the levels of attachment security to mothers and to fathers at T1 [Mothers ($M=0.51$, $SD=0.33$), Fathers ($M=0.49$, $SD=0.32$) ($t(804)=-0.64$, $p=0.526$)] or T2 [Mothers ($M=0.52$, $SD=0.33$), Fathers ($M=0.49$, $SD=0.34$) ($t(653)=-0.93$, $p=0.353$)].

As shown in Table 2, a higher level of attachment security at 4 years of age predicted a higher level of emotion regulation at 6 years of age as well as increased emotion regulation from the ages of 6 to 8. Beyond these effects of attachment security, children who evinced further increases in security from 4 to 6 years of age also evinced greater emotion regulation at age 6 and greater increases in emotion regulation from ages 6 to 8. Thus, there was an effect of change in attachment even when the intercept of attachment was controlled.

5-HTTLPR-X-Attachment

Before addressing the issue of genetic moderation of changes in attachment on changes in emotion regulation, it should be noted that SS carriers evinced decreased emotion regulation from 6 to 8 years of age compared with LL carriers ($\beta=-0.10$, $p=0.035$) (Table 2). Thus, a main effect of 5-HTTLPR on changes in emotion regulation was observed.

With regard to genetic moderation, inspection of Table 3 revealed no moderation by 5-HTTLPR of the effect of age-4 level and ages 4-to-6 changes in attachment on the *level* of emotion regulation at age 6. However, the effect of *change* in attachment from ages 4 to 6 years on *change* in emotion regulation from ages 6 to 8 years did prove to be genetically moderated, in that the effect in question was strongest for the SS group ($\beta=0.63$, $p=0.001$) and significantly different from that of the SL carriers ($Wald=16.36$, $p=0.001$) and LL carriers ($Wald=5.33$, $p=0.021$). The latter two groups did not differ from one another regarding this effect on change in emotion regulation; **hence, L carriers were grouped together in the subsequent analysis.**

Differential Susceptibility vs. Diathesis Stress. Notably, changes in attachment theoretically range from -1, a result of being fully secure at age 4 and becoming fully insecure at age 6, to 1, a result of being fully insecure at age 4 and becoming fully secure at age 6. A score of 0 indicates no change in attachment security over time. Because such scoring revealed that some children obtained scores of either -1 or 1, the crossover point central to distinguishing the two models of interaction should be significantly different from these maximum and minimum observed values to conform to differential susceptibility.

As illustrated in Figure 1, the results from the Widaman et al. (2012) method provided support for the differential susceptibility model. Not only did the crossover point for the simple slopes of the two allelic groups — S homozygotes and L carriers — fall quite close to 0, but the 95% *CI* included neither the minimum observed value (i.e., -1) nor the maximum observed value (i.e., 1) ($C=0.29$, 95% $CI=-0.02-0.59$).

The question thus remained whether the results conformed to weak or strong differential susceptibility. The strong version presumes that the less-susceptible group is not susceptible at all, whereas the weak version presumes only that the less-susceptible group is less—but still—susceptible than the more-susceptible group. Following Belsky et al. (2013), we compared a strong model, in which the effect of change in attachment on later emotion regulation was fixed at zero for the L carriers, with a weak model, in which the effect was freely estimated. The difference in model fit using Satorra and Bentler's (2001) procedure was significant ($\chi^2=6.05$, $df=1$, $p=0.01$), thereby supporting weak differential susceptibility. However, the effect among L carriers (the combined SL/LL group) was modest ($\beta=0.13$, $p=0.01$) compared with the stronger effect among SS carriers (Table 3).

Secondary Analyses and Supplementary Material

In light of other data available in the TESS Study archive, questions could be raised regarding what the results of our study would be if we had relied on parent rather than teacher

reports of child emotion regulation or focused on disorganized attachment. We therefore conducted secondary analyses.

Parent-reported emotion regulation: First, we reran the analyses with the parent-reported ERC ($\alpha=.65$). The correlations between parent and teacher reports were only modest ($r=.12$ to $-.22$); hence, similarity in the findings between teacher and parent ratings should not be expected. There were no main effects of attachment (neither intercept nor change) on parent-reported emotion regulation (neither intercept nor change). However, as can be seen in the online supplement (Table S1), findings resembling those obtained with teacher reports were found with respect to our GXE analyses: 1) increased attachment security predicted better emotion regulation at 6 years of age for the SS group. This result was significantly different from that of the LL group ($W=5.34, p=0.021$), and there was a tendency for SS to have a steeper increase than the SL group ($W=3.0, p=0.084$), and 2) increased attachment security predicted increased emotion regulation from 6 to 8 years of age for the SS group, and this increase was significantly stronger than in the LL group ($W=4.30, p=0.038$). Overall, the analyses with parent-reported emotion regulation replicated some of the teacher-generated data but were insufficient to conduct the original analyses of differential susceptibility vs. diathesis-stress.

Attachment disorganization: Second, our main findings pertained to the degree of attachment security. However, because children are rated on organized insecure attachment strategies (A and C) as well as disorganization (D) (Main & Solomon, 1990), it could be that low security scores, and thus our findings, did not merely reflect insecurity but rather disorganization. We therefore tested whether the effect of security (on teacher-reported emotion regulation) would remain if we adjusted for disorganization. The disorganization variable was scored similarly to attachment security, and these variables proved to be highly and negatively correlated ($r=-.68$ at T1, $r=-.62$ at T2).

The effects of disorganization on emotion regulation were first investigated alone (for full results, see Table S2). Disorganization at 4 years predicted emotion regulation at 6 years ($\beta = -.32, p < .001$) and change in emotion regulation from 6 to 8 years of age ($\beta = -.21, p = .002$). Furthermore, change in disorganization from 4 to 6 years of age predicted emotion regulation at 6 years ($\beta = -.23, p = .001$). However, change in disorganization only predicted change in emotion regulation from 6 to 8 years of age to a marginal extent ($\beta = -.12, p = 0.091$).

When all analyses from which these results emerged were rerun controlling for attachment security, all effects of disorganization were reduced to insignificance (Table S3) (note: multicollinearity diagnostics revealed acceptable values [Variance Inflation Factors (VIF) < 1.9]). These results dissuaded us from addressing the GXE issue using disorganization as the E variable. In sum, secondary analyses provided some additional, although not identical, evidence that attachment is related to future emotion regulation and that attachment disorganization does not add predictive power over and above attachment security.

Discussion

Attachment researchers have theorized and provided evidence that secure attachment promotes the development of emotion regulation, with other work making clear that emotion regulation is predictive of a wide range of psychosocial outcomes and psychopathological conditions. However, beyond the early-childhood years, the available evidence consistent with the claim that attachment security enhances emotion regulation is modest and methodologically limited (Parrigon et al., 2015). Thus, we sought to extend existing research, taking advantage of a three-wave longitudinal study of a Norwegian community sample to investigate whether changes in attachment predicted changes in emotion regulation and whether such an anticipated effect proved most pronounced among children who were homozygous for the 5-*HTTLPR*-S allele. Recall that analytic possibilities were circumscribed

by the fact that attachment was only measured at ages 4 and 6 and emotion regulation only at ages 6 and 8.

The most notable finding emerging from this inquiry was that an increase in attachment from 4-6 years of age forecast an increase in emotion regulation from 6-8 years of age, even with initial levels of attachment security and emotion regulation controlled. This effect was considerably stronger for children who were homozygous for the S allele of 5-*HTTLPR* than for the L carriers. However, because the effect in question also proved evident in the case of L carriers, the weak rather than strong version of differential susceptibility best characterized the findings. In other words, although an increase in security predicted a prospective increase in emotion regulation for all children, irrespective of their genotype, this effect was most pronounced in the case of S homozygotes. These results extend research on the putative effect of attachment security on emotion regulation beyond the preschool years and add further evidence of differential susceptibility in the context of 5-*HTTLPR*. They also contribute to the literature on change and stability in attachment representations. We elaborate on these observations below, beginning with the last point.

Stability and Change in Attachment Security

The stability of attachment security from 4 to 6 years of age, as measured by the MCAST, was modest to moderate in magnitude. Only two other studies have investigated change and stability in attachment security as measured at a representational level during early childhood. Our results closely resemble Stievenart et al.'s (2014) findings of modest two-year stability among 3- to 8-year-olds, but they diverge from Green et al.'s (2000) report of high stability across a 6-month interval in 5- to 7-year-olds.

The level of stability identified herein accords well with meta-analytic findings of moderate stability of attachment (Fraley, 2002; Pinquart et al., 2013; see also Groh et al., 2014). In so doing, these results prove somewhat inconsistent with Pinquart and associates'

(2013) meta-analysis, which indicated that studies such as ours (which focus on attachment after infancy rely on a representational measure of attachment, assess stability within a 2-year period and include a representative community sample) should generate larger stability coefficients. Although impossible to determine at this time, this divergence could be due to the developmental period studied, during which considerable socio-cognitive change takes place involving, for example, moral concepts and theory of mind (see Smetana, 2013). It is possible that as attachment stability increases after the age of 6 (Pinquart et al., 2013; see also Green, 2000), the current findings may represent a time window in development in which considerable change in attachment takes place before IWMs become more consolidated.

Beyond the question of stability of individual differences, and in accordance with Stievenart et al. (2014), repeated measurements of attachment security revealed increasing levels of security, on average, over time. This developmental trend has, to some extent, been indicated by others when studying attachment from infancy to the late preschool years (see Solomon & George, 2008). Correspondingly, increased insecurity has been reported with a risk sample (Vondra et al. (2001).

Despite the normative finding of increased security across time, it should be noted that factors beyond attachment could influence the current results. Even though very young children are capable of generating narratives (Szaflarski et al., 2012), these capacities improve with age (Curenton, 2011). However, MCAST's scoring criteria does not adjust for age, and although narrative abilities are strongly grounded in linguistic skills and general cognitive skills (Szaflarski et al., 2012), only verbal comprehension was controlled in this study. Consequently, we cannot exclude the possibility that the children simply became better at telling secure stories. It should be noted, however, that stories may become more coherent without becoming more secure. Notably, MCAST scoring requires coding not only the children's speech but also how they have the dolls behave (Green et al., 2007).

Changes in Attachment Forecast Changes in Emotion Regulation

Because development is ongoing and because attachment earlier in life is presumed to influence subsequent behavior later in life (Sroufe, 2016), we predicted that whether a child became more secure or insecure over time would forecast whether he or she became more skilled in emotion regulation. Beyond indicating that attachment and emotion regulation continue to develop over the two-year period that each was studied, we found that the level and change in attachment security across the late preschool years predicted the subsequent level and change in capacities for emotion regulation into the early middle childhood years. These findings linking development in a relational arena, attachment, with future development in the capacity to regulate emotions could be of clinical importance, especially given the central role of emotion regulation in most forms of psychopathology (Cole & Deater-Deckard, 2009). Hence, efforts to foster secure attachment relationships should not be restricted to the very first years of life, as increasing security across the transition to school and/or preventing its decline would clearly seem to have prospective benefits.

The focus on security per se seems to be important in light of the results of the secondary analysis. Recall that upon including both security and disorganization in the same model, we found that security rather than disorganization emerged as a significant predictor of emotion regulation. This was true despite the rather strong correlation between the two attachment measures.

Differential Susceptibility and 5-*HTTLPR*

By adopting a GXE approach, we investigated whether the effect of changes in attachment security on changes in emotion regulation would vary as a function of child genotype. This turned out to be the case. Upon becoming more secure, children who were homozygous for the 5-*HTTLPR*-S allele displayed the greatest increase in emotion regulation. Just as importantly, SS carriers evinced less positive growth in emotion regulation—and more

so than other children—when attachment security decreased over time; indeed, for the SS children, the effect of change in attachment security on change in emotion regulation proved rather strong ($\beta=0.63$, $p<0.001$).

This pattern is consistent with the theory that children may vary in terms of their developmental plasticity, and such increased responsiveness to the environment operates in a for-better-and-for-worse manner (Belsky et al., 2007). Notably, the present findings are consistent with meta-analytic evidence indicating that the moderation effects of 5-HTTLPR of diverse environmental factors and processes prove more consistent with differential susceptibility than diathesis stress (van Ijzendoorn et al., 2012; van Ijzendoorn and Bakermans-Kranenburg, 2015) and that emotional reactivity is especially linked to individuals who are homozygous for the S allele (Miller et al., 2013).

In considering the differential-susceptibility-related results emerging from this investigation, it is important to remember that even L carriers were affected by changes in attachment security, just not as much as S homozygotes; these results are consistent with the weak version of differential susceptibility. When considering the limited prevalence of 5-HTTLPR SS homozygotes in the current sample (SS=18.4%), changes in attachment seemed to exert a strong impact on only a minority of the children. For the majority of children, this impact was more limited.

Teacher vs parent reports of emotion regulation yielded somewhat mixed findings with regard to genotype moderation at 6 years of age. **Here, the effect of attachment at 4 years of age** only came out significant with parent reports. Given that previous studies have reported attachment-X-5-HTTLPR effects in preschoolers (Kochanska et al. (2009)) and adolescents (Starr et al., 2013; Zimmermann et al., 2009), our mixed GXE results at 6 years of age may be methodological rather than substantial. Notwithstanding, our results discerned a *main* effect of the SS genotype on emotion regulation at 8 years of age. Thus, we cannot exclude that there

are processes taking place beyond the preschool years, as middle childhood begins, in which the 5-HTTLPR polymorphism comes into play as a *more* potent moderator as well as a predictor. We can only speculate about possible age effects: Although individual differences in brain development related to emotion regulation are far from identified (Johnstone & Walter, 2014), it is, for example, documented that children apply increasingly more cognitive emotion-regulation strategies with age (Perlman & Pelphrey, 2010). This was illustrated with a sample of 5- to 11-year-olds, in which the older children were reported to use the more dorsal “cognitive” areas of the anterior cingulate cortex (a specialized prefrontal region implicated in emotion regulation), whereas the younger children engaged the more ventral “emotional” areas (Perlman & Pelphrey, 2010). Thus, possibly, from 6-8 years we may be tapping in to the shift in prefrontal activation in which the more reactive SS carriers may lag behind their less reactive peers, while at the same time profiting from the more secure strategies in times of distress. Indeed, social experiences throughout the lifespan influence the development of brain areas involved in self-regulation (see Kolb et al., 2012 for a review). However, such development does not seem to be linear or easy to predict (Ahmed, Bittencourt-Hewitt & Sebastian, 2015), hence, age effects could be a complex matter. In addition, age, even ethnicity, may further be involved in regard to which alleles function as susceptibility factors (Davies & Cicchetti, 2014; van Ijzendoorn et al., 2012).

Along with possible biological explanations, children’s contexts extend and become more complex as well. With respect to the increasing external demands in school/early middle childhood, we suspect that SS children struggle to adapt in school, perhaps especially from 7 to 8 years of age, given that the first year in Norwegian schools with 6 year-olds is less demanding and more similar to day care.

What is especially important to appreciate is that no matter how interesting the results of the current investigation prove to be, much remains to be learned about the serotonergic

system (Canli & Lesch, 2007). In fact, readers need to be cautioned that the moderational effect detected herein may not even be a function of 5-*HTTLPR* but rather of some other polymorphisms that are associated with 5-*HTTLPR* variants. Indeed, similar to most GXE work, this work remains correlational in character as it is based on observational data. However, it should be highlighted that recent experimental work has chronicled the moderation effect of 5-*HTTLPR* (Belsky et al., 2013; Belsky & van Ijzendoorn, 2015; van Ijzendoorn & Bakermans-Kranenburg, 2015).

From a clinical perspective, this study indicates that interventions for dysregulated children should include a relational focus, which takes underlying attachment insecurity into account. This requires parent involvement. However, the current results also imply that clinicians should expect attachment interventions to be efficient for some children more than for others. Future replication studies, as well as randomized interventions studies, may further clarify the direct implications of our GXE findings.

Strengths and Limitations

The stringent recruitment procedure, relatively large community sample, **and** longitudinal design are indisputable strengths of this study. Additionally, observational measures of attachment representations across time are rare for large samples; due to the potential bias of self-reported or parent-reported emotion regulation, the use of teacher reporting should also be considered a strength of this investigation (Adrian et al., 2012). Certainly, the reduction of reporter bias is especially important when testing differential-susceptibility, as a self-reported assessment of environment — in this case, parent-child attachment — could involve heritable response biases (van Ijzendoorn & Bakermans-Kranenburg, 2015). Another strength of this inquiry was the competitive evaluation of alternative models of person-X-attachment interactions (i.e., diathesis-stress vs. differential susceptibility).

Like most other research, the work presented herein was not without limitations. Although this study was informed by prior GXE research and studies documenting (modest) associations between parenting and attachment security (Fearon & Belsky, 2016), one should not presume that attachment security is a pure reflection of the rearing environment. Although behavior-genetic studies have consistently indicated that attachment is not heritable in infancy (see Fearon, Shmueli-Goetz, Viding, Fonagy & Plomin, 2014 for a discussion), a recent study documented significant heritability in adolescents (Fearon et al., 2014). Thus, we cannot rule out the possibility that the attachment effects chronicled herein reflected genetic or other organismic influences rather than true environmental ones. In fact, given that environmental measures are more error prone than genetic measurements (van Ijzendoorn & Bakermans-Kranenburg, 2015), it would be especially useful for experimentalists to determine whether the effects documented herein can be more or less replicated when efforts are made, via intervention, to foster attachment security across the transition to school.

Furthermore, the current study was not positioned to examine child effects. Undoubtedly, the effect of attachment development on emotion regulation does not exclude the possibility of the reverse relation; thus, reciprocal analyses of the dynamics between attachment and emotion regulation are warranted (Parrigon et al., 2015). Beyond reciprocity, the lack of complete measurement points of attachment and emotion regulation created limitations with respect to the temporal ordering of change processes. Although we can only assume that changes from 4 to 6 years of age continued to operate from 6 to 8 years of age independently of possible later changes in attachment, theory (Bowlby, 1969) and evidence (Pinquart et al., 2013) suggest that attachment is substantially more stable beyond the ages of 6.

With regard to measurement, it should be noted that the measure of emotion regulation applied herein does not provide insight into the regulation of emotions in real time due to the

use of a questionnaire. Rather, it provides what can be described as a depiction of emotions “as regulated” and in rather general terms. Thus, future work should consider measuring emotion regulation in real time. Lastly, the findings should be interpreted in the context of substantial missing information. However, this attrition was only marginally associated with the study variables, and FIML was applied to adjust for missingness.

Conclusions

Despite these important limitations, the results of our investigation indicate that changes in attachment representations are common and that such changes across the transition to school forecast changes in emotion regulation early in the elementary school years; this is more so the case for a minority of children who are homozygous for the 5-*HTTLPR* short-allele than for other children. Thus, our findings extend the limited literature on attachment and emotion regulation in the “forgotten years” of early middle childhood.

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Table 1

Descriptive statistics and zero-correlations for all study variables

	M / %	S.D.	Min	Max	1	2	3	4	5
1. Attachment security 4 years	0.52	0.39	0.00	1.00	-				
2. Attachment security 6 years	0.67	0.34	0.00	1.00	0.28***	-			
3. Emotion regulation 6 years	3.33	0.47	1.25	4.00	0.10*	0.20***	-		
4. Emotion regulation 8 years	3.42	0.49	1.50	4.00	0.14**	0.24***	0.43***	-	
5. Verbal comprehension 4 years	6.73	1.92	0.8	10.3	0.12*	0.10*	0.13**	0.05	-
6. 5-HTTLPR SS	18.2				0.05	0.06	-0.03	-0.11*	-0.02
7. 5-HTTLPR SL	51.2				0.00	0.00	0.00	0.02	0.01
8. 5-HTTLPR LL	29.6				-0.05	-0.05	0.03	0.06	0.00

Note: * $p < 0.05$, ** $p < 0.01$. *** $p < 0.001$

Table 2

Predictors of emotion regulation at 6 years and from 6 to 8 years of age

	Emotion regulation at 6 years of age				Change in emotion regulation from 6 to 8 years of age			
	<i>B</i>	95% CI	β	<i>p</i>	<i>B</i>	95% CI	β	<i>p</i>
Emotion regulation at 6 years of age	-	-	-	-	-0.31	-0.35; -0.26	-0.58	<0.001
Attachment security at 4 years of age	0.28	0.12; 0.44	0.27	<0.001	0.09	0.02; 0.17	0.23	<0.001
Attachment security-change from 4 to 6 years of age	0.52	0.27; 0.77	0.26	<0.001	0.20	0.08; 0.31	0.19	<0.001
5-HTTLPR SS* carriers	-0.05	-0.12; 0.07	-0.02	0.313	-0.04	-0.09; 0.00	-0.10	0.035
5-HTTLPR SL* carriers	0.03	-0.05; 0.11	0.04	0.389	0.01	-0.03; 0.05	0.02	0.657
Verbal comprehension at 4 years of age	0.06	0.01; 0.05	0.11	0.007	0.00	0.02; 0.09	-0.02	0.626

Note: *In comparison to LL carriers (baseline)

Table 3

Emotion regulation at 6 years and from 6 to 8 years of age according to attachment security at 4 years of age as well as change from 4 to 6 years of age across three 5-HTTLPR genotypes

	Emotion regulation at 6 years of age											
	5-HTTLPR-SS		5-HTTLPR-SL		5-HTTLPR-LL		SS vs. SL		SS vs. LL		SL vs. LL	
	β	p	β	p	β	p	Wald	p	Wald	p	Wald	p
Attachment at 4 years of age	0.31	0.047	0.17	0.052	0.36	<0.001	0.45	0.50	0.25	0.62	2.31	0.13
Attachment change from 4 to 6 years of age	0.24	0.11	0.21	0.014	0.33	<0.001	0.00	0.97	0.58	0.45	1.08	0.30
	Change in emotion regulation from 6 to 8 years of age											
	5-HTTLPR-SS		5-HTTLPR-SL		5-HTTLPR-LL		SS vs. SL		SS vs. LL		SL vs. LL	
	β	p	β	p	β	p	Wald	p	Wald	p	Wald	p
Attachment at 4 years of age	0.51	<0.001	0.14	0.052	0.27	0.01	4.63	0.031	1.18	0.28	0.98	0.32
Attachment change from 4 to 6 years of age	0.63	<0.001	0.06	0.32	0.25	0.006	16.36	<0.001	5.33	0.021	1.00	0.32

Figure 1

The effect of change in attachment on the change in emotion regulation according to 5-HTTLPR

