AFFECT DYSREGULATION
AND
ADOLESCENT PSYCHOPATHOLOGY
IN THE FAMILY CONTEXT

Anna Neumann
The studies reported in chapters 2 and 4 of the present thesis were performed at the Department of Developmental Psychology, VU University Amsterdam. No financial support was received for these studies. The studies reported in chapters 3 and 5 used data of the RADAR study. RADAR has been financially supported by main grants from the Netherlands Organisation for Scientific Research (GB-MAGW 480-03-005, GBMAGW 480-08-006), and Stichting Achmea Slachtoffer en Samenleving (SASS), and various other grants from the Netherlands Organisation for Scientific Research, the VU University Amsterdam and Utrecht University. The study reported in chapter 6 used data of the Edinburgh Study of Youth Transitions and Crime (ESYTC) and the analyses reported here were conducted in collaboration with Prof. Dr. Barbara Maughan. The ESYTC was funded by the Economic and Social Research Council, the Nuffield Foundation and the Scottish Executive Committee. Dr Maughan is supported by the Medical Research Council, United Kingdom. The analyses described in chapter 6 were supported by a grant from the Medical Research Council (MRC G0500953).

ISBN: 9789086595051

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Cover design: Anna Neumann. The photographs show parts of a painting by Zakol.

Printed by: Ipskamp, Enschede
VRIJE UNIVERSITEIT

AFFECT DYSREGULATION AND ADOLESCENT PSYCHOPATHOLOGY IN THE FAMILY CONTEXT

ACADEMISCH PROEFSCHRIFT

ter verkrijging van de graad Doctor aan
de Vrije Universiteit Amsterdam,
op gezag van de rector magnificus
prof.dr. L.M. Bouter,
in het openbaar te verdedigen
ten overstaan van de promotiecommissie
van de faculteit der Psychologie en Pedagogiek
op woensdag 24 november 2010 om 11.45 uur
in de aula van de universiteit,
De Boelelaan 1105

door
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geboren te Keulen, Duitsland
promotor: prof. dr. J.M. Koot

copromotor: prof. dr. P.A.C. van Lier
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Adolescent Affect Dysregulation
- General Introduction
Affective dysregulation is considered a central facet of most forms of psychopathology (e.g., Berenbaum, Raghavan, Le, Vernon, & Gomez, 2003; Bradley, 2000; Cicchetti, Ackerman, & Izard, 1995; Cole & Deater-Deckard, 2009; Gratz & Roemer, 2004; Keenan, 2000), including internalizing problems, such as anxiety (Mennin, Heimberg, Turk, & Fresco, 2002) and mood disorders (Gotlib, Joormann, Minor, & Cooney, 2006), as well as externalizing problems, such as conduct disorder (Beauchaine, Gatzke-Kopp, & Mead, 2007). The central position of affect dysregulation in the conceptualization of psychopathology manifests itself in current criteria for the diagnosis of mental disorders, as depicted in the *Diagnostic and Statistical Manual of Mental Disorders –Fourth Edition* (DSM-IV-TR; American Psychiatric Association [APA], 2000). For instance, criteria for depression include prolonged dysphoria and/or irritability, as well as mood lability, while criteria for anxiety disorders are persistent fear or excessive worry, and an inability to regulate these emotions, and high anger and irritability, a lack of guilt and labile mood are criteria for disruptive behavior disorders.

In turn, apart from being reflections of neurological activity, affective experiences are highly social phenomena, and are regarded as both sources and consequences of social interactions (Bell & Calkins, 2000; Zajonc, 1998). What is more, in addition to social interactions as inducers of affective experiences, many agree that one learns how to regulate affect in interactions within close social relationships, particularly the parent-child relationship (Fox & Calkins, 2003; Kopp, 1989; Morris, Silk, Steinberg, Myers, & Robinson, 2007; Zeman, Cassano, Perry-Parrish, & Stegall, 2006). Style and effectiveness of affect regulation in turn, also shape the nature of social relationships, resulting, for example in undesirable parent-child interactions, that may add to the risk for onset or aggravation of symptoms of psychopathology. Thus, considering the social context of affective experiences seems quintessential to our understanding of their relation with psychopathology. Specifically, the social context may influence the way affect is regulated, and, in turn seems to be influenced by affective regulation, and affective dysregulation.

Adolescence constitutes an important developmental period for the study of associations between affective dysregulation and psychopathology, and the role of the parent-child relationship for several reasons. Main developmental tasks of adolescents are achieving autonomy from parents, acquiring a deeper sense of identity and building intimate relationships with others. In order to do so, adolescents must increasingly rely on themselves for regulating emotions and behavioral impulses. That this at times is hard, especially in adolescence, is shown by heightened negative emotions (Larson & Lampman-Petraitidis, 1989) and heightened variability of emotions (Larson, Csikszentmihalyi, & Graef, 1980) in adolescence as compared to childhood and adulthood, adolescent rises in prevalence rates of internalizing (e.g., anxiety, depression) and externalizing problems (aggressive and rule-breaking behavior) (e.g., Silk, Steinberg, & Morris, 2003), and at least temporal decreases in the quality of the adolescent-parent relationship (Furman & Buhrmeester, 1992). For example, adolescent strivings for autonomy may lead to increased or intensified conflict with parents (Laursen, Coy, & Collins, 1998), as the adolescent may feel restricted in these strivings by parental efforts to remain in control of the adolescent’s behavior. At
the same time, feelings of warmth, trust and support tend to decrease in the relationship between parents and adolescents (e.g., de Goede, Branje, & Meeus, 2009).

Despite the fact that adolescence presents a time of important changes in affective experience and regulation, of increasing psychopathology, and pervasive changes in social relationships, compared to research on affect in children and adults, research on affective development, and its sources and consequences during adolescence is still relatively scarce. The current thesis aims to address affective dysregulation, and its role in the development of psychopathology in early-mid adolescence in the context of the parent-adolescent relationship.

Several topics will be discussed in this introduction. First, the focus will be on associations between affective dysregulation and psychopathology, in general. In this section, I will also discuss different conceptualizations and operationalizations of affect dysregulation, and introduce the ones employed in the present thesis. Then, I turn to the question whether the link between emotion dynamics and psychopathology is specific (dynamics of discrete emotions linked to specific types of psychopathology) or general (general emotion dynamics linked to various types of psychopathology). Next, I will discuss affective dysregulation in the context of the parent-adolescent relationship, before I put links between the parent-adolescent relationship and psychopathology in the spotlight. Subsequently, I will highlight ways in which affective dysregulation and parent-adolescent interactions may jointly influence adolescent development, and how the broader environmental context may play a role. Finally, an outline of the studies that form the empirical part of this thesis, will be presented.

*Affect Dysregulation and Psychopathology*

Affect arises in response to events that are appraised as meaningful to the individual (Frijda, 1988). Whether an event elicits positive or negative affect depends on how the event relates to an individual’s goals. An event that leads (closer) to the achievement of one’s goal will induce positive affect, while an event that endangers or hinders goal achievement will lead to negative affect (Carver, 2004; Frijda, 1988). In accordance with the functional approach to emotions (e.g., Thompson, 1994), both positive and negative affect is regarded as serving a regulatory function in that it organizes behavior (e.g., Cole & Deater-Deckard, 2009; Gratz & Roemer, 2004; Stams, Juffer, & van IJzendoorn, 2002). Affect helps the individual to appraise and deal with daily events (Cole & Hall, 2008). At the same time, affect is regarded as inherently (to be) regulated. That is, humans are generally able to regulate both their internal affective states and their associated behavioral reactions (Cole & Hall, 2008) in the service of long-term goals (e.g., the intensity of negative affect in response to an insult can be down-regulated, in order not to act aggressively if a long-term goal is the maintenance of a positive long-term social relationship with the person who insulted). Leaving from the broad definition of *affect regulation* as the modulation of emotional responses (i.e., changing the intensity, duration or valence of an affective response), *affect dysregulation* refers to maladaptive patterns of affect regulation, that is, to patterns of affect
regulation that are costly in the pursuit of long-term goals, for instance because they undermine the maintenance of positive social relationships, and personal well-being (Cole & Hall, 2008).

Theoretically, affect dysregulation and psychopathology are thought to be linked through the subjective experience of stress that activates regulatory responses (e.g., Bradley, 2000). If the regulatory responses are unsuccessful, the experience of stress is maintained or even exacerbated. Under high levels of stress, the individual may not be able to learn adaptive regulatory behaviors, and may instead engage in dysfunctional behaviors to alleviate distress. Combined, the subjective experience of distress and dysfunctional behaviors may result in symptoms of mental pathology, or, if severe or continued in disorders. Consequently, psychopathology is regarded from a dynamic regulatory perspective, stressing the function of psychopathologic symptoms as (maladaptive) coping strategies (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996) one engages in to deal with one’s affective experiences. In addition to negative affect experienced during stress, positive affect may have to be regulated, too. Positive affect is associated with feelings of interest, energy, and confidence, feelings which are clearly important for continued efforts to achieve a goal (e.g., Watson & Clark, 1997). Consequently, if one manages to upregulate the experience of positive affect, even in the face of adversity, one is better able to deal with obstacles. It should be mentioned, that sometimes positive affect may have to be down-regulated as well, for instance when infatuation keeps interfering with work one needs to concentrate on.

Recently, research on affect dysregulation and its association with psychopathology has increased rapidly (see for instance Southam-Gerow & Kendall, 2002), and some of these studies have focused on adolescence. Consequently, diverse indices of affect dysregulation have been linked to psychopathology in adolescents. Cross-sectional studies have linked high levels and variability of negative emotions to symptoms of depression (Larson, Raffaelli, Richards, Ham, & Jewell, 1990; Silk et al., 2003), and externalizing problems in adolescents (Silk et al., 2003). Additionally, depressive symptoms in adolescents’ are associated cross-sectionally with maladaptive affect regulation strategies (Yap, Allen, & Ladouceur, 2008). Nevertheless, compared to studies with children, research on affect dysregulation in adolescence remains comparatively sparse (e.g., Morris et al., 2007; Zeman et al., 2006). One likely reason for this relative lack of research on affect dysregulation in adolescents (despite its clear clinical significance) may be the limited number of available measures of affect dysregulation for adolescents (Zeman et al., 2006).

Affect is influenced by, and influences several domains of functioning, including biological (e.g., the functioning of the autonomic nervous system), cognitive, and behavioral processes (Cole, Martin, & Dennis, 2004; Thompson, Lewis, & Calkins, 2008). Consequently, affect regulation and dysregulation may also be apparent at different levels of experience and cognition, including the dynamics of affect (its intensity and variability; Silk et al., 2003), affect regulation strategies (e.g., distraction, cognitive reinterpretation; Gross & Thompson, 2007), and meta-affective experiences (e.g., nonacceptance of affective responses; Gratz & Roemer, 2004). Some go as far to maintain that the dearth of research on the development of affect regulation may in part be due to the equivocality of affect regulation and dysregulation as a concept (Cole et al., 2004).
ADOLESCENT AFFECT DYSREGULATION: INTRODUCTION

Acknowledging its multifaceted nature, the present thesis focuses on three dimensions of affect dysregulation: (1) affect dynamics representing the direct affective experience, (2) meta-affect experiences, representing the intersection of affect and cognition, and (3) impulsivity, acknowledging the close links between action and behavior (Carver, 2004), each of which have been related to psychopathology. Regarding affect dynamics, dysregulation may be reflected in heightened and prolonged negative affect and heightened variability of negative as well as positive affect. Affect dynamics are important, as they lie at the core of the affective experience, serving as inputs for regulatory attempts, and reflecting their outcome at the same time. At the meta-cognitive level, affect dysregulation may be reflected by perceived difficulties regarding several of its aspects including the clarity of affective experiences, the non-acceptance of affective responses, and low affect-regulation self-efficacy, each of which may indicate increased risk for psychopathology (Bradley, 2000). Impulsivity, the “predisposition toward rapid, unplanned reactions to internal or external stimuli […]” (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001, p. 1784) can be regarded as a failure to regulate the behavioral response ‘given in’ by the action-readiness component of affect, and has been associated especially with externalizing problems such as aggression and delinquency (e.g., Loeber, 1990), but also with internalizing problems (Carver, Johnson, and Joormann, 2008). This study will explore the relation between each of these dimensions of affect dysregulation with adolescent psychopathology.

Affect Dysregulation and Psychopathology: General or Specific Associations?

As evident in conceptualizations of mental disorders as described in the DSM-IV, it is generally accepted that individuals with diverse forms of psychopathology evidence heightened levels of negative affect. However, what is needed now, is greater specificity of links between patterns of affective dysregulation and distinct forms of psychopathology (Cole & Deater-Deckard, 2009). On the level of emotion dynamics, ‘functional continuity’ between discrete emotions (e.g., sadness, anger) and specific forms of psychopathology (e.g., depression, conduct problems) is often assumed (e.g., Cole & Hall, 2008; Malatesta & Wilson, 1988; Muris & Ollendick, 2005; Rothbart & Bates, 2006). That is, patterns of affective responding that have become consolidated over time are thought to lead to specific forms of psychopathology (Zahn-Waxler, Klimes-Dougan, & Slattery, 2000). Accordingly, individuals who are often fearful are considered to be more likely to develop anxiety-related than other psychological disorders, while individuals who are easily angered are more likely to display symptoms of disruptive behavior disorders (e.g., Muris & Ollendick, 2005). However, empirical evidence for specific links between affect dynamics and forms of psychopathology in adolescence is rather mixed and may heavily depend on the method used to assess emotions (e.g., observations [Keltner, Moffitt, & Stouthamer-Loeber, 1995] versus self-report [Larson et al., 1990; Silk et al., 2003]).

Indeed, a non-specific emotion dysregulation factor, affecting dysregulation in all discrete emotions alike has been proposed as a correlate of internalizing and externalizing psychopathology (Silk et al., 2003). It might well be that the dynamics of discrete emotions are associated with
psychopathology in non-specific ways, while affect dysregulation at the meta-cognitive level, as
described above, relates more specifically to diverse forms of psychopathology. For instance, one
might expect that the non-acceptance of affective responses (in general, not just non-acceptance of a
specific discrete emotion) relates more strongly to depression than to aggressive behavior, while a
lack of emotional awareness associates specifically with delinquent behavior (Herpertz, Werth,
Lukas, Qunaibi, Schuerkens, Kunert et al., 2001).

The present thesis will study the specificity of associations between the dynamics of discrete
emotions and psychopathology, as well as between different dimensions of affect dysregulation at
the meta-cognitive level and diverse forms of psychopathology.

**Affect Dysregulation in the Context of the Parent-Adolescent Relationship**

Regardless of whether in general or specific ways, it is clear that affect dysregulation is a
clinically significant construct (e.g., Gratz & Roemer, 2004). Therefore, a vital question regards its
origins. While clearly in part genetically influenced (e.g., Hariri & Forbes, 2007), many researchers
agree that affect regulation and dysregulation are closely linked to, or originate from, suboptimal
parent-child relationships (e.g., Kopp, 1989; Morris et al., 2007; Zeman et al., 2006), and affect
dysregulation is indeed often observed within problematic family environments (Morris et al.,
2007). The relationship context is important for affective dysregulation for two main reasons: first,
many of the most intense affective experiences are evoked during social interactions, especially
with interaction partners that one feels close to such as family members (Bowlby, 1980), and
secondly, because one also learns strategies for dealing with affective experiences in close social
relationships, primarily in the parent-child relationship (Kopp, 1989; Morris et al., 2007). In
addition, given the special nature of the parent-child relationship, both parenting behaviors and the
quality of the parent-child relationship resulting from repeated parent-child interactions may impact
adolescents’ affective experiences and regulation. Below, I will first discuss associations between
relationship quality and affective experiences and affect (dys)regulation, and then turn to links
between parenting, affective experiences and affect (dys)regulation.

Regarding close social relationships as sources of affective experiences, empirical evidence
shows consistently that positive affect is associated with increases in belonging (e.g., reconciliation
after a fight), while negative affect is linked with decreases in belonging (e.g., when having an
altercation with or loosing someone dear) (Baumeister & Leary, 1995). In adolescence specifically,
intense affect is experienced within the context of parent-child interactions (Collins & Laursen,
2006). Thus, regarding affect and its (dys)regulation, both warmth, support, and acceptance on the
one hand, and conflict on the other hand, are important aspects of close social relationships. The
experience of a warm, accepting and supportive relationship is associated with the experience of
positive affect (Baumeister & Leary, 1995) and a sense of emotional security (Bowlby, 1969;
Cummings & Davies, 1996). Felt security reduces wariness giving space for unrestricted
exploration and consequent competence development (Bretherton, 1985). Moreover, warm and
trusting relationships in which one feels emotionally secure, offer good opportunities for learning
ADOLESCENT AFFECT DYSREGULATION: INTRODUCTION

how to deal with emotions, for instance via modeling, discussion of emotional experiences, or direct instruction (e.g., Morris et al., 2007). Conversely, relationships characterized by high levels of conflict lead to the experience of elevated levels of negative affect, such as anger, sadness, or anxiety. Highly conflictual interactions between adolescents and parents also likely preclude the learning of adaptive strategies for the regulation of affect, for instance because negative affect continues to rise and the conflict escalates, thereby blocking the exploration of alternative ways for regulating the interaction and associated emotions. Given the inherently dyadic nature of the parent-child relationship, it should be mentioned that relationship quality and adolescent affect dysregulation are likely linked bidirectionally: High levels of negative affect and high affective reactivity in the adolescent may cause irritation and negative affect in the parent, leading to a less positive evaluation of the relationship by both parent and child.

In addition to dyadic relationship aspects, such as warmth and conflict, parenting behaviors may also impact adolescent affective experiences and regulation. Given that a developmental task of adolescence is the achievement of autonomy, an important parenting construct in adolescence is parental control, in the form of behavioral as well as psychological control (Barber, 1996; Barber & Harmon, 2002; Silk, Morris, Kayana, & Steinberg, 2003). Behavioral control is aimed at controlling the adolescent’s actions (e.g., when and how to clean his room) while psychological control is aimed at controlling the adolescent’s inner world, that is at what he is feeling and thinking. Associations between behavioral control and affect dysregulation may be u-shaped, with optimal levels providing a safe environment in which one can deal with ones affect. Effects of parental control may also depend on child characteristics: highly impulsive adolescents may profit from higher levels of parental control than their less impulsive peers. Parental psychological control likely interferes with adolescent affect regulation by precluding possibilities for the child to freely express emotions and to learn to be accepting of emotional responses. Again, links between adolescent affect dysregulation and parenting are likely bidirectional. For instance, adolescents who are highly impulsive and emotionally reactive may be harder to control than their less impulsive peers.

Taken together, it is clear that the quality of parent-child interactions, be it in the relationship or the parenting context, may both support a child’s affect regulation competencies, or place it at risk for the development of maladaptive patterns of affect regulation (Cole & Deater-Deckard, 2009). The present thesis investigated in how far the quality of parent-adolescent relations and parenting still provide an important context for affect regulation and dysregulation in adolescence.

Parent-Adolescent Relationship and Adolescent Psychopathology

Diverse family factors have been associated repeatedly with child/adolescent psychopathology, including attachment, parenting styles, and family relationship quality (see for instance Cummings, Davies, & Campbell, 2000). In spite of the large number of family factors linked to child/adolescent outcomes, several questions remain. In particular, the directionality of
effects is not entirely clear: although cross-sectional associations have often been replicated, longitudinal links seem less robust (for reviews, see Bögels & Brechman-Toussaint, 2006, for child anxiety; Rothbaum & Weisz, 2004 or Wamboldt & Wamboldt, 2000, for child externalizing problems). Empirical evidence has been found for parent-driven effects (Burt, McGue, Iacono, & Krueger, 2006; Rueter, Scaramella, Wallace, & Conger, 1999; Williams, Conger, & Blozis, 2007, child-driven effects (Anderson, Lytton, & Romney, 1986), and bidirectional associations (Burt, McGue, Krueger, & Iacono, 2005; Richmond & Stocker, 2008). However, some studies also report only cross-sectional, but no longitudinal associations (Vuchinich, Bank, & Patterson, 1992). One possible explanation for the mixed results is that parent-adolescent relationships might not influence adolescent mental health directly, but indirectly, for instance via adolescent emotional processes. In addition, the influence of parent-adolescent relationship quality on adolescent development, may interact with child- (e.g., affect dysregulation) and contextual (family, school, neighborhood factors). Each of these possibilities is discussed in more detail below.

*Parent-Adolescent Relationship, Adolescent Affective Dysregulation and Psychopathology*

Both contextual and individual risk factors are thought to add to the development of psychopathology, at least in part, through their impact on an individual’s self-regulation (Cole & Deater-Deckard, 2009). As affective dysregulation is regarded as a core component of most forms of psychopathology, and affective dysregulation is thought to be impacted by adverse social experiences, it has repeatedly been hypothesized as one possible mechanism in links between family factors and psychopathology (Beauchaine et al., 2007; Cole, Michel, & Teti, 1994; Cummings, Schermerhorn, Davies, Goeke-Morey, & Cummings, 2006; Maughan & Cicchetti, 2002; Morris et al., 2007; Yap, Allen, & Sheeber, 2007), and between the parent-adolescent relationship and adolescent psychopathology in particular.

Research has provided some support for the mediating role of emotion dysregulation in links between social experiences and mental health problems in children. For instance, Maughan and Cicchetti (2002) identified child emotion regulation abilities as a mediating variable in links between maltreatment and socioemotional adjustment in a sample of 4-6 year-olds. Also, the effects of parental warmth on child externalizing problems have been shown to be partly indirect through child emotion dysregulation (Eisenberg, Losoya, Fabes, Guthrie, Reiser, Murphy et al., 2001).

At the same time, positive parent-adolescent interactions may be conceptualized as a protective factor against child affective characteristics that may otherwise predispose them to internalizing or externalizing problems, such as heightened affective reactivity and high impulsivity. For instance, conceivably, adequate levels of behavioral control may attenuate the association between high impulsivity and externalizing problems.

In sum, interactions in the parent-adolescent relationship and adolescent affective dysregulation may be associated with adolescent psychopathology in additive, mediating, or
moderating ways. The present thesis explored several ways in which indices of affect dysregulation and parent-adolescent interactions combine to influence adolescent development.

The Broader Social Context

As emphasized by a number of researchers (e.g., Sameroff, 2010), and most notably in Bronfenbrenner’s bioecological model (e.g., Bronfenbrenner, 1986) the development of youths and their families does not take place in a vacuum, but is impacted by the broader social context as well. Factors indexing family adversity, such as single parenthood and low socioeconomic status have consistently been associated with the development of psychopathology in children and adolescents (e.g., Amato, 2000; Loeber & Stouthamer-Loeber, 1986). For adolescents, who increasingly spend time outside the home and the direct supervision of their parents, neighborhood risk may gain in importance for socio-emotional development (Leventhal & Brooks-Gunn, 2000). Family and neighborhood level risk may impact youth development directly, as well as indirectly, for instance through decreasing the quality of family relationships and parenting (e.g., Kohen, Leventhal, Dahinten, & McIntosh, 2008; Mrug & Windle, 2009). In addition, theoretical formulations, such as the differential susceptibility hypothesis (Belsky & Pluess, 2009; Pluess & Belsky, 2010) highlight that the impact of environmental adversity on development is not the same for everyone, but depends on individual difference characteristics, such as temperament and emotionality. For instance, the association between living in disadvantaged neighborhoods and antisocial behavior, has been shown to be moderated by levels of youth impulsivity (Lynam, Caspi, Moffitt, Wikström, Loeber, & Novak, 2000; Meier, Slutske, Arndt, & Cadoret, 2008; though not all studies have found such interactive effects e.g., Vazsonyi, Cleveland, & Wiebe, 2006).

The Present Thesis

The existing literature provides substantial evidence that affective dysregulation is associated with most, if not all, forms of psychopathology. Additionally, there is evidence that the development of affect regulation and dysregulation is closely linked to experiences in the parent-child relationship, at least for younger children. Consequently, affect dysregulation has often been hypothesized as an explanatory mechanism in associations between the parent-child relationship and child internalizing and externalizing problems, and there is some evidence for this hypothesis, at least for children. Finally, it is clear that individual risk factors, such as affective dysregulation, play a role in individual development in concert with contextual risk factors, such as family and neighborhood adversity. However, especially for adolescence, longitudinal studies of the relation between affective dysregulation and psychopathology are sorely needed to shed light on the precise nature of this association. Do affective dysregulation and symptoms of psychopathology simply co-occur, or does affective dysregulation actually underlie the development and persistence of internalizing and externalizing problems? Regarding the link between affect dysregulation and psychopathology, it is also not clear whether dysregulation in specific emotions, and/or specific
forms of dysregulation, underlies specific forms of psychopathology, or whether affective dysregulation is better described as a general vulnerability factor for the development of psychosocial problems. Further, empirical evidence for links between the parent-adolescent relationship and adolescent affective dysregulation is scarce, and longitudinal studies on these links are missing. Finally, while it is well known that individual and contextual risk interact in the prediction of individual development, the precise nature of the interplay of individual characteristics with contextual (family and neighborhood) adversity remains poorly understood. However, it is important to get a better grip on the role of adolescent affective dysregulation in adolescent psychopathology, and its potential origins, since self-regulation of affect is a factor that is modifiable by therapeutic efforts (Bradley, 2000; Thompson, Lewis, & Calkins, 2008) and, successful therapeutic effects are likely to show their effects in many fields of psychosocial well-being and functioning (all social relationships, school, work).

Goals of the Present Thesis

The main goal of the present thesis is to study affect dysregulation in adolescence, as related to the development of internalizing and externalizing problems, and address the role of the parent-adolescent relationship and broader contextual factors in this relation. Given the paucity of research on the origins and consequences of affect dysregulation in adolescence, specific aims are to:

1. Study links between adolescent affect dysregulation, in the rather ‘basic’ form of emotional dynamics (level and variability of experienced emotions), and the metacognitive level of self-perceived emotion regulation difficulties, as an underlying personal risk factor for the development of psychopathology from early to mid adolescence. Special attention will be paid to generality versus specificity of links between different forms of affect dysregulation and forms of psychopathology.

2. Study the potential role of parents for adolescent affective dysregulation and adolescent psychopathology: Specific questions will be:
   a. Is adolescent affective dysregulation related to the quality of the parent-adolescent relationship?
   b. Does parent-adolescent relationship quality affect the development of internalizing and externalizing problems directly or indirectly through its effects on adolescents’ affect regulation?

3. Study the role of the broader social context in links between affective dysregulation, the parent-adolescent relationship and the development of psychopathology. Attention will be paid to family (e.g., single parent) as well as neighborhood (e.g., economic disadvantage) adversity.
Parent-adolescent relationship
- Conflict
- Support
- Parenting (control, monitoring)

Affective dysregulation
- High levels of negative affect
- Low levels of positive affect
- High emotional variability
- High self-perceived emotion regulation difficulties
- High impulsivity

Psychopathology
- Internalizing
  - Anxiety
  - Depression
- Externalizing
  - Aggressive Behavior
  - Delinquent Behavior
  - Antisocial Behavior

Adolescent gender

Family Adversity
- Single versus two-parent
- Socioeconomic status

Neighborhood Adversity
- Economic deprivation
- Low informal social control

Figure 1.1. General model of the impact of social and emotional difficulties on adolescent psychopathology.
CHAPTER 1

Design of the Study

Five studies, using four different samples, were performed to explore the aims outlined above. Studies 1 and 2 focus on the first specific aim of the present thesis; that is the establishment of links between different forms of affect dysregulation and psychopathology. Study 3 addresses the second goal, the question of links between the parent-adolescent relationship quality and parental control and adolescent affect dysregulation. Studies 4 and 5 focus on the interplay of affect dysregulation, and the parent-adolescent relationship as related to the development of psychopathology. Additionally, study 5 takes the broader family and neighborhood context into consideration. Study 1 uses a cross-sectional sample of 870 Dutch adolescents aged 11-17 years. Study 2 employs data from another sample of 452 Dutch adolescents followed longitudinally from age 13 to age 14 years. Study 3 makes use of a cross-sectional sample of 463 German 11-19 year olds. Study 4 uses the same sample of Dutch adolescents as is used in study 2; however, adolescents are now followed from age 13 to age 15, and data from the adolescents’ mothers and fathers are included as well. Finally, study 5 uses a sample of 4,597 Scottish adolescents from Edinburgh, followed from age 12 to age 15. An overview of the samples and measures used in each study is provided in Table 1.1.

Outline of the Present Thesis

The first two chapters of the present thesis aim at establishing links between emotion dysregulation and adolescent psychopathology in adolescence. Chapter 1 focuses on the assessment of perceived emotion regulation difficulties in male and female adolescents, and how they relate to self-reported internalizing (anxiety, depression) and externalizing (aggressive behavior, delinquent behavior) problems. Chapter 2 explores adolescent emotion dynamics (level and variability of happiness, anger, anxiety, and sadness) in the 1-year continuity of adolescent anxiety disorders symptoms, depression, and aggressive behavior. Chapter 4 explores links between emotion regulation difficulties and the parent-adolescent relationship. Having established associations between emotion dysregulation and adolescent psychopathology in chapters 2 and 3, and between the parent-adolescent relationship and emotion dysregulation in chapter 4, chapter 5 addresses the potential indirect role of emotion dysregulation in hypothesized links between the parent-adolescent relationship and adolescent psychopathology (generalized anxiety disorder symptoms, physical aggression). Finally, chapter 6 aims at illustrating the complex interplay of personal and family risk and the broader social context in the development of adolescent antisocial behavior.
### Table 1.1

*Overview of Study Designs, Samples, and Measures used in Chapters 2-6*

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<th>Study design</th>
<th>N</th>
<th>Ages</th>
<th>Outcome</th>
<th>Social relationship/context measures</th>
<th>Affect dysregulation measures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cross-sectional</td>
<td>870</td>
<td>11 - 17</td>
<td>Anxiety disorder symptoms</td>
<td>--</td>
<td>Perceived emotion regulation difficulties</td>
</tr>
<tr>
<td></td>
<td></td>
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<td>Depressive symptoms</td>
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<td></td>
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<td>Aggressive behavior</td>
<td></td>
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<td></td>
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<td></td>
<td>Delinquent behavior</td>
<td></td>
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<tr>
<td>Longitudinal</td>
<td>452</td>
<td>13 - 14</td>
<td>Anxiety disorder symptoms</td>
<td>--</td>
<td>Emotion dynamics</td>
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<tr>
<td></td>
<td></td>
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<td>Depressive symptoms</td>
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<td></td>
<td></td>
<td></td>
<td>Aggressive behavior</td>
<td></td>
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</tr>
<tr>
<td>Cross-sectional</td>
<td>463 (177 + 286)</td>
<td>11 - 19</td>
<td>Perceived emotion regulation difficulties</td>
<td>Maternal behavioral control</td>
<td>Perceived emotion regulation difficulties</td>
</tr>
<tr>
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<td></td>
<td>Maternal psychological control</td>
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<td>Adolescent-mother negative interactions and support</td>
<td></td>
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<td></td>
<td>Adolescents' behavioral control</td>
<td></td>
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<tr>
<td>Longitudinal; multi-informant</td>
<td>452</td>
<td>13 - 15</td>
<td>Generalized anxiety disorders symptoms</td>
<td>Adolescent-mother negative interactions and support</td>
<td>Emotion dynamics</td>
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<td></td>
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<td>Physical aggression</td>
<td></td>
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<td></td>
<td>Adolescents' behavioral control</td>
<td></td>
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<tr>
<td>Longitudinal; multi-informant</td>
<td>4,597</td>
<td>12 - 15</td>
<td>Antisocial behavior</td>
<td>Neighborhood low informal social control and economic disadvantage</td>
<td>Impulsivity</td>
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<td>Family type</td>
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<td>Parental knowledge</td>
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</tr>
</tbody>
</table>
Multidimensional Assessment of Emotion Regulation Difficulties in Adolescents Using the Difficulties in Emotion Regulation Scale

Anna Neumann
Pol A. C. van Lier
Kim L. Gratz
Hans M. Koot

(2010) *Assessment, 17*, 138-149
Recent research has led to an increased interest in the role of emotional processes in normal and atypical development (Southam-Gerow & Kendall, 2001), with the regulation and dysregulation of emotions being a primary focus of this research. Indeed, emotion regulation (ER) difficulties have been implicated in several forms of developmental psychopathology (e.g., Bradley, 2000; Cole, Michel, & Teti, 1994; Gross, 1998). Furthermore, ER skills have been positively linked to both prosocial behavior (e.g., Shields, Cicchetti, & Ryan, 1994) and resiliency to multiple risks (Lengua, 2002) among children.

In contrast to the growing body of literature on ER among children and adults, few studies have investigated ER and ER difficulties in adolescents (Gross, 1998; Zeman, Cassano, Perry-Parrish, & Stegall, 2006). However, emerging evidence for the central role of ER processes in adolescent development (e.g., Garnefski, Kraaij, & van Etten, 2005; Silk, Steinberg, & Morris, 2003) highlights the importance of examining ER among adolescents. One likely reason for the relative lack of research in this area (despite its clear clinical significance) may be the limited number of available measures of ER for adolescents (Zeman et al., 2006). Thus, the primary goal of the present study was to extend the extant research on adolescent ER difficulties by exploring the factor structure and psychometric properties of an existing adult measure of ER difficulties among adolescents.

One promising measure for the comprehensive assessment of ER difficulties is the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). Originally developed for use with adults, the DERS was designed to provide a comprehensive assessment of clinically-relevant ER difficulties across multiple domains. Moreover, suggesting its potential utility for adolescents, the DERS is based on a conceptual definition of ER influenced most directly by theoretical literature on ER in youth (Cole et al., 1994; Thompson, 1994). Whereas much of the literature on ER in adulthood emphasizes the control and reduction of negative emotions, the childhood literature emphasizes the functionality of emotions and the problems associated with deficits in the capacity to experience the full range of emotions, with some developmental researchers defining ER as “…the extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions to accomplish one’s goals” (Thompson, 1994, pp. 27-28). As such, the DERS is based on a conceptualization of ER as adaptive ways of responding to emotions, including accepting responses, the ability to experience and differentiate the full range of emotions, and the control of behaviors in the face of emotional distress (see Gratz & Roemer, 2004). Further, items of the DERS focus mainly on the regulation of negative emotional states, as difficulties in this domain are considered to have particular clinical relevance.

In support of the utility of the DERS among adults, scores on this measure have been found to have good test-retest reliability over a period of 4 to 8 weeks in a sample of college students ($\rho_{tt} = .88$; Gratz & Roemer, 2004) and high internal consistency within clinical (e.g., Gratz, Tull, Baruch, Bornovalova, & Lejuez, 2008; Fox, Axelrod, Paliwal, Sleeper, & Sinha, 2007) and nonclinical populations (e.g., Gratz & Roemer, 2004; Johnson, Zvolensky, Marshall, Gonzalez, Abrams, & Vujanovic, 2008). Further, research using this measure with adults has repeatedly linked the DERS to clinically-relevant phenomena in both clinical and nonclinical samples. Specifically, scores on
the DERS showed statistically significant relations with behaviors thought to serve an emotion-regulating function, including deliberate self-harm (Gratz & Roemer, 2008), intimate partner abuse perpetration among men (Gratz, Paulson, Jakupcak, & Tull, 2009), and cocaine-dependence (Fox et al., 2007). Further, scores on the DERS have been found to be heightened among individuals with psychiatric disorders thought to be characterized by ER difficulties, including borderline personality disorder (BPD; vs. non-PD outpatients; Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2006), probable PTSD (vs. trauma-exposed individuals without PTSD; Tull, Barrett, McMillan, & Roemer, 2007), and panic attacks (vs. non-panickers; Tull & Roemer, 2007). Finally, the DERS demonstrates statistically significant associations with a number of constructs thought to be related to ER difficulties, including positive associations with negative affect (Johnson et al., 2008; Vujanovic, Zvolensky, & Bernstein, 2008), depression and anxiety symptom severity (Roemer, Lee, Salters-Pedneault, Erisman, Orsillo, & Mennin, 2009; Vujanovic et al., 2008), anxiety sensitivity (Johnson et al., 2008; Vujanovic et al., 2008), and experiential avoidance (Gratz & Roemer, 2004; Tull & Gratz, 2008; Tull & Roemer, 2007), and negative associations with emotional expression and processing (Johnson et al., 2008), mindfulness (Baer, Smith, Hopkins, Krietemeyer, & Toney, 2006; Roemer et al., 2009), and self-compassion (Roemer et al., 2009).

Further, studies provide support for the utility of the DERS subscales, finding that particular subscales are differentially associated with specific forms of psychopathology. Salters-Pedneault et al. (Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006) found that all DERS subscales (with the exception of lack of emotional awareness) were significantly elevated among individuals with (vs. without) probable generalized anxiety disorder when controlling for negative affect. In contrast, only the specific subscales of difficulties controlling impulsive behaviors when distressed, limited access to effective ER strategies, and lack of emotional clarity have been found to differentiate between trauma-exposed individuals with and without probable post traumatic stress disorder (PTSD) when controlling for negative affect (Tull et al., 2007). As such, research has consistently linked the DERS and its subscales to a variety of forms of psychopathology in adults.

Research on Emotion Regulation Difficulties in Adolescence

Although relatively understudied (in comparison to ER in children and adults), research on ER in adolescents provides preliminary evidence for the importance of specific aspects of ER and related constructs to adolescent development. Some evidence comes from research in (trait) emotional intelligence, which focuses on understanding others’ emotions in addition to one’s own, and on perceived competencies (instead of perceived difficulties, as in the present study; Petrides, Fredrickson, & Furnham, 2004). This research provides evidence that perceptions of how one deals with emotions are associated with academic performance and deviant behavior (Petrides et al., 2004), and self-esteem, anxiety, and depression (Fernandez-Berrocal, Alcaide, Extremera, & Pizarro, 2006). Of greater relevance to the present study, studies examining ER strategies (Garnefski et al., 2005; Silk et al., 2003) and physiological markers of ER (e.g., Beauchaine, Gatzke-Kopp, & Mead, 2007) have found that ER is associated with internalizing and externalizing
problems (Garnefski et al; 2005), depression and problem behavior (Silk et al., 2003), and conduct problems (Beauchaine et al., 2007) among adolescents. Further, some evidence suggests that ER difficulties may play a greater role in adolescent internalizing than externalizing problems (Garnefski et al., 2005).

These studies provide preliminary evidence for the importance of certain aspects of ER to adolescent functioning; however, other important dimensions of ER remain unstudied among adolescents. For example, two of the most commonly used measures of emotional intelligence (the Trait Meta Mood Scale and Swinburne University Emotional Intelligence Test; see Salovey, Mayer, Goldman, Turvey, & Palfai, 1995 and Luebbers, Downey, & Stough, 2007, respectively) do not assess the ability to control behaviors when experiencing negative emotions or the acceptance of emotions. Likewise, measures of ER strategies (e.g., the Cognitive Emotion Regulation Scale; Garnefski, Kraaij, & Spinhoven, 2002, cf. Garnefski, et al., 2005) focus only on this particular dimension of ER difficulties, to the exclusion of aspects such as the awareness, understanding, and acceptance of emotions. To better understand the nature and role of ER in adolescence, a comprehensive measure that assesses all theoretically relevant aspects of ER difficulties is needed.

The Present Study

The goal of the present study was to examine if a widely-used and empirically-supported measure of ER difficulties in adults (the DERS) has utility in the assessment of ER difficulties among adolescents. To this end, we examined three questions. First, we examined whether the factor structure of the DERS previously found in adults is replicable among a sample of adolescents. Given that the conceptualization of ER on which the DERS is based in theoretical literature on ER among youth, we expected that this would be the case. Additionally, based on findings pertaining to emotional development among youth we expected that the specific dimensions of ER difficulties assessed in the DERS would be observable and stable among adolescents (Stegge & Meerum-Terwogt, 2007).

The second question pertains to potential gender differences in ER difficulties. Findings of lower emotional expression/verbalization among boys versus girls (Brody & Hall, 1993) suggest that boys may have less emotional awareness than girls. Further, a meta-analysis of gender differences in temperament showed that girls are better at inhibiting inappropriate behavioral responses than boys (Else-Quest, Hyde, Goldsmith, & van Hulle, 2006). Conversely, compared to their male peers, adolescent girls have been found to use more maladaptive coping strategies, such as resignation (Hampel & Petermann, 2006), and to report experiencing higher levels of guilt, shame, and self-directed hostility (Hamilton & Jensvold, 1992), some of which may be in response to emotions that are perceived as inappropriate. Accordingly, we hypothesized that female adolescents, compared to their male peers, would report greater emotional awareness and less difficulty controlling their behaviors when distressed, but also more emotional nonacceptance (in the form of secondary emotional responses to negative emotions) and less access to ER strategies perceived as effective. Before levels of ER difficulties between male and female adolescents can be
compared, however, gender invariance in the measurement of ER difficulties must be established. Thus, we examined whether the DERS demonstrates measurement invariance with respect to male and female participants.

The study’s third question pertained to the association between ER difficulties and psychopathology in adolescence. We explored the concurrent validity of DERS scores by studying the association between the DERS and adolescents’ symptoms of internalizing and externalizing psychopathology. Based on previous studies using the DERS in adult samples (as well as evidence of the role of ER strategies in adolescent psychopathology), we hypothesized that scores on the DERS subscales would be associated with both externalizing and internalizing difficulties among adolescents, with generally stronger relations between the DERS and internalizing as opposed to externalizing problems.

METHOD

Participants

All 1,003 students at a school for secondary education, including Atheneum (60.3%), Gymnasium (21.5%), and HAVO (a Dutch acronym for ‘higher general secondary education’; 18.2%), in Amsterdam, The Netherlands, were invited to participate in the study. These school forms represent higher levels of secondary education in the Netherlands and are attended by approximately 56% of all secondary school students in the Netherlands (Centraal Bureau voor Statistiek [CBS], n.d.). Parents of six of these 1,003 (0.6%) students did not grant permission for their children to complete the questionnaires, and another 78 students (7.8%) were not present on the day of testing. All other adolescents took part in the study. Further, to ensure that this adolescent sample does not overlap in age with adult samples, only adolescents under the age of 18 were included, resulting in the exclusion of an additional 49 students. Thus, the final sample consisted of 870 adolescents (441 girls, 429 boys) with a mean age of 14.34 years ($SD = 1.60$; age range 11-17 years). Female and male participants did not differ significantly in age, $t (870) = 1.00, p > .05$, Cohen’s $d = .07$. Almost all (95.5%) of the participants were born in the Netherlands; however, 40.1% reported having at least one parent born outside the Netherlands. This percentage is comparable to that of all Atheneum, Gymnasium, and HAVO students with a minority background (defined as having at least one parent born outside the Netherlands) found in larger cities of the Netherlands (CBS, n.d.), and is higher than that found for the Netherlands as a whole (20% students with minority status).
CHAPTER 2

Measures

Emotion Regulation

Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). The DERS is a 36 item self-report questionnaire that assesses clinically-relevant difficulties in ER (with a particular emphasis on negative emotions). Items are scored on six scales, labeled: Lack of Emotional Awareness (6 items), Lack of Emotional Clarity (5 items), Difficulties Controlling Impulsive Behaviors when Distressed (6 items), Difficulties Engaging in Goal-Directed Behavior When Distressed (5 items), Nonacceptance of Negative Emotional Responses (6 items), and Limited Access to Effective ER Strategies (8 items). Items are scored on a five-point scale ranging from 1 (almost never) to 5 (almost always). Subscale scores are obtained by summing the corresponding items. Evidence has been provided in support of the reliability of DERS scores. Specifically, DERS scores have been found to demonstrate good test-retest reliability over a period of 4 to 8 weeks in a sample of college students (ρI = .88; Gratz & Roemer, 2004), and both the overall DERS score and subscale scores have been found to have high internal consistency within both clinical (e.g., Fox et al., 2007; Gratz et al., 2008) and nonclinical populations (e.g., Gratz & Roemer, 2004; Johnson et al., 2008). Support for the construct and predictive validity of DERS scores within both clinical and nonclinical populations have also been found (Fox et al., 2007; Gratz, Bornovalova, Delany-Brunsey, Nick, & Lejuez, 2007; Gratz & Roemer, 2004, 2008; Gratz et al., 2006, 2009). The Flesch-Kincaid Grade Level of the DERS is 5.3, meaning the questionnaire should be understandable by average 5th graders (Kincaid, Fishburne, Rogers, & Chissom, 1975). For the purpose of the present study, the DERS was translated to Dutch. First, the scale was translated independently from English to Dutch by three translators, who then discussed their translations and combined them into one. Next, the translated scale was administered to 46 Dutch high school students (28 girls, M age = 12.65, age range 12-13 years). Difficult or misinterpreted items were rephrased (n = 2). Although we did not use a formal backtranslation procedure before the study was conducted, the Dutch version of the DERS used in the study was back-translated to English by a professional translator after the assessment. The back-translated DERS was consistent with the original DERS.

Externalizing Problem Behavior

Youth Self-Report (YSR) – Externalizing Items (Achenbach, 1991). The 30 YSR Externalizing items assess Aggressive Behavior (19 items; e.g., “I physically attack people”, and “I argue a lot”) and Delinquent Behavior (11 items; e.g., “I hang around with others who get in trouble”, and “I steal from home”). Each item is scored on a three-point scale of 0 (not true), 1 (somewhat or sometimes true), or 2 (very or often true). Raw scores were summed to obtain scores for Aggressive and Delinquent Behavior, respectively. The Dutch version of the YSR Externalizing scales (Verhulst, Van der Ende, & Koot, 1997) was used with permission of the authors. Support
for YSR scores’ construct and predictive validity have been provided (Verhulst et al., 1997). Cronbach’s alphas were .79 for the scale Aggressive Behavior and .70 for Delinquent Behavior in the present adolescent sample. The factor structure of the Dutch YSR was found to be similar to the US version (de Groot, Koot, & Verhulst, 1996). To assess whether administering the YSR externalizing scales outside the standard item set of the entire YSR affected the variance of the scores, we compared the standard deviations (SDs) in our sample to the SDs of the Dutch general population sample (Verhulst et al., 1997). SDs were highly similar for female adolescents in the two samples (4.05 in the present sample versus 4.51 in the general population for Aggressive Behavior; 2.26 versus 2.21 for Delinquent Behavior) and somewhat higher for male adolescents in the present sample (5.87 versus 4.87 for Aggressive Behavior and 3.57 versus 2.57 for Delinquent Behavior) compared to the general Dutch sample. Given that the variance of the scale scores was at least as high in the present sample, compared to the general population in which the full YSR was used, the likelihood of detecting potential relations between the YSR externalizing scores in our sample and DERS scales does not seem to be compromised by the fact that only the YSR externalizing scales were administered.

**Internalizing Problems**

*Screen for Child Anxiety Related Emotional Disorders (SCARED; Birmaher, Ketharpal, Brent, Cully, Balach, Kaufman, et al., 1997).* The SCARED is a 38-item self-report questionnaire that assesses anxiety disorder symptoms in children and adolescents consistent with the DSM-IV (APA, 1994) classification scheme (Generalized Anxiety, Separation Anxiety, Somatic/Panic, Social Phobia, and School Phobia). Examples of items are “When I get frightened, I feel like I am choking” (Somatic/Panic), “I feel shy with people I don’t know well” (Social Phobia), and “I am a worrier” (Generalized Anxiety). In the present study, only the total anxiety score will be used. Items are rated on a three-point scale, with 0 (almost never), 1 (sometimes), and 2 (often). Evidence for the SCARED scores’ concurrent validity has been demonstrated (Muris, Merckelbach, Mayer, van Brakel, Thissen, Moulard et al., 1998), and the original five factor structure has been shown to apply to the Dutch SCARED (Hale, Raaijmakers, Muris, & Meeus, 2005). Cronbach’s alpha was .93 for the SCARED total score in the present adolescent sample.

*Reynolds Adolescent Depression Scale - 2nd Edition (RADS-2; Reynolds, 2002).* The RADS-2 assesses the severity of self-reported depressive symptoms in adolescents. The RADS-2 contains 30 items and four subscales, named Dysphoric Mood, Anhedonia/Negative Affect, Negative Self-Evaluation and Somatic Complaints. Examples of items are “I feel sad” (Dysphoric Mood) and “I feel I am bad” (Negative Self-Evaluation). In the present study, only the RADS-2 total score will be targeted for analyses, because the focus lies on establishing the usefulness of the DERS in research with adolescents, rather than on showing how ER difficulties relate to diverse aspects of depression. Adolescents are asked to indicate on a four-point rating scale (ranging from 1 “almost never” to 4 “most of the time”) the extent to which each item applies to them. The total score is calculated by summing responses on all items. RADS-2 scores have shown adequate internal consistency (α =
CHAPTER 2

.92) and test–retest reliability ($r = .80$) in a sample of high school students, and the construct validity of scores on this measure has also been supported (e.g., scores on this scale have been found to differentiate between clinically depressed adolescents and non-depressed adolescents; Reynolds, 2002). Cronbach’s alpha for the total scale was .90 in the present sample. The RADS-2 was translated to Dutch using the procedure described by Varni, Seid, and Rode (1999), including forward and backward translation, and pilot testing.

Procedure

The adolescents’ parents received written information about the study and the possibility to disallow their children’s participation. Adolescents themselves were informed about the study in their classrooms, and completed the questionnaires after completing an informed assent form. Because assessment sessions lasted only 30 – 45 minutes per class, it was not possible for students to complete all of the measures of interest. Therefore, whereas all students completed the DERS ($N = 870$), the other measures of interest in this study were distributed across participants, with 215 completing the YSR Externalizing subscale, 212 completing the SCARED, and 197 completing the RADS-2. Respondents were assigned to one of the questionnaire packages randomly, stratified by gender and age. Upon completion of the questionnaires, adolescents received a small gift in return for their participation.

RESULTS

**DERS Factor Structure: Confirmatory Factor Analysis in the Adolescent Sample**

We first tested whether the factor structure of the DERS in our adolescent sample was equivalent to the structure found for adults using a confirmatory factor analysis (CFA). Six latent variables were specified, corresponding to the six subscales of the DERS, which were allowed to correlate. The CFA and all following structural models were analyzed in Mplus 4.21 (Muthén & Muthén, 1998-2007). Model fit was determined through the comparative fit index and Tucker-Lewis Index (CFI & TLI; exact fit = 1.00, close fit 0.95 - 0.99, acceptable fit 0.90 – 0.95; Bentler & Bonett, 1980) and root mean square error of approximation (RMSEA; exact fit = 0.00, close fit 0.06 – 0.01, acceptable fit 0.08 – 0.06; Browne & Cudeck, 1993). Results are shown in Table 2.1. Based on model modification indices, item 33 of the original Difficulties Engaging in Goal-directed Behavior When Distressed scale (“When I’m upset, I have difficulty thinking about anything else”) was allowed to cross-load on the Limited Access to ER Strategies scale. When allowing for this cross-loading, model fit was acceptable (CFI = .92, TLI = .91, RMSEA = .045 (90% CI = .043 - .048), suggesting that the structure of the DERS in adolescents is equivalent to that found among adults (Gratz & Roemer, 2004). The correlations between the six subscales were low to medium in size (range -.12 to .54, median = .35), suggesting that the subscales tap different aspects of ER
difficulties (see Table 2.2). Cronbach’s alphas were satisfactory to high within this adolescent sample (range = .72-.87; see Table 2.1).
Table 2.1
Factor Loadings for DERS Items Obtained from Confirmatory Factor Analysis in Dutch Adolescents (N = 870)

<table>
<thead>
<tr>
<th>Item</th>
<th>Factor Loadings</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Lack of Emotional Awareness (α = .73 for boys, α = .76 for girls)</td>
<td></td>
</tr>
<tr>
<td>2. I pay attention to how I feel.</td>
<td>.56</td>
</tr>
<tr>
<td>6. I am attentive to my feelings.</td>
<td>.66</td>
</tr>
<tr>
<td>8. I care about what I am feeling.</td>
<td>.79</td>
</tr>
<tr>
<td>10. When I’m upset, I acknowledge my emotions.</td>
<td>.25</td>
</tr>
<tr>
<td>17. When I’m upset, I believe my emotions are valid and important.</td>
<td>.57</td>
</tr>
<tr>
<td>34. I take time to figure out what I am really feeling.</td>
<td>.49</td>
</tr>
<tr>
<td>II. Lack of Emotional Clarity (α = .74 for boys, α = .83 for girls)</td>
<td></td>
</tr>
<tr>
<td>1. I am clear about my feelings.</td>
<td>.59</td>
</tr>
<tr>
<td>4. I have no idea how I am feeling.</td>
<td>.57</td>
</tr>
<tr>
<td>5. I have difficulty making sense out of my feelings.</td>
<td>.74</td>
</tr>
<tr>
<td>7. I know exactly how I am feeling.</td>
<td>.59</td>
</tr>
<tr>
<td>9. I am confused about how I am feeling.</td>
<td>.70</td>
</tr>
<tr>
<td>III. Difficulties Controlling Impulsive Behaviors When Distressed (α = .86 for boys, α = .83 for girls)</td>
<td></td>
</tr>
<tr>
<td>3. I experience my emotions as overwhelming &amp; out of control.</td>
<td>.38</td>
</tr>
<tr>
<td>14. When I’m upset, I become out of control.</td>
<td>.81</td>
</tr>
<tr>
<td>19. When I’m upset, I feel out of control.</td>
<td>.83</td>
</tr>
<tr>
<td>24. When I’m upset, I feel I can remain in control over my behavior.</td>
<td>.62</td>
</tr>
<tr>
<td>27. When I’m upset, I have difficulty controlling my behavior.</td>
<td>.72</td>
</tr>
<tr>
<td>32. When I’m upset, I lose control over my behavior.</td>
<td>.83</td>
</tr>
<tr>
<td>IV. Difficulties Engaging in Goal-directed Behaviors When Distressed (α = .81 for boys, α = .82 for girls)</td>
<td></td>
</tr>
<tr>
<td>13. When I’m upset, I have difficulty getting work done.</td>
<td>.76</td>
</tr>
<tr>
<td>18. When I’m upset, I have difficulty focusing on other things.</td>
<td>.77</td>
</tr>
<tr>
<td>20. When I’m upset, I can still get things done.</td>
<td>.52</td>
</tr>
<tr>
<td>26. When I’m upset, I have difficulty concentrating.</td>
<td>.79</td>
</tr>
<tr>
<td>33. When I’m upset, I have difficulty thinking about anything else</td>
<td>.44 [VI {.35}]</td>
</tr>
<tr>
<td>V. Nonacceptance of Negative Emotional Responses (α = .72 for boys, α = .81 for girls)</td>
<td></td>
</tr>
<tr>
<td>11. When I’m upset I become angry at myself for feeling that way.</td>
<td>.66</td>
</tr>
<tr>
<td>12. When I’m upset, I become embarrassed.</td>
<td>.56</td>
</tr>
<tr>
<td>21. When I’m upset, I feel ashamed with myself.</td>
<td>.63</td>
</tr>
<tr>
<td>23. When I’m upset, I feel like I am weak.</td>
<td>.59</td>
</tr>
<tr>
<td>25. When I’m upset, I feel guilty.</td>
<td>.62</td>
</tr>
<tr>
<td>29. When I’m upset, I become irritated with myself.</td>
<td>.65</td>
</tr>
</tbody>
</table>
VI. Limited Access to ER Strategies ($\alpha = .80$ for boys, $\alpha = .87$ for girls)

15. When I’m upset I believe I’ll remain that way for a long time. $\cdot 67$
16. When I’m upset I believe that I’ll end up very depressed. $\cdot 70$
22. When I’m upset, I know that I can find a way to feel better. $\cdot 40$
28. When I’m upset I believe there is nothing I can do to feel better. $\cdot 67$
30. When I’m upset, I start to feel very bad about myself. $\cdot 68$
31. When I’m upset I believe that wallowing in it is all I can do. $\cdot 77$
35. When I’m upset, it takes me along time to feel better. $\cdot 68$
36. When I’m upset, my emotions feel overwhelming. $\cdot 65$

*Note. DERS = Difficulties in Emotion Regulation Scale; ER = Emotion Regulation; Numbers in square brackets indicate the factor on which an item showed a cross loading (given in {}).*

Table 2.2

Correlations among DERS Scales in Adolescents ($N = 870$)

<table>
<thead>
<tr>
<th>DERS Scales</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Lack of Emotional Awareness</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Lack of Emotional Clarity</td>
<td></td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 Difficulties Controlling Impulsive Behavior</td>
<td>.10*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 Difficulties Controlling Impulsive Behavior</td>
<td></td>
<td>.34**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 Difficulties Engaging in Goal-directed Behavior</td>
<td>-.10*</td>
<td>.35**</td>
<td>.42**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 Nonacceptance of Negative Emotional Responses</td>
<td>-.12**</td>
<td>.35**</td>
<td>.39**</td>
<td>.37**</td>
<td></td>
</tr>
<tr>
<td>6 Limited Access to ER Strategies</td>
<td>-.09*</td>
<td>.47**</td>
<td>.50**</td>
<td>.54**</td>
<td>.54**</td>
</tr>
</tbody>
</table>

*Note. DERS = Difficulties in Emotion Regulation Scale; ER = Emotion Regulation.*

* $p < .01$ (one-tailed). ** $p < .001$ (one-tailed). 1 = Lack of Emotional Awareness. 2 = Lack of Emotional Clarity. 3 = Difficulties Controlling Impulsive Behavior when Distressed. 4 = Difficulties Engaging in Goal-directed Behavior when Distressed. 5 = Nonacceptance of Negative Emotional Responses. 6 = Limited Access to Emotion Regulation Strategies.

Gender Differences in DERS Subscales

Next, we tested for measurement invariance between male and female participants, specified by subscale. Results are shown in Table 2.3. We started by fitting baseline sex difference configural models, in which males (reference category) were contrasted with females. Adequate model fit for this model is a pre-requirement for further testing of invariance. A latent factor was considered for each scale, indicated by the items. The variance of the latent factor was fixed at 1, and the means
were fixed at 0 for both male and female adolescents. Factor loadings were freely estimated in both samples. All configural models had exact to acceptable fit to the data (see Table 2.3).

Next, we specified metric invariance models in which the factor loadings were held equal between the male and female samples, in order to test whether items contribute equally to the total score for male and female adolescents. In the male sample, the variance of the latent factor was fixed at 1, whereas this was freely estimated in the female sample. The means of the latent factors were fixed at zero in both samples. As these metric invariance models were nested within the configural models, deterioration of model fit (usually assessed using the chi-square difference test) is the outcome of interest. However, the chi-square difference test has substantial power in large samples (n = 200) to detect small discrepancies of no theoretical or practical consequence (Chen, Sousa, & West, 2005). We therefore considered only a decrease in the CFI greater than .01 to be an indication of a meaningful decrement in fit (see Cheung & Rensvold, 2002). Although some $\chi^2$ difference tests were significant, no drop in CFA > .01 was observed.

We then examined the factorial invariance of the DERS among female and male adolescents, in order to test whether the items’ intercepts are equivalent for males and females. Latent factor means and variances were fixed at 0 and 1 respectively in males, and estimated freely in females. A decrease in CFI > .01 was found for the subscales of Lack of Emotional Awareness ($\Delta$ CFI = .044), Difficulties Engaging in Goal-directed Behavior when Distressed ($\Delta$ CFI = .014), and Nonacceptance of Negative Emotional Responses ($\Delta$ CFI = .017).
<table>
<thead>
<tr>
<th>Scale</th>
<th>χ²</th>
<th>df</th>
<th>CFI</th>
<th>TLI</th>
<th>RMSEA</th>
<th>90% C. I. RMSEA</th>
<th>Δχ²</th>
<th>Δdf</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lack of Emotional Awareness (6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Configural</td>
<td>2.74</td>
<td>12</td>
<td>1.000</td>
<td>1.000</td>
<td>0.00</td>
<td>0.00 – 0.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metric</td>
<td>16.00</td>
<td>17</td>
<td>1.000</td>
<td>1.000</td>
<td>0.00</td>
<td>0.00 – 0.04</td>
<td>13.26</td>
<td>5</td>
</tr>
<tr>
<td>Strong factorial</td>
<td>74.69</td>
<td>22</td>
<td>0.956</td>
<td>0.940</td>
<td>0.07</td>
<td>0.06 – 0.09</td>
<td>58.69</td>
<td>5**</td>
</tr>
<tr>
<td>Lack of Emotional Clarity (5)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Configural</td>
<td>16.90</td>
<td>6</td>
<td>0.991</td>
<td>0.971</td>
<td>0.07</td>
<td>0.03 – 0.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metric</td>
<td>27.12</td>
<td>10</td>
<td>0.987</td>
<td>0.973</td>
<td>0.06</td>
<td>0.04 – 0.09</td>
<td>10.22</td>
<td>4*</td>
</tr>
<tr>
<td>Strong factorial</td>
<td>39.29</td>
<td>14</td>
<td>0.980</td>
<td>0.972</td>
<td>0.06</td>
<td>0.04 – 0.09</td>
<td>12.17</td>
<td>4*</td>
</tr>
<tr>
<td>Difficulties Controlling Impulsive Behavior When Distressed (6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Configural</td>
<td>16.23</td>
<td>16</td>
<td>1.000</td>
<td>1.000</td>
<td>0.01</td>
<td>0.00 – 0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metric</td>
<td>23.88</td>
<td>21</td>
<td>0.999</td>
<td>0.998</td>
<td>0.02</td>
<td>0.00 – 0.05</td>
<td>7.65</td>
<td>5</td>
</tr>
<tr>
<td>Strong factorial</td>
<td>34.06</td>
<td>26</td>
<td>0.996</td>
<td>0.996</td>
<td>0.03</td>
<td>0.00 – 0.05</td>
<td>10.18</td>
<td>5</td>
</tr>
<tr>
<td>Difficulties Engaging in Goal-directed Behavior When Distressed (5)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Configural</td>
<td>17.20</td>
<td>8</td>
<td>0.994</td>
<td>0.984</td>
<td>0.05</td>
<td>0.02 – 0.09</td>
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<td></td>
</tr>
<tr>
<td>Metric</td>
<td>19.07</td>
<td>12</td>
<td>0.995</td>
<td>0.992</td>
<td>0.04</td>
<td>0.00 – 0.07</td>
<td>1.87</td>
<td>4</td>
</tr>
<tr>
<td>Strong factorial</td>
<td>43.42</td>
<td>16</td>
<td>0.981</td>
<td>0.976</td>
<td>0.06</td>
<td>0.04 – 0.09</td>
<td>24.35</td>
<td>4**</td>
</tr>
<tr>
<td>Nonacceptance of Negative Emotional Responses (6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Configural</td>
<td>26.05</td>
<td>14</td>
<td>0.990</td>
<td>0.978</td>
<td>0.04</td>
<td>0.02 – 0.07</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metric</td>
<td>37.57</td>
<td>19</td>
<td>0.985</td>
<td>0.976</td>
<td>0.05</td>
<td>0.02 – 0.07</td>
<td>11.52</td>
<td>5*</td>
</tr>
<tr>
<td>Strong factorial</td>
<td>63.46</td>
<td>24</td>
<td>0.968</td>
<td>0.960</td>
<td>0.06</td>
<td>0.04 – 0.08</td>
<td>25.89</td>
<td>5**</td>
</tr>
<tr>
<td>Limited Access to Emotion Regulation Strategies (8)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Configural</td>
<td>122.04</td>
<td>38</td>
<td>0.963</td>
<td>0.946</td>
<td>0.07</td>
<td>0.06 – 0.09</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metric</td>
<td>129.12</td>
<td>45</td>
<td>0.963</td>
<td>0.954</td>
<td>0.07</td>
<td>0.05 – 0.08</td>
<td>7.08</td>
<td>7</td>
</tr>
<tr>
<td>Strong factorial</td>
<td>156.80</td>
<td>52</td>
<td>0.954</td>
<td>0.951</td>
<td>0.07</td>
<td>0.06 – 0.08</td>
<td>27.68</td>
<td>7**</td>
</tr>
</tbody>
</table>

*Note. DERS = Difficulties in Emotion Regulation Scale; CFI = comparative fit index; TLI = Tucker Lewis index; RMSEA = root mean square error of approximation. * p < .05 (two-tailed). ** p < .001 (two-tailed).

Mean Differences in DERS Scales Between Males and Females

A multivariate analysis of variance (MANOVA) on the DERS factors showed a significant overall effect of gender, Pillai’s Trace: $F(6, 863) = 21.04, p <.001$. No gender differences were found for Difficulties Controlling Impulsive Behaviors When Distressed. Female participants reported significantly higher levels of Lack of Emotional Clarity, Difficulties Engaging in Goal-directed Behaviors when Distressed, Nonacceptance of Negative Emotional Responses, and Limited Access to ER Strategies. Male participants reported higher levels of Lack of Emotional
CHAPTER 2
Awareness (see Table 2.4). The observed gender differences on the subscales Lack of Emotional Awareness, Difficulties Engaging in Goal-directed Behavior when Distressed, and Nonacceptance of Negative Emotional Responses should be interpreted with caution, as they may reflect gender-based differences in the ratings of items in addition to true gender differences in these ER difficulties.

**DERS Scales and Externalizing and Internalizing Problems**

Given that subsamples completed only one measure of psychopathology (see Procedure), we first examined whether the subsamples differed on the DERS. Results of a MANOVA showed no overall effect of sample on DERS scores, Pillai’s Trace: $F(12, 1234) = 1.65, p > .05$.

Zero-order correlations between the DERS subscales and each measure of psychopathology are in Table 2.5. Small but statistically significant positive associations between DERS subscales and adolescent externalizing problems were found. Correlations between DERS subscales and internalizing problems were generally large and positive.

In order to determine if particular DERS subscales are uniquely related to internalizing and externalizing problems, a series of standard multiple regression analyses were conducted for each measure of psychopathology. The DERS subscales were entered together with gender in the first step of the equation (see Table 2.6). Results provide support for the differential relevance of particular DERS subscales to different forms of psychopathology. Specifically, both Difficulties Controlling Impulsive Behaviors When Distressed and Difficulties Engaging in Goal-directed Behavior When Distressed were associated with Aggressive Behavior; Lack of Emotional Awareness was associated with Delinquent Behavior; and Lack of Emotional Clarity, Nonacceptance of Negative Emotional Responses, and Limited Access to ER Strategies were associated with Anxiety and Depression.
### Table 2.4
**Mean DERS, Anxiety, Depression, Aggression and Delinquency Scores (Standard Deviations) for Female and Male Adolescents**

<table>
<thead>
<tr>
<th>DERS Scale</th>
<th>Total sample</th>
<th>Males</th>
<th>Females</th>
<th>F</th>
<th>Cohen’s d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lack of Emotional Awareness</td>
<td>N = 870</td>
<td>N = 429</td>
<td>N = 441</td>
<td>51.47**</td>
<td>.49</td>
</tr>
<tr>
<td>Lack of Emotional Clarity</td>
<td>18.45 (4.92)</td>
<td>19.63 (4.74)</td>
<td>17.31 (4.81)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Difficulties Controlling Impulsive Behavior when Distressed</td>
<td>8.96 (3.27)</td>
<td>8.39 (3.00)</td>
<td>9.51 (3.46)</td>
<td>26.27**</td>
<td>-.35</td>
</tr>
<tr>
<td>Difficulties Engaging in Goal-directed Behavior when Distressed</td>
<td>10.82 (4.51)</td>
<td>10.94 (4.68)</td>
<td>10.71 (4.34)</td>
<td>.58</td>
<td>.05</td>
</tr>
<tr>
<td>Nonacceptance of Negative Emotional Responses</td>
<td>10.52 (4.00)</td>
<td>10.04 (4.34)</td>
<td>10.98 (4.31)</td>
<td>12.21**</td>
<td>-.22</td>
</tr>
<tr>
<td>Limited Access to ER Strategies</td>
<td>15.28 (5.64)</td>
<td>14.17 (4.80)</td>
<td>16.35 (6.17)</td>
<td>34.00**</td>
<td>-.39</td>
</tr>
<tr>
<td>Aggression</td>
<td>N = 215</td>
<td>N = 108</td>
<td>N = 107</td>
<td>7.77 (5.03)</td>
<td>.18</td>
</tr>
<tr>
<td>Delinquency</td>
<td>3.65 (3.11)</td>
<td>4.53 (3.57)</td>
<td>2.77 (2.26)</td>
<td>18.66**</td>
<td>.59</td>
</tr>
<tr>
<td>Anxiety</td>
<td>N = 212</td>
<td>N = 98</td>
<td>N = 114</td>
<td>55.39 (10.85)</td>
<td>.88</td>
</tr>
<tr>
<td>Depression</td>
<td>N = 197</td>
<td>N = 92</td>
<td>N = 105</td>
<td>51.45 (11.99)</td>
<td>.48</td>
</tr>
</tbody>
</table>

**Note.** DERS = Difficulties in Emotion Regulation Scale; ER = Emotion Regulation. *F*-values as obtained from (post hoc) univariate analyses of variance. ** *p < .001 (two-tailed).
### Table 2.5

Zero-order Correlations between DERS Subscales and Externalizing and Internalizing Problems in Adolescents

<table>
<thead>
<tr>
<th>DERS scales</th>
<th>YSR</th>
<th>SCARED</th>
<th>RADS-2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Aggression</td>
<td>Delinquency</td>
<td>Anxiety</td>
</tr>
<tr>
<td>Lack of Emotional Awareness</td>
<td>.04</td>
<td>.22**</td>
<td>-.09</td>
</tr>
<tr>
<td>Lack of Emotional Clarity</td>
<td>.07</td>
<td>-.01</td>
<td>.51***</td>
</tr>
<tr>
<td>Difficulties Controlling Impulsive Beh when Distressed</td>
<td>.30***</td>
<td>.10</td>
<td>.28***</td>
</tr>
<tr>
<td>Difficulties Engaging in Goal-directed Beh when Distressed</td>
<td>.29***</td>
<td>.04</td>
<td>.48***</td>
</tr>
<tr>
<td>Nonacceptance of Negative Emotional Responses</td>
<td>.11</td>
<td>.05</td>
<td>.52***</td>
</tr>
<tr>
<td>Limited Access to ER Strategies</td>
<td>.25***</td>
<td>.07</td>
<td>.69***</td>
</tr>
</tbody>
</table>

*Note.* DERS = Difficulties in Emotion Regulation Scale; YSR = Youth Self-Report; SCARED = Screen for Child Anxiety Related Emotional Disorders; RADS = Reynolds Adolescent Depression Scale; ER = Emotion Regulation; Beh = Behavior. ** *p < .01 (one-tailed). *** *p < .001 (one-tailed).
Table 2.6

*Standardized Regression Weights of DERS Subscales Regressed on Internalizing and Externalizing Problems in Adolescents*

<table>
<thead>
<tr>
<th>DERS scales</th>
<th>YSR Aggression N = 215</th>
<th>YSR Delinquency N = 215</th>
<th>SCARED Anxiety N = 210</th>
<th>SCARED Depression N = 197</th>
<th>RADS-2 Anxiety N = 215</th>
<th>RADS-2 Depression N = 197</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lack of Emotional Awareness</td>
<td>.12</td>
<td>.17**</td>
<td>-.14</td>
<td>-.06</td>
<td>.17**</td>
<td>.45***</td>
</tr>
<tr>
<td>Lack of Emotional Clarity</td>
<td>-.13</td>
<td>-.15</td>
<td>.21**</td>
<td>.45***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Difficulties Controlling Impulsive Beh when Distressed</td>
<td>.19*</td>
<td>.04</td>
<td>-.13</td>
<td>-.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Difficulties Engaging in Goal-directed Beh when Distressed</td>
<td>.25**</td>
<td>.04</td>
<td>.08</td>
<td>.06</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonacceptance of Negative Emotional Responses</td>
<td>-.03</td>
<td>.08</td>
<td>.20***</td>
<td>.23***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Limited Access to ER Strategies</td>
<td>.09</td>
<td>.07</td>
<td>.48****</td>
<td>.25***</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. DERS = Difficulties in Emotion Regulation Scale; ER = Emotion Regulation; Beh = Behavior. *p < .05 (two-tailed). **p < .01 (two-tailed). ***p < .001 (two-tailed).*

Given that the associations of the DERS subscale scores with internalizing problems were somewhat stronger than their associations with externalizing problems, we carefully inspected the questionnaires for possible overlapping items. No evidence of item overlap between the SCARED and the DERS was found. Further, although five items of the RADS-2 had possible overlap with DERS items, exclusion of these overlapping items had only a minimal influence on the regression weights (change in $\beta$s = ± .01-.02)

**DISCUSSION**

Results of the present study provide preliminary evidence for the utility of the DERS as a measure of ER difficulties in adolescents. The factor structure of the DERS previously established among adults was replicated in our adolescent sample. Further, metric invariance in the assessment of ER difficulties between male and female adolescents was found for all subscales, and strong factorial invariance between male and female adolescents was found for three of the six subscales. Finally, results demonstrated a number of gender differences in levels of self-reported ER difficulties, as well as meaningful associations between DERS scores and externalizing and internalizing problems.

Findings from confirmatory factor analyses revealed that the structure of the DERS in adolescents is equivalent to that previously found among adults. Further, the internal consistency coefficients of the factors were acceptable to high (average alpha for the subscales = .81) and
CHAPTER 2
comparable to those reported by Gratz and Roemer in their adult sample (2004; average alpha for
the subscales = .85).

With respect to gender differences in ER difficulties, findings indicated factor loading
equivalence for all subscales, and strong factorial invariance for the Lack of Emotional Clarity,
Difficulties Controlling Impulsive Behaviors When Distressed, and Limited Access to ER
Strategies subscales. With regard to the three subscales for which strong factorial invariance was
not found (i.e., Lack of Emotional Awareness, difficulties Engaging in Goal-directed Behavior
When Distressed, and Nonacceptance of Negative Emotional Responses), a (limited) portion of the
observed gender differences obtained using these scales may reflect differences in the interpretation
or rating of some of the items, in addition to actual differences in ER difficulties. For all other
subscales, evidence of factorial invariance suggests that gender differences in mean levels can be
attributed to true differences in self-reports of ER difficulties (cf. Gregorich, 2006).

Gender differences in levels of specific self-reported ER difficulties provided some support
for the hypotheses, as female adolescents reported significantly greater emotional nonacceptance,
greater emotional awareness, and less access to effective ER strategies than male adolescents.
Findings that female adolescents may have less access to effective ER strategies than their male
counterparts are consistent with findings that adolescent females score higher on measures of
maladaptive coping than adolescent males (Hampel & Petermann, 2006). In addition, findings that
male adolescents reported lower levels of emotional awareness than female adolescents are
consistent with Gratz and Roemer’s (2004) finding of gender differences on this subscale in
particular. Indeed, the effect sizes of these gender differences were comparable across these two
studies (Cohen’s $d = .42$ as calculated from Gratz & Roemer’s [2004] report, and $d = .49$ in the
present investigation). Contrary to hypotheses, however, female adolescents also reported lower
emotional clarity and greater difficulties engaging in goal-directed behaviors when distressed.
Further, our hypothesis that male participants would report greater difficulties controlling impulsive
behaviors when distressed was not confirmed. Although past studies have found that girls are better
at inhibiting inappropriate behavioral responses than boys (Else-Quest et al., 2006), it is possible
that boys develop better inhibitory control as they age, becoming closer to their female counterparts
in this regard during adolescence. Despite providing suggestive support for gender differences in
levels of specific ER difficulties, however, it is important to note that findings of gender differences
in lack of emotional awareness, difficulties engaging in goal-directed behavior when distressed, and
nonacceptance of negative emotional responses in particular may be due in part to the fact that boys
and girls use a different zero-point in response to some of the items of these subscales.

As expected, different dimensions of ER difficulties demonstrated statistically significant
and specific associations with both externalizing and internalizing problems, providing support for
the construct validity of DERS scores within this adolescent sample, as well as the utility (and
distinctiveness) of the DERS subscales. Also, whereas ER difficulties together accounted for 15%
and 13% of the variance in Aggressive Behavior and Delinquent Behavior, respectively, the DERS
subscales accounted for 58% and 59% of the variance in Anxiety and Depression, respectively.
Findings of a stronger relationship between ER difficulties as assessed with the DERS and
internalizing (vs. externalizing) problems are consistent with the results of Garnefski et al. (2005), who found that cognitive ER strategies explained more of the variance in internalizing than externalizing problems. Further, the higher explained variance of DERS scales with internalizing than with externalizing problems was not attributable to item overlap between the DERS and internalizing scales. Moreover, findings that ER difficulties accounted for almost twice as much variance in aggressive behavior than in delinquent behavior are in line with past findings suggesting that aggression implies more emotional involvement than delinquency (specifically, psychopathic delinquency; see Herpertz, Werth, Lukas, Qunaibi, Schuerkens, Kunert et al., 2001).

Several limitations warrant consideration. First, the present study was based on a general community sample, and associations might be different for clinical populations of adolescents. Generalizability of the results is further restricted by the fact that all participants attended the same school. An additional limitation is the exclusive reliance on self-report measures, which may be influenced by an individual’s willingness or ability to report accurately on their behaviors. Further, whereas the sole use of self-report data does not pose a limitation for our conclusions regarding the factor structure and internal consistency of the DERS scales in this sample, it may have resulted in an overestimation of the links between ER difficulties and internalizing and externalizing problems due to shared-method variance (e.g., Fergusson & Horwood, 1987). Nonetheless, it is important to note that scores on the DERS and its subscales have been found to be associated with behavioral, neurological, and experimental measures of related constructs, including behavioral measures of the willingness to experience emotional distress (Gratz et al., 2007), an experimental measure of emotion regulation (Gratz et al., 2006), and activation of the rostral anterior cingulate cortex (an area of the brain thought to be associated with inhibitory control) among cocaine dependent patients (Li, Huang, Bhagwagar, Milivojevic, & Sinha, 2008). Given that the DERS items focus primarily on the regulation of negative emotional states, future research should examine the role of difficulties with the regulation of positive emotional states in adolescent psychopathology as well. A final limitation regards the generalizability of the relation between DERS scores and externalizing problems. Use of the YSR externalizing problem items out of the context of the standard items may have influenced the results in unknown ways.

Despite the clear clinical significance of this line of research, the study of ER in adolescents is still in its infancy. Future research needs to examine how dimensions of ER difficulties develop over the course of adolescence, how they relate to the development of psychopathology, and the moderating roles of personal and social factors (e.g., social relations, and hormones and brain development) in the relationship between ER difficulties and psychopathology. This study suggests the potential utility of the DERS for future research on ER in adolescents, providing preliminary evidence for the reliability (specifically, internal consistency) and validity of scores on this measure among community adolescents.
Emotional Dynamics in the Development of Early Adolescent Psychopathology: A One-Year Longitudinal Study

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Manuscript submitted for publication
Dysregulated emotions are thought to play a significant role in the etiology and maintenance of many forms of psychopathology (e.g., Bradley, 2000; Cole & Hall, 2008). In fact, negative or dysregulated emotions are diagnostic symptoms of many disorders as described in the DSM-IV (American Psychiatric Association [APA], 1994). For instance, excessive anxiety and worry, and/or intense fear are implicated in the anxiety disorders, while irritability, anger, and mood lability are implicated in the disruptive behavior disorders (conduct disorder and oppositional defiant disorder; APA, 1994). High levels and prolonged duration of negative emotions and heightened emotional variability may be signs of emotional dysregulation (Cole & Hall, 2008). Individual differences in emotion regulation and their relation with the development of psychopathology become especially relevant during the developmental period of adolescence. Adolescence is characterized by an increase in the intensity and frequency of (negative) emotions (e.g., Larson & Lampman-Petraidis, 1989), heightened levels of emotional variability (Larson, Csikszentmihalyi, & Graef, 1980), increases in several types of psychopathology (e.g., Moffitt, Caspi, Dickson, Silva & Stanton, 1996; Wight, Sepulveda, & Aneshensel, 2004), and increased demand for independent self-regulation. However, longitudinal studies that address the role of emotion dysregulation in the development of psychopathology in adolescence are scarce. In consequence, little is known about the role of emotion dysregulation in general, and about the role of emotional dynamics (the intensity and variability of emotions; Silk, Steinberg, & Morris, 2003) in the development of adolescent behavioral and emotional problems, in particular. The present study focused on the role of the dynamics of four basic emotions (happiness, anger, anxiety, and sadness) in the one-year change of internalizing and externalizing psychopathology in early adolescence.

Emotional Dynamics and Psychopathology in Adolescence

Emotion regulation concerns the modulation of an initial emotional response (Cole & Hall, 2008), and the term “emotion dysregulation” refers to maladaptive patterns of emotion regulation, that is, to patterns of emotion regulation that are costly in the pursuit of long-term goals such as maintaining social relationships and well-being (Cole & Hall, 2008). The dysregulation of emotions may be studied at different levels of experience and cognition, including emotional dynamics (Silk et al., 2003), emotion knowledge (e.g., knowing that one may experience different emotions at the same time and that emotional experiences can be modulated; e.g., Meerum-Terwogt & Olthof, 1989), emotion regulation strategies (e.g., distraction, cognitive reinterpretation; Gross & Thompson, 2007), and meta-emotion experiences (e.g., nonacceptance of emotional responses; Gratz & Roemer, 2004). This study is focused on the role of emotion dynamics, as these reflect the outcome of regulatory efforts and processes. At the level of emotion dynamics, emotion dysregulation may be reflected in the form of heightened and prolonged negative emotions, and heightened variability of both negative and positive emotions (Cole & Hall, 2008; Silk et al., 2003). We define emotional variability (EV) here as “the frequency with which an individual’s emotions change, and the extremity of these changes”. It should be noted that some variability of emotions is clearly expected as a result of normal emotional reactivity to environmental stressors. Heightened
variability, however, may be an indication of extreme emotional reactivity and ineffective attempts at emotion regulation.

As indicated earlier, early to mid adolescence is an opportune time to study emotional dynamics, and its links with psychopathology. EV is heightened in adolescents compared to both younger children and adults as shown in a study, in which EV was assessed several times a day over the course of 1 week (Larson et al., 1980). Although heightened EV may partly be a sign of normative developmental changes in adolescence (Larson et al., 1980), comparatively high levels of EV in adolescents may indicate emotional dysregulation, and be indicative of future psychopathology (Schneiders, Nicolson, Berkhof, Feron, van Os, & de Vries, 2006).

Several cross-sectional studies demonstrate that in adolescence, high levels of negative emotions, and high levels of EV are related to symptoms of depression (Larson, Raffaelli, Richards, Ham, & Jewell, 1990; Silk et al., 2003), and externalizing problems (Silk et al., 2003). These findings are commonly interpreted as suggesting that emotion dysregulation influences the development of psychopathology. However, given the cross-sectional nature of these studies, the role of emotion dysregulation and EV in the development of psychopathology needs further clarification. Therefore, in the present study, we first tested the hypothesis that high levels of negative emotions, low levels of positive emotions, and high variability of both positive and negative emotions add to the growth or development of different types of psychopathology. In addition, we addressed questions on the importance of intensity versus variability of emotions regarding the development of behavioral/emotional problems, on the specificity of the emotion by psychopathology type association, and the potential gender-specificity of these associations.

A first question is whether it is emotion dysregulation in general or dysregulation of specific emotions that is linked to the development of specific forms of psychopathology. For example, does dysregulated anger relate specifically to aggressive problems, and dysregulated sadness relate specifically to depression? ‘Functional continuity’ between discrete emotions and specific forms of psychopathology is often assumed (e.g., Cole & Hall, 2008; Malatesta & Wilson, 1988; Muris & Ollendick, 2005). That is, it is assumed that patterns of emotional responding become consolidated over time and consequently lead to specific forms of psychopathology (Zahn-Waxler, Klimes-Dougan, & Slattery, 2000). Accordingly, individuals who are often fearful are thought to be more likely to develop anxiety-related than other psychological disorders, individuals who are often sad, and experience little happiness will show depressive symptoms, and individuals who are easily angered will display symptoms of disruptive behavior disorders (e.g., Muris & Ollendick, 2005). Empirical evidence for specific links between emotions and specific forms of psychopathology in adolescence is rather mixed and may heavily depend on the method used to assess emotions. One cross-sectional study addressing links between observer-rated facial expressions of emotions (anger, fear, sadness, and embarrassment) and internalizing and externalizing problems in early-adolescent boys, provided support for the idea of specific links (Keltner, Moffitt, & Stouthamer-Loeber, 1995): boys with externalizing problems showed more anger, and boys with internalizing problems showed more fear. By contrast, studies focusing on the internal experience rather than the expression of emotion tend to find nonspecific associations between discrete emotions and forms of
CHAPTER 3

Psychopathology. High and variable levels of sadness have been associated with depressive symptoms, and high and variable levels of anger with aggressive problem behavior (Larson et al., 1990; Silk et al., 2003). However, both high and variable anger and anxiety also correlate with depressive symptoms, and high and variable levels of sadness and anxiety correlate with aggressive problem behavior scores in adolescents (Silk et al., 2003). As the present study used adolescent daily reports of experienced emotions we expected to find mainly nonspecific associations between levels and variability of emotions and change in psychopathology.

The study of basic emotional processes in the development and maintenance of psychopathology is not just of theoretical interest, but may also inform intervention and treatment. For instance, the question whether links between discrete negative emotions and psychopathology are better characterized as specific or as general, has potentially important implications for preventive interventions and treatment: if fear, as well as anger and sadness turn out to all play a role in anxiety disorder symptoms, teaching anxious youths how to deal with feelings of fear is insufficient.

Secondly, comparatively little is known about the role of positive emotions in the development of psychopathology. It has long been assumed that elevated negative emotions are a common feature of anxiety and mood disorders, whereas diminished positive affect is specific for mood disorders (e.g., Clark & Watson, 1991). A recent review and meta-analysis however, has shown significantly diminished positive affect in social anxiety (Kashdan, 2007). Regarding externalizing problems, results of studies that have included indices of positive emotions are mixed: no differences in the display of happiness between aggressive and non-aggressive youth (Orobio de Castro, Merk, Koops, Veerman, & Bosch, 2005), lower state but not trait happiness in delinquent youth than a comparison group (Plattner, Karnik, Jo, Hall, Schallauer, Carrion et al., 2007), and higher happiness in response to antisocial acts in adolescent males with conduct disorder have all been reported (Cimbora & McIntosh, 2003). We hypothesize that, in addition to high levels of negative emotions, low levels of positive emotions are related to increased levels of anxiety, depression, and aggressive behavior.

A third question concerns the gender-specificity of the hypothesized associations. It has been reported that males and females differ in levels of specific types of psychopathology (for a review see Zahn-Waxler, Shirtcliff, & Marceau, 2008), as well as in mean levels and variability of negative emotions (Silk et al., 2003). While it is uncertain whether adolescent sex needs to be taken into account when studying the role of emotional dynamics in the development of internalizing and externalizing problems, it might be that sex-related differences in emotional dynamics in part underlie the established gender differences in levels of internalizing and externalizing problems. For instance, emotion dysregulation may be more strongly associated with internalizing problems for females, and more strongly with aggressive behavior for males. The present study tested whether the associations between emotional dynamics and psychopathology hold across adolescent gender.
The Present Study

The present study addressed the role of the intensity and variability of happiness, anger, anxiety, and sadness in the development of internalizing and externalizing psychopathology in 452 adolescents followed from age 13 to 14. Adolescents’ experiences of emotions were obtained from three five-day periods, during which adolescents reported on their emotions every day. These intensive assessments of emotional experience contain two strengths: they minimize recall bias, inherent in many other self-report methods of subjective experience, and they maximize ecological validity (Shiffman, Stone, & Hufford, 2008). We expected to find that high levels and high variability of negative emotions (anger, anxiety, and sadness), predict the stability of adolescents’ anxiety, depression, and aggressive behavior scores from age 13 to age 14, in non-specific ways, thereby extending earlier cross-sectional findings (Silk et al., 2003). Further, we expected that positive emotions also play a role in the continuity of symptoms of psychopathology. Specifically, we predicted that low levels of - and high variability in - happiness partly predict the one-year continuity of adolescents’ anxiety, depression, and aggressive behavior scores. An additional question that was tested is whether the level and variability of emotions both contribute uniquely to the development of psychopathology. This is an important question, because the intensity and variability of emotions tend to correlate, but their contributions to symptoms of psychopathology have not been tested in one model (e.g., Silk et al., 2003). Finally, we tested whether the associations between emotional dynamics and psychopathology hold across adolescent gender.

METHOD

Participants

The sample used in the present study is the RADAR (Research on Adolescent Development and Relationships) study sample (see van Lier, Frijns, Neumann, den Exter Blokland, Koot, & Meeus, 2010 for an extensive description). Target adolescents were approached in grade 6 at 230 schools in the western and central parts of the Netherlands. A two step inclusion phase (teacher screen, followed by parent interviews) was used to include 497 families (target adolescent, mother, father, and sibling) as well as a friend of the target adolescent in an intensive longitudinal study. In the first step of the inclusion process, adolescents at increased risk for developing externalizing symptoms were oversampled, because of a specific focus of the RADAR study on delinquency development. High risk was determined by teacher ratings of the externalizing scales of the Teacher’s Report Form (TRF; Achenbach, 1991; Verhulst, van der Ende, & Koot, 1997). Youths who received a T-score ≥ 60 were labeled as high risk. Teacher information was available on 5,150 children. Due to the full family design and intense data collection, which requires a firm grip of the Dutch language, only adolescents who were identified by teachers as being of Dutch origin were selected (N = 3,237). Due to logistical reasons, 1,544 of these children were included in the second
sample selection phase. In this phase, youths’ parents were approached by phone. 463 adolescents were excluded, due to incorrect phone details, or because they did not meet additional requirements for inclusion in the study (both parents present, and presence of a sibling ≥ 10 years), and 583 actively refused to participate or failed to provide written consent, resulting in the inclusion of 497 families (291 target adolescents at average risk and 206 at high risk for externalizing problems). Family socioeconomic status is low in 11% of the sample, and the majority of participants (97%) had a Dutch-Caucasian background.

For the present study, participants were included if complete data were available for at least one annual assessments (N = 452, 55.3% male). Adolescents’ mean age at Time 1 was 13.37 years (SD = .61). Male and female adolescents did not differ regarding risk group status, $\chi^2(1) = .20$, $p > .05$, or age, $t(485) = .65$, $p > .05$.

Procedure

For the present study, data were collected during two home visits (time[T]1 and T5), and three internet assessments (T2, T3, and T4). Each assessment was separated from the next by a three months interval. During the home visits, adolescents completed questionnaires regarding symptoms of anxiety disorders, depression, and aggressive behavior as part of larger questionnaire packages under the supervision of trained interviewers. During the internet assessments, adolescents filled out their experiences of different emotions during that day, via internet. These assessments were completed for five consecutive days (Monday to Friday). Each day, participants received an invitation via e-mail to participate (at approximately 5.30 p.m.) and logged in to the RADAR website for data collection. Reminder e-mails were sent to participants who had not completed their assessment 1.5 hours after the initial invitation was sent, followed by text messages and phone calls to those who had still not completed their assessment 1.5 hours after the reminder had been sent.

Measures

Emotional Dynamics. Adolescents completed the Daily Mood Scale, an internet version of the Electronic Mood Device (Hoeksma, Sep, Vester, Groot, Sijmons, & deVries, 2000) at each of the three internet assessments (T2, T3, and T4) on five consecutive days for a total of 15 days. Each day, adolescents rated the intensity of happiness, anger, anxiety, and sadness on a 9-point Likert scale, ranging from ‘not at all’ to ‘very much’ during the daily internet sessions. Each emotion was tapped with three items (glad, happy, and cheerful for happiness; angry, cross, and short-tempered for anger; afraid, anxious, and worried for anxiety; sad, down, and dreary for sadness). These three items per emotion were summed into a total score per day. Cronbach’s $\alpha$ ranged from .86 to .94 for happiness, from .87 to .97 for anger, from .72 to .94 for anxiety, and from .92 to .97 for sadness across the total 15 days of assessment. Emotion level and variability scores were calculated only for participants who provided valid data on at least four days in total or on three consecutive days in a week.
Levels of Happiness, Anger, Anxiety, and Sadness were calculated for each emotion by summing emotion scores of all days within each week, divided by the number of valid assessments. Next, scores were summed per emotion across the three internet assessments.

Variability of Happiness, Anger, Anxiety, and Sadness. The variability scores were calculated per discrete emotion as the difference between scores in adjacent days within each internet assessment week. First, the absolute difference between consecutive data entries was calculated. For each participant, absolute difference scores were summed to a weekly difference score, and divided by the number of valid observations (to control for missing days). The weekly difference scores of the three internet assessment weeks were summed for every emotion, resulting in a single variability score each for happiness, anger, anxiety, and sadness. The validity of this way of calculating EV is supported by a number of findings. First, the measure shows moderate intraindividual stability over the course of three (rs = .35 and .49) and six months (r = .30), supporting the notion that EV is an individual difference characteristic (Larsen, 1987). Second, the notion of EV as a measure of emotional dysregulation receives some support from the fact that correlations between the variability and level of negative emotions are large and positive (rs = .51 to .68, ps < .001), but large and negative between variability and level of happiness (rs = -.46 to -.51, ps < .001). Third, and also supporting the notion of EV as emotion dysregulation, in a subsample of the present sample (N = 158), EV scores were positively related to adolescent self-reported difficulties with emotional clarity and impulse control, as well as with the nonacceptance of emotional responses, low confidence in one’s ability to modulate negative emotional responses, and difficulties engaging in goal-directed behavior when distressed (rs between .21, p < .05 and .34, p < .001) as assessed with the Difficulties in Emotion Regulation Scale (unpublished data, for the Difficulties in Emotion Regulation Scale see Gratz & Roemer, 2004; Neumann, van Lier, Gratz, & Koot, 2010). Finally, small but significant positive associations have been found between adolescents’ and mothers’ EV scores (mean r = .18, p < .05; Neumann, Lichtwarck-Aschoff, van Lier, Frijns, Meeus, & Koot, 2008).

Anxiety Disorder Symptoms were assessed with the Screen for Child Anxiety Related Emotional Disorders (SCARED; Birmaher, Ketharpal, Brent, Cully, Balach, Kaufman, et al., 1997). Adolescents’ self-reports of scores on the Social Phobia scale (5 items; “I feel shy with people I don’t know well”) and the Generalized Anxiety scale (8 items; e.g., “I am a worrier”) were used. Generalized Anxiety and Social Phobia are especially relevant during adolescence, as indicated by higher standardized mean scores compared to other anxiety disorders across adolescence (Hale, Raaijmakers, Muris, van Hoof, & Meeus, 2008 ). Items are rated on a three-point scale, with 0 (almost never), 1 (sometimes), and 2 (often). The Dutch SCARED has demonstrated good validity in adolescents (Hale, Raaijmakers, van Hof, & Meeus, 2005). In the present sample Cronbach’s αs were .77 and .80 for Social Phobia and .84 and .85 for Generalized Anxiety for T1 and T5, respectively. The two scales were combined into one ‘anxiety’ score.

Depressive Symptoms were assessed with the Reynolds Adolescent Depression Scale - 2nd Edition (RADS-2; Reynolds, 2002). Adolescent completed the subscales ‘Dysphoric Mood’ (8 items; “I feel like crying”), ‘Negative Self-Evaluation’ (8 items; “I feel I am bad”) and ‘Somatic
CHAPTER 3

Complaints’ (7 items; “I am tired”). Items are rated on a four-point scale ranging from 1 (almost never)” to 4 (most of the time). The scales were summed into a total depression score. Cronbach’s alphas of the total depression score were .89 at T1 and .94 at T5 for the present sample.

Aggressive Behavior was assessed through the Youth Self-Report (YSR; Achenbach, 1991). Adolescents completed the 19 items of the Aggressive Behavior subscale (e.g., “I physically attack people”, and “I argue a lot”). Each item is scored on a three-point scale including 0 (not true), 1 (somewhat or sometimes true), or 2 (very or often true). The validity of the Dutch version of the YSR was demonstrated (Verhulst, Van der Ende, & Koot, 1997). In the present sample, Cronbach’s alphas were .85 at T1 and .88 at T5.

Adolescent Sex was dummy coded (0 = male, 1 = female).

Risk Status of the sample was dummy coded (0 = low/average risk, 1 = at-risk). This variable indicates whether or not the adolescent received heightened cut-off scores on teacher and parent-reported externalizing symptoms at the initial screen before entry into this study (age 12). Because it is uncertain how risk status related expected level differences in psychopathology and emotion dynamics may affect their mutual relations we decided to control for Risk Status in our analyses.

Attrition and Missing Data

Complete data for anxiety disorder and depressive symptoms and aggressive behavior was available for 99% of the sample at age 13 and 95% at age 14. Seventy percent of the sample had complete data for emotion dynamics, while 19 percent missed emotion dynamics data for one of the three internet assessments, 7% missed data on two of the internet assessments, and emotion dynamics data were missing completely for 4% of the sample. Adolescents missing emotion dynamics data did not differ from the rest on the sample on any of the psychopathology measures.

Statistical Analyses

Path analysis models were run in Mplus5 (Muthén & Muthén, 2007). The analyses were performed in four stages. We started off by testing whether the level of the four emotions predicted changes in psychopathology scores from age 13 to 14. Models were run separately for each form of psychopathology in combination with each emotion. Presence of a significant indirect path from age 13 psychopathology to age 14 psychopathology through the level of an emotion was tested by using the IND command in Mplus, which calculates the joined significance of the indirect pathways according to the formula by MacKinnon and colleagues (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). Next, we tested whether the Variability of the four emotions predicted the one-year development of psychopathology, again separately for each form of psychopathology and each emotion. Third, to test whether variability or level is more important in the development of psychopathology, models were run in which the level of an emotion, and its variability were included as possible mediators. All models were controlled for sex and risk status. Finally, to test
for sex-differences in associations, multiple group models were run in which the associations were estimated for males and females separately. All models were corrected for non-normal distributions by maximum likelihood estimation with robust standard errors (MLR). Missing data were accounted for by full information maximum likelihood estimation.

RESULTS

Descriptive Statistics

Descriptive statistics for all study variables for males and females are displayed in Table 3.1. Females reported significantly higher levels of Anxiety Disorder Symptoms and Depressive Symptoms than males at both annual assessments, while no sex differences were found for Aggressive Behavior. Females also reported higher Sadness levels than males, but males and females did not differ on the Levels of Happiness, Anger, and Anxiety. Finally, females showed higher levels of Variability in Happiness and Sadness than males.

Zero-order Correlations Between all Study Variables

Most zero-order correlations between study variables were statistically significant and in the expected direction (see Table 3.2). A few results are highlighted. First, the levels of the three negative emotions (happiness, anger, and anxiety) showed small-to-medium sized positive correlations with the three problem areas, while Happiness Level correlated negatively with the three problem areas. Second, the variability of the three negative emotions and happiness showed small-to-medium positive correlations with Anxiety Disorder Symptoms, Depressive Symptoms, and Aggressive Behavior. Third, the level of the negative emotions showed large positive associations with variability of the same emotions, while the level of happiness correlated negatively with Happiness Variability. Finally, anger, anxiety, and sadness level all correlated strongly and negatively with each other, but strongly and positively with happiness level. The variabilities of all four emotions showed strong positive associations.
### Table 3.1

*Means and Standard Deviations of Anxiety, Depression, Aggression, and Emotional Variability for Males and Females*

<table>
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<th></th>
<th>Range</th>
<th>M</th>
<th>SD</th>
<th>Range</th>
<th>M</th>
<th>SD</th>
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<td>19.23</td>
<td>5.49</td>
<td>35.93***</td>
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<td>23-85</td>
<td>41.69</td>
<td>11.94</td>
<td>10.55**</td>
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<td>Depressive Symptoms T5</td>
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<td>23-80</td>
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<td>18.90</td>
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<td>6.67</td>
<td>6.88</td>
<td>6.52*</td>
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</table>

*Note.* T1 = Annual home assessment at age 13. T5 = Annual home assessment at age 14. * *p < .05. ** *p < .01. *** *p < .001.
Table 3.2

Bivariate Correlations Between Study Variables

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<td>-.42</td>
<td>-.45</td>
<td>-.32</td>
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<td>.59</td>
<td>.63</td>
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<tr>
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<td>Sadness Level</td>
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<td>.35</td>
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<td>.34</td>
<td>.40</td>
<td>.30</td>
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<td>.71</td>
<td>.48</td>
<td>.64</td>
<td>.53</td>
<td>.72</td>
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</table>

Note. All correlations are significant with $p < .05$ (one-tailed). T1 = Annual home assessment at age 13. T5 = Annual home assessment at age 14.
CHAPTER 3
The Role of Emotion Level in the Development of Psychopathology

Possible effects of the levels of the four emotions in the development of psychopathology were investigated with the models described above. These models were fully saturated; therefore fit indices are not presented. Results are in Table 3.3. Anxiety Disorder and Depressive Symptoms showed medium stability from age 13 to 14 ($\beta$s = .47 to .53 for Anxiety Disorder, and $\beta$s = .38 - .44 for Depressive Symptoms), while the stability of Aggressive Behavior was high ($\beta$s = .58 - .62). Anxiety Disorder Symptoms significantly predicted levels of all four emotions (negatively for happiness), and levels of three emotions (with anger as the exception) also predicted later Anxiety Disorder Symptoms beyond the prediction from earlier Anxiety symptoms. Tests of indirect effects showed significant effects of happiness level and high anxiety and sadness in the development of Anxiety Disorders Symptoms from T1 to T5. For Depressive Symptoms, the results were highly similar, with the exception that Anger also played a role in the one-year development of Depressive Symptoms. The development of Aggressive Behavior was predicted by levels of anger, anxiety, and sadness, but not happiness.

The Role of Emotional Variability in the Development of Psychopathology

Results of the models testing for the role of variability of the four emotions in the development of Anxiety Disorder, Depressive, and Aggressive Behavior symptoms, were highly similar to the results for the Levels of the four emotions (see Table 3.4). The variabilities of all four emotions contributed significantly to the one year development of Anxiety Disorder Symptoms and Depressive Symptoms. The variabilities of anger and anxiety also predicted the one-year development of Aggressive Behavior, while the variabilities of happiness and sadness did not.

Variability and Levels of Discrete Emotions in the Development of Psychopathology

To test whether emotional level or variability played a bigger role in the development of psychopathology, models were run in which both the level and the variability of the four discrete emotions were included (see Table 3.5). Because neither the level nor the variability of happiness played a role in the development of Aggressive Behavior, models for Aggressive Behavior were
Table 3.3

The Role of the Levels of Discrete Emotions in the 1-year Development of Anxiety, Depression, and Aggressive Behavior

<table>
<thead>
<tr>
<th>PP</th>
<th>Emotion</th>
<th>Direct effects PP T1 → PP T5</th>
<th>Indirect effect PP T1 → EL</th>
<th>Direct effects EL → PP T5</th>
<th>Indirect effect PP T1 → EL → PP T5</th>
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</thead>
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<td>β</td>
<td>B</td>
<td>SE</td>
</tr>
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<td>Anxiety T5</td>
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<tr>
<td></td>
<td>Angry</td>
<td>.55</td>
<td>.05</td>
<td>.53***</td>
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<td>.05</td>
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<td></td>
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<td>.05</td>
<td>.51***</td>
<td>.22</td>
</tr>
<tr>
<td>Depression T5</td>
<td>Happy</td>
<td>.42</td>
<td>.06</td>
<td>.42***</td>
<td>-.13</td>
</tr>
<tr>
<td></td>
<td>Angry</td>
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<td>.06</td>
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<td>.38***</td>
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<tr>
<td></td>
<td>Sad</td>
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<tr>
<td>Aggressive Beh T5</td>
<td>Happy</td>
<td>.68</td>
<td>.05</td>
<td>.62***</td>
<td>-.21</td>
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<tr>
<td></td>
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<td>.59***</td>
<td>.21</td>
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<tr>
<td></td>
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<td>.05</td>
<td>.60***</td>
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</tbody>
</table>

# Table 3.4

*The Role of the Variability of Discrete Emotions in the 1-year Development of Anxiety, Depression, and Aggressive Behavior*

<table>
<thead>
<tr>
<th>PP</th>
<th>Emotion</th>
<th>Direct effects PP T1 → PP T5</th>
<th>Direct effects PP T1 → EV</th>
<th>Direct effects EV → PP T5</th>
<th>Indirect effect PP T1 → EV → PP T5</th>
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<td>B</td>
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<td>.51</td>
<td>.05</td>
<td>.50***</td>
<td>.30</td>
</tr>
<tr>
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<td>.53</td>
<td>.05</td>
<td>.51***</td>
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<td>.05</td>
<td>.50***</td>
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<tr>
<td></td>
<td>Sad</td>
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<td>.05</td>
<td>.50***</td>
<td>.36</td>
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<td>.05</td>
<td>.48***</td>
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<td>.71</td>
<td>.05</td>
<td>.64***</td>
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</tr>
<tr>
<td></td>
<td>Angry</td>
<td>.68</td>
<td>.05</td>
<td>.61***</td>
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<td></td>
<td>Anxious</td>
<td>.67</td>
<td>.05</td>
<td>.61***</td>
<td>.27</td>
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<tr>
<td></td>
<td>Sad</td>
<td>.69</td>
<td>.05</td>
<td>.63***</td>
<td>.33</td>
</tr>
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</table>

limited to the three negative emotions. Both Anxiety Disorder Symptoms and Depressive Symptoms showed significant positive links to variability of all emotions, and to anger, anxiety, and sadness levels. Further, Anxiety Disorder and Depressive Symptoms showed significant negative links to happiness level. However, Anxiety Disorder Symptoms were predicted by the variability, but not the levels of the four discrete emotions. Depressive Symptoms on the other hand, were predicted by the levels of happiness (negative association) and anger, anxiety and sadness (positive associations), but not by their variability. The indirect effects reflect these findings: the indirect pathways from Anxiety Disorder Symptoms at age 13 to Anxiety Disorder Symptoms at age 14 via EV (but not emotion levels) were generally statistically significant, as were the indirect pathways from Depression T1 to Depression T5 via emotion levels (but not EV). Significant indirect effects in the development of Aggressive Behavior were found for the levels of Anger and Sadness, but not for Anxiety, and not for EV. The sizes of the significant indirect effects ranged from .01 to .14, indicating small effects.

Gender

To test for possible gender differences in the role of emotion dynamics for the development of psychopathology, multigroup models were specified by gender for the final models including level and variability of emotions. A fully constrained model (i.e., a model in which all estimates were constrained to be equal for male and females adolescents) was compared to models in which individual path estimates were freed one at a time (i.e., allowed to vary across gender). Few gender differences were found. Specifically, Anxiety Disorder Symptoms at age 13 predicted Variability in Happiness and Anger more strongly for male ($\beta = .31$ and $\beta = .30$, both $p < .001$ for Happiness and Anger Variability respectively) than for female participants ($\beta = .21$ and $\beta = .20$, both $p < .01$ for Happiness and Anger Variability respectively), $\Delta \chi^2(1) = 5.40$ for Happiness and $\Delta \chi^2(1) = 5.09$, for Anger Variability, both $p < .05$. Conversely, Depressive Symptoms predicted Happiness Variability for female ($\beta = .30, p < .001$), but not for male ($\beta = .09, \text{ns}$) participants, $\Delta \chi^2(1) = 4.89, p < .05$. Anxiety and Sadness Levels predicted age 14 Aggressive Behavior for female ($\beta = .31$ and $\beta = .23$ for Anxiety and Sadness Levels respectively, both $p < .01$), but not male ($\beta = .06$ and $\beta = .04$ for Anxiety and Sadness Levels respectively, both $p > .05$) adolescents, $\Delta \chi^2(1) = 4.34$ for Anxiety Level and $\Delta \chi^2(1) = 4.33$, for Sadness Level, both $p < .05$. 


### Table 3.5

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<th>PP T1 → EV/EL</th>
<th>EV/EL → PP T5</th>
<th>PP T1 → EV/EL → PP T5</th>
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<td>.52**</td>
<td>.33/.20</td>
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ADOLESCENT EMOTION DYNAMICS

DISCUSSION

The present study examined the role of adolescent emotional dynamics in the development of psychopathology from age 13 to 14. Results showed that individual differences in the dynamics of happiness, anger, anxiety, and sadness partly account for the development of adolescent anxiety disorder and depressive symptoms, and that the dynamics of the negative emotions, but not happiness, partly account for the development of aggressive behavior. Taking our hypothesis into account, these findings revealed three overarching results. First, most links between the four basic emotions and the three problem areas were non-specific. That is, the dynamics of the four emotions were similarly related to the forms of psychopathology studied. This was particularly true for anxiety disorder and depressive symptoms, while associations between emotion dynamics and aggressive behavior were a bit more specific. In particular, anger played a more consistent role than the other emotions in aggressive behavior. Second, the variability, but not the levels of emotions predicted the development of anxiety disorder symptoms, while the levels, but not the variability, of emotions predicted depressive symptoms and aggressive behavior, when effects of both variability and levels of an emotion were studied in one model. Third, despite the fact that females had higher levels of anxiety and depression, the role of emotional dynamics in the development of psychopathology was similar for both sexes; with the main exception that aggressive behavior was predicted by levels of sadness and anxiety for female, but not for male adolescents.

Building on the assumption that elevated levels of negative emotions, diminished levels of happiness, and elevated emotional variability are all indices of emotion dysregulation, the results add to a growing body of evidence showing that emotion dysregulation predicts symptoms of anxiety, depression and aggressive behavior in children and adolescents (Beauchaine, Gatzke-Kopp, & Mead, 2007; Bosquet & Egeland, 2006; Yap, Allen & Ladouceur, 2008). These findings are consistent with theory proposing that emotion dysregulation underlies the development of psychopathology (Bradley, 2000). Our results are also consistent with earlier cross-sectional research linking adolescent negative emotions and emotional variability with symptoms of depression and problem behavior in adolescents (Larson et al., 1990; Silk et al., 2003).

The finding that the dynamics of happiness, anger, anxiety, and sadness contributed in mostly non-specific ways to different problem areas, may be surprising from the view of functional continuity between emotions and psychopathology. However, our findings are consistent with the notion of a general emotion dysregulation factor (Silk et al., 2003; Steinberg & Avenevoli, 2000), and are in line with findings that most forms of psychopathology include dysregulation in more than one discrete emotion. For instance, increased anger (Moscovitch, McCabe, Antony, Rocca, & Swinson, 2008), and diminished happiness (Kashdan, 2007) have been implicated in the anxiety disorders, and depression involves the dysregulation of both positive and negative emotions (Gross & Levenson, 1997). The fact that, in contrast to the current findings, some studies report specific links between discrete emotions and forms of psychopathology in adolescents (Keltner et al., 1995) and children (e.g., Eisenberg, Cumberland, Spinrad, Fabes, Shepard, Reiser et al., 2001; Rydell, Berlin, & Bohlin, 2003), may be due to differences in the assessment of emotions. While we studied
the adolescents’ own emotional experience, the above studies used videotaped observations of emotional displays (Keltner et al., 1995) or observer ratings of emotions (Eisenberg et al., 2001; Rydell et al., 2003). For an emotional experience to be observable, it must have been translated into some form of behavior (e.g., facial or verbal expression). The way in which an internal experience is expressed may very well be an important factor in determining the specific form of psychopathology that one develops. For instance, when both anger and anxiety are often experienced, but the expression of anger is usually suppressed, while anxiety is shown more freely, findings based on observations of these expressions may suggest that the individual is more likely to develop an anxiety disorder than antisocial personality disorder. As our results show, however, at an experiential level, high levels and variability of negative emotions (and low levels of positive emotions), are implicated in non-specific ways in adolescent mental health problems. This suggests that the specificity hypothesis does not hold for experiential data. Future research needs to explicitly address the question of what determines emotional expression – candidate variables may be intrapersonal variables, such as behavioral inhibition versus behavioral activation (Gray, 1982) as well as social variables, such as cultural and gender specific display rules for emotions (e.g., Brody, 2000).

This non-specificity may have implications for our understanding of comorbidity. Anxiety disorders, mood disorders, and disruptive disorders show strong co-occurrence in childhood and adolescence. Caron and Rutter (1991) suggest that one possible reason for overlap between two disorders is that they share the same risk factor or factors. This possibility arises from the fact that many psychiatric disorders are multifactorial in origin and that many causal factors are not diagnosis-specific. However, it is not known whether the shared risk factors mechanism of several factors mentioned by these authors (e.g., temperamental variables, family adversity) does in fact account for patterns of comorbidity. It may well be that emotional dysregulation is one of the risk mechanisms underlying multiple disorders, including the ones addressed in the present study.

Some specificity in links between the type of emotion dynamics and forms of psychopathology was still found: increased variability in all emotions was specifically related to anxiety disorder symptoms, while elevated levels of negative emotions and diminished levels of happiness, but not their variability were specifically related to depression. This finding is consistent with reports that the development of anxiety and depressive disorder symptoms of adolescents from the general community, though parallel, occurs as two distinct disorders (Hale, Raaijmakers, Muris, van Hoof, & Meeus, 2009), and may have implications for our understanding of depression versus anxiety disorder. What differentiates individuals at risk for depression from individuals at risk for anxiety disorders, may be that the former experience “learned helplessness” (Abramson, Seligman, & Teasdale, 1978), while the latter alternate between hope and fear. Thus, depression may be characterized by the belief that there is little that one can do about one’s situation in general, and about one’s affective state in particular. In contrast, inherent in the excessive worry experienced by individuals at risk for anxiety disorders, may be not only the fear that things will not turn the way one wishes, but also the hope that they will – leading to rapid changes in emotional states. This interpretation, though speculative, is consistent with findings that symptoms of anxiety often
precede symptoms of depression (Cole, Peeke, Martin, Truglio, & Seroczynski, 1998). While there is still hope in anxiety, it is lost in depression, as reflected in the present study by heightened variability of emotional states in anxiety, and rather high and stable levels of negative emotions, and low happiness in depression. Future research may investigate the role of emotional dynamics in the development of anxiety and depression using longer timeframes and including more cognitive predictors such as hope and helplessness.

While some associations between anxiety and sadness dynamics on the one hand, and the development of aggressive behavior on the other hand, were found, anger clearly played the most prominent role in adolescents’ aggressive behavior. Compared to the internalizing problems, aggressive behavior thus appears less emotional. This is in line with research showing that self-reported emotion dysregulation (Neumann et al., 2010) explains more of the variance of internalizing than of externalizing problems. However, it is still possible, that certain forms of externalizing problems, which were not studied in the present investigation, are more emotional. It has been suggested that reactive aggression is more emotional than proactive aggression, and it has indeed been found that reactive aggression is related to poorly regulated responses to emotional stimuli (Vitaro, Brendgen, & Tremblay, 2002), while proactive aggression is related to callousness and emotional shallowness (Frick, Cornell, Bodin, Dane, Barry, & Loney, 2003; Marsee & Frick, 2007).

In addition to sex differences in the levels of emotion variability and anxiety and depression, some sex differences in associations between psychopathology and emotion dynamics were found. Anxiety disorder symptoms predicted happiness and anger variability more strongly for males, whereas depressive symptoms predicted happiness variability for females only. Further, sadness and anxiety predicted aggressive behavior for female, but not male adolescents. These results suggest that emotions do play different role for the development of male versus female psychopathology development. However, since the findings are somewhat inconclusive, replication across different samples is needed to adequately test the hypothesis that emotion dynamics relate differently to male and female development.

Some limitations of this study need to be mentioned. First, the sample consisted of white, mostly middle-class Dutch adolescents; thus caution should be exercised in generalizing the results to the general adolescent population. It is also unclear how the present results generalize to clinical populations. Further, the sample represented only a small age-range of adolescence. Plus, although the sample size was reasonably large, and we used repeated measures, the statistical power of this study may have been too limited to detect potential sex differences in associations. Another limitation is that EV and internalizing and externalizing problems were assessed by adolescent self-report only. Associations between EV and psychopathology might have been inflated by shared source variance. Also, EV is generally assessed over several days, with multiple assessments of emotional intensity per day (e.g., Larson et al., 1980, Silk et al., 2003). In the present study, EV was indexed as EV over the course of three one-week periods, with one assessment of emotional intensity per day. It would be interesting to see if the present results replicate with EV measured intensively over the course of one or several days. However, again, our findings were in accordance
CHAPTER 3

with theoretical assumptions and earlier empirical studies. Therefore, not having a within-day variability measure of EV did not seem to overly affect our results.

Clearly, especially with regard to the variability of emotions, more work is needed to understand its nature and development in adolescence, and how it relates to emotional reactivity to stressors, and emotion regulation strategies. For instance, how do emotional reactivity and emotion regulation strategies interact, i.e., can some highly emotionally reactive individuals modulate their emotions in such effective ways that emotion dysregulation does not result? Further, concerning the link to psychopathology, an important issue would be: when does EV become so strong that it becomes a risk for the development of emotional problems? Clearly, emotions and moods that resist change are maladaptive as well (Cole & Hall, 2008). Future studies of the sources and consequences of EV may also want to include pubertal status, as pubertal status has been related to both depressive and aggressive affect (Brooks-Gunn, Graber, & Paikoff, 1994), and recent evidence shows that pubertal status and emotional reactivity to experimentally induced stress interact in the prediction of internalizing symptoms (Leen-Feldner, Reardon, & Zvolensky, 2007).

In sum, results of the present study imply that when studying the emotional underpinnings of (internalizing) psychopathology, researchers may want to focus less on the specific emotions, and more on the general form the dysregulation takes, as indicated by high levels of negative, and low levels of positive emotions, or highly variable emotions. In addition to application to research and theory, the study of basic emotional processes in adolescence is also informative for prevention and intervention efforts, as early forms of emotion dysregulation can indicate risk for psychopathology (Cole & Hall, 2008). An important message regarding intervention from the present study, then, is to focus on the entire emotional spectrum, rather than on discrete emotions.
Emotion Regulation Difficulties in Adolescents: Associations with Parenting and Mother-Adolescent Relationship Quality

Anna Neumann
Hans M. Koot

A German version of this manuscript is submitted for publication.
Difficulties in emotion regulation (ER) have been said to underlie most forms of psychopathology (Bradley, 2000). Although in adolescence the prevalence of emotional and behavioral problems increases (Silk, Steinberg, & Morris, 2003), research on ER in adolescence is scarce. While several factors are thought to underlie ER, including neurophysiology, temperament/personality, cognitive, and social factors (for a review see Morris, Silk, Steinberg, Myers, & Robinson, 2007), especially in early childhood, parents are acknowledged to play a major role in their child’s emotional development (e.g., Kopp, 1989). A similar role for parents has been hypothesized for adolescent ER, but this role has rarely been investigated. The goals of the present investigation are to explore the associations of parenting (Study 1) and aspects of the parent-adolescent relationship (Study 2) with adolescent ER difficulties, and the potential moderating role of adolescent gender in these associations.

In keeping with the functional approach to emotions (e.g., Thompson, 1994), ER is understood here as “… the extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying reactions to accomplish one’s goals” (Thompson, 1994; pp. 27-28). More specifically, in this study ER is defined as a multidimensional construct involving: (a) the awareness, understanding, and acceptance of emotions; (b) access to adaptive strategies for modulating the intensity and/or duration of emotional responses; and (c) the ability to control behaviors, including inhibiting impulsive behaviors and engaging in goal-directed behaviors, when experiencing emotional distress (see Gratz & Roemer, 2004). Perceived deficits in any or all of these dimensions are considered indicative of ER difficulties. It has been shown that these difficulties can be differentiated, and are linked to internalizing and externalizing problems in adolescents (Neumann, van Lier, Gratz, & Koot, 2010). Given the very limited research on direct links between parenting and adolescent ER, hypotheses on this link are mainly derived from evidence on the association between parenting and adolescent behavioral/emotional problems.

Several studies have shown that parents play a significant role in the development of ER in children. Parents socialize their children’s ER in a number of ways, including direct instruction, modeling, and their reactions to their children’s emotional displays (Saarni, 1999). Recent research has demonstrated that direct emotion socialization remains important during adolescence (e.g., Klimes-Dougan, Brand, Zahn-Waxler, Usher, Hastings, Kendziora, et al., 2007). Nevertheless, for adolescents, who are thought to be able to regulate their emotions more independently from parents than children (Kopp, 1989), it has been suggested that more indirect sources, such as general parenting behaviors, and the general quality of the parent-adolescent relationship become relatively more important as influences on the quality of ER (Zahn-Waxler, Klimes-Dougan, Kendziora, 1998).

Three generally agreed upon core dimensions of parenting (e.g., Barber, 1996), are ‘acceptance versus rejection’, ‘firm versus lax behavioral control’, and ‘psychological control versus psychological autonomy granting’. Parental control gains special importance in adolescence, when a major developmental task of the adolescent is to achieve more autonomy from the parent. Parental behavioral control is aimed at controlling the adolescent’s behavior (Barber, Olsen, & Shagle, 1994), e.g., to prescribe a curfew. Low parental behavioral control has consistently been
linked to heightened levels of child externalizing behaviors (for a review see Barber & Harmon, 2002). To our knowledge, no studies have directly examined associations between parental behavioral control and adolescent ER. However, it seems likely that these links are rather weak, for instance, because by definition, behavioral control is aimed at the adolescent’s behavior, not at the adolescent’s internal emotional world (Barber et al., 1994).

By contrast, parental psychological control is aimed at controlling the child’s psychological world (Barber et al., 1994). High psychologically controlling parents wish to control everything for their child, including the way they should feel. When dissatisfied with their child, these parents react by (threat of) withdrawing love or guilt induction. These parental behaviors likely interfere with child emotional development by precluding possibilities for the child to freely express emotions and to learn to be accepting of emotional responses. Parental psychological control has been positively linked to both internalizing and externalizing problems (Barber & Harmon, 2002), and to difficulties with self-regulation (Moilanen, 2005). We expect high psychological control to be related with increased difficulties in ER.

Over time, the relation between parenting and child behaviors becomes increasingly bidirectional (Kuczynski, 2003). While parents still need to exert certain degrees of power over their adolescent child, support and rejection are likely to be mutually determined by the adolescent and his/her parent. In general terms, if the parent-adolescent relationship is characterized by trust, support and warmth, adolescents are probably more likely to turn to their parents for support in emotional situations. In such a climate, adolescents can learn that (novel) emotional situations need not be overwhelming, but can be handled. In addition, when trust in the relationship is high, adolescents will be more likely to discuss emotional experiences with their parents, which is likely to increase such ER dimensions as emotional awareness and clarity. In contrast, when levels of conflict and negative interactions in the parent-adolescent relationships are high, negative affect might escalate in interactions between adolescents and parents, and chances for learning effective ER are precluded. The adolescent might also hide his/her feelings from his/her parents in the first place, thereby also precluding opportunities for learning adaptive ER. Moreover, heightened levels of negative interactions in the parent-adolescent relationship might indicate ER difficulties on the side of the parent, and provide the adolescent with a model of ineffective ER, such as poor impulse control. Thus, linking ER difficulties with levels of support and negative interaction in the parent-adolescent relationship is expected to yield negative and positive associations with these two relational aspects, respectively.

There exists evidence for gender as a moderating factor in the link between psychosocial factors and developmental outcomes, such as ER. It has been shown that an authoritarian parenting style, which is characterized by low levels of warmth and high levels of both behavioral and psychological control, is related to externalizing problems for boys, and to both externalizing and internalizing problems for girls (e.g., Hart, Newell, & Olsen, 2004). Though indirectly, this suggests that high levels of parental control might be more strongly associated with adverse developmental outcomes, including ER problems, for girls than for boys. In addition, parents teach their girls more relationship-oriented ER strategies, while teaching boys more problem-oriented and instrumental
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ER strategies (e.g., Eisenberg, Cumberland, & Spinrad, 1998). If boys and girls internalize these respective ER strategies in childhood, adolescent females should be more likely than adolescent males to try to regulate their emotions in the context of (close) social relationships. Accordingly, females’ ER competencies are likely to profit more from positive relationships with their parents, while at the same time troubled relationships are likely to pose a greater threat for female adolescents than for male adolescents to develop ER difficulties. Thus, it is likely that gender moderates the link between relationship quality and adolescent ER difficulties, such that the link is stronger for girls than for boys.

In sum, the goals of the present investigation are to explore how parents (specifically mothers) play a role in their adolescent child’s emotional development, and to investigate possible moderating effects of gender. Since parents may influence their adolescent child’s ER in more than one way, two studies are conducted: In Study 1, the focus is on parenting variables, specifically behavioral and psychological control, while Study 2 focuses on aspects of the parent-child relationship, namely negative interaction, and support.

STUDY 1

Study 1 addressed the relation between maternal behavioral and psychological control, and adolescent ER difficulties. We expected that psychological control would be positively related to all dimensions of adolescent ER difficulties, while behavioral control would not, and that the relation between psychological control and ER difficulties would be stronger for girls than for boys.

METHOD

Participants

A sample of 286 students was recruited from three secondary schools in Cologne, Germany (147 boys, 139 girls; mean age 14.99 years, SD = 1.56, range 11 – 19 years). Schools were chosen to represent different levels of secondary education in Germany and thus included students from a Gymnasium (N = 139), a Gesamtschule (N = 119), and a Hauptschule (N = 28). Boys and girls did not differ significantly regarding age, t(283) = .72, p > .05, or the type of school they attended, χ²(N = 285, df = 2) = .38, p > .05. Most of the students were born in Germany (92.3%). Of those students who were not born in Germany, most were born in Poland or Kazakhstan (1.0% each).

Measures

Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). The DERS is a 36-item self-report questionnaire that assesses clinically relevant difficulties in emotion regulation. The questionnaire consists of six subscales, labeled Lack of Emotional Awareness (6 items), Lack of Emotional Clarity (5 items), Impulse Control Difficulties (6 items), Difficulties Engaging in
Goal-directed Behavior (5 items), Nonacceptance of Emotional Responses (6 items), and Limited Access to ER Strategies (8 items). Items are scored on a five-point scale ranging from 1 (almost never) to 5 (almost always). Subscale scores are constructed by summing responses to the corresponding items. Reliability of the DERS is good: Cronbach’s alpha has been reported to be .93 for the total scale, and Cronbach’s alphas lay between .80 and .89 for the subscales in a sample of college students (Gratz & Roemer, 2004). Construct and predictive validity have been demonstrated in adults (Gratz & Roemer, 2004). Its factor structure has been confirmed for adolescents, and relations between the DERS scales and behavioral/emotional problems in adolescents have been shown (Neumann et al., 2010). Reliability of the DERS in the present sample is acceptable, with Cronbach’s alphas ranging from .61 (Lack of Emotional Clarity) to .78 (Impulse Control Difficulties) (mean alpha = .72).

Children’s Report of Parent Behavior Inventory-30 (CRPBI-30, Schludermann & Schludermann, 1988). Twenty items of the CRPBI-30, which assess adolescent-perceived maternal psychological and behavioral control were used in the present study. Ten items each refer to Behavioral Control (e.g., “My mother insists that I do exactly what I am told”), and Psychological Control (e.g., “My mother says that if I really cared for her, I would not do things that cause her to worry”). In the present sample reliability of the Psychological Control ($\alpha = .75$) and Behavioral Control scales ($\alpha = .72$) was acceptable. Evidence for the convergent and discriminant validity of the 108 item version of the CRPBI has been reported (Schwarz, Barton-Henry, & Pruzinsky, 1985). The CRPBI-30 shows meaningful links to adolescent impulse control and behavioral/emotional problems (Moilanen, 2005).

Procedure

For the purpose of the present study, the DERS and the CRPBI were translated to German. The first author translated the questionnaires to German. Then, the German translations were back translated into English by a native German speaker, who teaches English at a secondary school in Germany. Inconsistencies were resolved by discussion and resulting changes were made.

In preparation of the study adolescents’ parents received written information about the investigation and the possibility to disallow their child’s participation in the study. Adolescents themselves were informed about the study in their classrooms, and completed the questionnaires after having filled in an informed assent form. All students completed the DERS, plus the CRPBI or the NRI (employed in Study 2). Participants always completed the DERS first. Classes in each school were randomly assigned to subsets of measures, while insuring that the measures were equally distributed across students of different schools and grade levels. Every adolescent received a small gift in return for their participation.
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RESULTS

Preliminary Analyses

Because age showed significant associations with three DERS scales \((r = .28, r = .30, \text{ and } r = .20, p < .01\) between age and Lack of Emotional Awareness, Lack of Emotional Clarity, and Impulse Control Difficulties, respectively) and we found a significant multivariate effect of secondary school on DERS scales, Pillai’s trace: \(F(12, 554) = 10.98, p < .001\) we controlled for age and educational level in all subsequent analyses.

Relations Between Parenting and Adolescent ER Difficulties

As shown in Table 4.1, maternal Psychological Control was positively and significantly related to three out of the six DERS scales for males, namely to Lack of Emotional Clarity, Impulse Control Difficulties, and Nonacceptance of Emotional Responses. Correlations were in the small-medium range (Cohen, 1988; range = .25 - .31; median = .27). Maternal Behavioral Control was not significantly related to any of the DERS scales for male participants. For girls, maternal Psychological Control was significantly and positively related to four DERS scales, specifically to Lack of Emotional Clarity, Impulse Control Difficulties, Difficulties Engaging in Goal-directed Behavior, and Limited Access to ER Strategies. In addition, for female adolescents, maternal Behavioral Control was significantly and positively related to Limited Access to ER Strategies, and to Difficulties Engaging in Goal-directed Behavior. All correlations were small (range = .18 - .29; median = .22).
Table 4.1
Partial Correlations Between Parenting and Adolescent-Mother Relationship Characteristics and Adolescent ER Difficulties for Male and Female Adolescents

<table>
<thead>
<tr>
<th></th>
<th>Awareness</th>
<th>Clarity</th>
<th>Impulsivity</th>
<th>Goals</th>
<th>Nonacceptance</th>
<th>Strategies</th>
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<td>.16</td>
<td>.10</td>
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<td>.25**</td>
<td>.14</td>
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<td></td>
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<tr>
<td>Behavioral Control</td>
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<td>.07</td>
<td>.19*</td>
<td>.05</td>
<td>.19*</td>
</tr>
<tr>
<td>Psychological Control</td>
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<td>.20*</td>
<td>.18*</td>
<td>.29***</td>
<td>.14</td>
<td>.29***</td>
</tr>
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<td><strong>Adolescent-Mother Relationship</strong></td>
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<td>.21*</td>
<td>.28**</td>
<td>.24*</td>
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<td>.08</td>
</tr>
<tr>
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<td>-.20*</td>
<td>.05</td>
<td>-.20*</td>
<td>-.07</td>
<td>-.16</td>
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<tr>
<td>Female adolescents (N = 86)</td>
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<td></td>
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<tr>
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<td>.54***</td>
<td>.43***</td>
<td>.26**</td>
<td>.45***</td>
<td>.61***</td>
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<tr>
<td>Support</td>
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<td>-.66***</td>
<td>-.42***</td>
<td>-.28*</td>
<td>-.47***</td>
<td>-.60***</td>
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</table>

Table 4.2
Hierarchical Regression Analyses Predicting Adolescent Emotion Regulation Difficulties from Gender and Parenting

<table>
<thead>
<tr>
<th>Step</th>
<th>Predictors</th>
<th>Awareness</th>
<th>Clarity</th>
<th>Impulsivity</th>
<th>Goals</th>
<th>Nonacceptance</th>
<th>Strategies</th>
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<td></td>
<td></td>
<td>$\beta$</td>
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<td>$\beta$</td>
<td>$\Delta R^2$</td>
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<td>$\Delta R^2$</td>
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<td>.00</td>
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<tr>
<td></td>
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<td>.13*</td>
<td>.13*</td>
<td>.12</td>
<td>.08</td>
<td>.07</td>
</tr>
<tr>
<td></td>
<td>Edu. Level</td>
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<td>.52***</td>
<td>.18**</td>
<td>-.07</td>
<td>-.05</td>
<td>-.03</td>
</tr>
<tr>
<td>2</td>
<td>Gender</td>
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<td>.00</td>
<td>.01</td>
<td>.00</td>
<td>.02</td>
<td>.00</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3a</td>
<td>Beh. Con</td>
<td>-.04</td>
<td>.00</td>
<td>.03</td>
<td>.00</td>
<td>.08</td>
<td>.01</td>
</tr>
<tr>
<td>3b</td>
<td>Psy. Con</td>
<td>-.06</td>
<td>.00</td>
<td>.18***</td>
<td>.03***</td>
<td>.21***</td>
<td>.04</td>
</tr>
<tr>
<td>4</td>
<td>Interaction effects</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4a</td>
<td>Beh. Con X Gender</td>
<td>.06</td>
<td>.00</td>
<td>.11</td>
<td>.00</td>
<td>.04</td>
<td>.00</td>
</tr>
<tr>
<td>4b</td>
<td>Psy. Con X Gender</td>
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<td>.00</td>
<td>-.06</td>
<td>.00</td>
<td>-.11</td>
<td>.00</td>
</tr>
</tbody>
</table>

EMOTION REGULATION DIFFICULTIES AND MOTHER-ADOLESCENT RELATIONSHIP

Adolescent Gender, Parenting and their Interaction Effects on Adolescent ER Difficulties

To examine adolescent gender (males = 0, females = 1) and parenting effects as well as their possible interactions on adolescent ER difficulties, hierarchical regression analyses were conducted separately for each of the dimensions of ER difficulties. Results are in Table 4.2. The first step of the regression analyses included the control variables age and school, and the second step adolescent gender. In the third step, parenting variables were entered one at a time, and in the fourth step parenting by gender interaction terms were entered one at a time. Parenting variables were centered before interaction terms were built.

Gender appeared to be significantly linked only to Limited Access to ER Strategies. Maternal Behavioral Control was significantly linked to Difficulties Engaging in Goal-directed Behavior, and to Limited Access to ER Strategies, while Psychological Control appeared to be significantly linked to Lack of Emotional Clarity, Impulse Control Difficulties, Difficulties Engaging in Goal-directed Behavior, Nonacceptance of Emotional Responses, and Limited Access to ER Strategies. \( R^2 \)'s ranged from .03 to .07. The Gender X Behavioral Control interaction significantly predicted Limited Access to ER Strategies (\( R^2 = .02 \)). Simple slopes calculated for boys and girls separately, showed that the link between Behavioral Control and Limited Access to ER Strategies is significant for girls (\( \beta = .24, p < .01 \)), but not for boys (\( \beta = .02, p > .05 \)).

STUDY 2

In Study 2, links between the quality of the adolescent-perceived relationship to his/her mother and adolescent ER difficulties, and gender as a possible moderating variable in this relationship, were investigated. We expected high levels of mutual support to be associated with low levels of ER difficulties, and high levels of negative interaction with high levels of ER difficulties. Further, these associations were expected to be stronger for girls.

METHOD

Participants

A sample of 177 students was recruited from the same three secondary schools in Cologne, Germany as for Study 1 (91 boys, 68 girls; mean age 14.64 years, \( SD = 1.50 \), range 12 – 19 years). The sample included students from a Gymnasium (\( N = 84 \)), a Gesamtschule (\( N = 71 \)), and a Hauptschule (\( N = 24 \)). Boys and girls did not differ significantly regarding age, \( t(174) = .81, p > .05 \), or the type of school they attended, \( \chi^2 (N = 177, \text{df} = 2) = 5.43, p > .05 \). Most of the students were born in Germany (89.9%). Of those students who were not born in Germany, most were born in Turkey, Russia, or Moldavia (1.1% each).
CHAPTER 4

Measures

**Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004).** See Study 1.

**Network of Relationships Inventory (NRI; Furman & Buhrmester, 1985).** The 15 items of the NRI, which were employed in the present study assess adolescents’ perception of their relationship to their mother along the dimensions Negative Interaction (6 items; e.g., “My mother and I get annoyed with each other’s behavior”), and Support (9 items; e.g., “I share my secrets and private feelings with my mother”). Participants rated each statement on a five-point scale, where 1 = “never” and 5 = “always”. Higher scores on the subscales represent higher levels of negative interaction between adolescent and mother, and higher levels of perceived maternal support, respectively. The reliability of the NRI scales was supported in a sample of 11-13 year-olds, with Cronbach’s alphas around .80 (Furman & Buhrmester, 1985). In the present sample, Cronbach’s alphas were high for both scales (α = .92 for Negative Interaction, α = .88 for Support).

**Procedure**

The NRI was translated to German for the purpose of this study, following the same procedure as for the translation of the DERS and the CRPBI for Study 1. The procedure of data collection for Study 2 was the same as the procedure for Study 1.

**RESULTS**

**Preliminary Analyses: Effects of Age and Educational Level**

Because age showed a significant association with three DERS scales (r = .28, for Lack of Emotional Awareness, r = .30 for Lack of Emotional Clarity, both ps < .001, and r = .20 for Impulse Control Difficulties, p < .01), and since we found a significant multivariate effect of secondary school on DERS scales, Pillai’s trace: F(12, 554) = 10.98, p < .001 we controlled for age and educational level in all subsequent analyses.

**Relation Between ER Difficulties and the Mother-Adolescent Relationship**

Results are in Table 4.3. For male adolescents, Negative Interaction was positively associated with four DERS scales, namely Lack of Emotional Awareness, Lack of Emotional Clarity, Impulse Control Difficulties, and Difficulties Engaging in Goal-directed Behavior (range = .21 - .32; median r = .26). Support appeared to be negatively associated with Lack of Emotional Clarity and Difficulties Engaging in Goal-directed Behavior (both rs = -.20).

For female adolescents, Negative Interaction was found to be positively correlated with all dimensions of ER difficulties (range = .23 - .61; median r = .42), while Support correlated...
Adolescent Gender, Adolescent-Mother Relationship, and their Interaction Effects on Adolescent ER Difficulties

To examine the effects of gender, the mother-adolescent relationship, and their possible interactions on adolescent ER difficulties, hierarchical linear regression analyses were conducted separately for all scales of the DERS in three steps following the procedure described in Study 1. As in Study 1, all analyses were controlled for adolescent age and educational level. A main effect for gender was found only for Limited Access to ER Strategies, indicating higher scores for females. Significant main effects of Negative Interaction ($R^2$s = .06 - .14) and Support ($R^2$s = .04 - .20) were revealed for all DERS scales.

Significant Gender X Negative Interaction effects were found for three DERS subscales, i.e. Lack of Emotional Clarity ($R^2 = .03$), Nonacceptance of Emotional Responses ($R^2 = .04$), and Limited Access to ER Strategies ($R^2 = .08$). Significant Gender X Support effects were found for the same three scales, plus for Impulse Control Difficulties ($R^2$s = .04 - .05). Results of tests for simple slopes by gender showed that the links between Negative Interaction and Support on the one hand and ER difficulties on the other hand, are significant for girls only, or are at least stronger for girls than for boys. Beta’s for these links were as follows: for Negative Interactions and Lack of Emotional Clarity: $\beta = .15$ for boys, $\beta = .53$ for girls; Nonacceptance of Emotional Responses: $\beta = .02$ for boys, $\beta = .45$ for girls; Limited Access to ER Strategies: $\beta = .03$ for boys, $\beta = .61$ for girls, all $p$s > .05 for boys and < .001 for girls; for Support and Lack of Emotional Clarity: $\beta = -.22$ for boys, $p < .05$ and $\beta = -.62$ for girls, $p < .001$; Impulsivity: $\beta = .05$ for boys, $p > .05$ and $\beta = -.41$, $p < .001$ for girls; Limited Access to ER Strategies: $\beta = -.19$, $p > .05$ for boys and $\beta = -.61$, $p < .001$ for girls.
## Table 4.3

### Hierarchical Regression Analyses Predicting Adolescent Emotion Regulation Difficulties from Gender and Relationship Variables

| Step | Predictors | Awareness | | Clarity | | Impulsivity | | Goals | | Nonacceptance | | Strategies |
|------|------------|-----------|---|---------|---|----------|---|---------|---|----------------|---|
|      |            | $\beta$  | $\Delta R^2$ | $\beta$  | $\Delta R^2$ | $\beta$  | $\Delta R^2$ | $\beta$  | $\Delta R^2$ | $\beta$  | $\Delta R^2$ |
| 1    | Control variables |  | .00 | | .00 | | .02 | | .01 | | .00 | | .00 |
|      | Age        | .00      | -.01 | | -.03 | | .05 | | -.06 | | .02 | | .00 |
|      | Educational Level | -.02 | | -.04 | | .15 | | -.08 | | .04 | | -.04 | |
| 2    | Gender     | -.05 | | .00 | | .12 | | .01 | | .06 | | .00 | | .14 | | .02 | | .05 | | .00 | | .19* | | .03 |
| 3    | Relationship |  |  |  |  |  |  |  |  |  |  |  |  |  |
| 3a   | NI          | .28*** | | .08*** | | .38*** | | .13*** | | .36*** | | .12*** | | .24** | | .06** | | .28*** | | .07*** | | .39*** | | .14*** |
| 3b   | Support    | -.28*** | | .08*** | | -.44*** | | .20*** | | -.21** | | .04** | | -.24** | | .06** | | -.30*** | | .09*** | | -.42*** | | .17*** |
| 4    | Interaction effects |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| 4a   | NI X Gender | -.23 | | .00 | | .63** | | .03** | | .30 | | .01 | | .06 | | .00 | | .69** | | .04** | | .97*** | | .08*** |
| 4b   | Support X Gender | -.22 | | .00 | | -.65** | | .04** | | -.68** | | .04** | | -.10 | | .00 | | .62* | | .04* | | -.73** | | .05 |

**Note.** Awareness = Lack of Emotional Awareness. Clarity = Lack of Emotional Clarity. Impulsivity = Impulse Control Difficulties. Goals = Difficulties Engaging in Goal-directed Behavior. Nonacceptance = Nonacceptance of Emotional Responses. Strategies = Limited Access to Emotion Regulation Strategies. NI = Negative Interaction. Main effects of Behavioral Control and Psychological Control and their Interactions have been tested separately in steps 3a and 4a, and 3b and 4b, respectively. * $p < .05$, **$p < .01$, *** $p < .001$. $N = 175$. 

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The results of the two present studies suggest that adolescents’ emotion regulation difficulties are related to the context of the mother-adolescent relationship. Study 1 showed that parenting variables, especially psychological control are linked to adolescent ER difficulties, while Study 2 showed that relationship variables, such as negative interaction and support are related to adolescent ER difficulties. In both studies stronger relations between these variables were found for girls than for boys.

As expected, maternal control is related to ER difficulties in 11-19 year olds. Results highlight the importance of distinguishing between behavioral and psychological control, as only the latter showed consistent associations with adolescent ER difficulties. It seems that, when mothers are overly involved in their adolescents’ psychological world, adolescents experience heightened levels of problems with the regulation of their emotions, possibly due to the fact that they have not learned to regulate their emotions in sufficiently independent ways. Indications that psychological control is related to adolescent ER difficulties are in line with research demonstrating that heightened levels of psychological control are associated with poorer impulse control (Moilanen, 2005), internalizing problems and certain externalizing behaviors, such as aggressive defiant behaviors (Barber & Harmon 2002; Moilanen, 2005; Silk et al., 2003).

Whether mothers engage in high levels of behavioral control seems to matter little for adolescent ER difficulties. Correlational and regression analyses revealed only two weak associations between behavioral control and dimensions of ER difficulties (difficulties engaging in goal-directed behavior, and limited access to ER strategies), and these reached significance only for girls. Nevertheless, results suggest that for female adolescents, too much maternal behavioral control interferes with the development of independent ER. It seems likely that results would be different for younger children. In childhood, reasonable levels of behavioral control might enhance the child’s feelings of security, including emotional security, thereby providing the child with an environment in which adaptive ER can be learned. Our results imply that strong maternal behavioral control that is still present in adolescence may result in the opposite.

Converging with our hypotheses, perceived support and levels of negative interaction in the mother-adolescent relationship were strongly related to adolescent ER in Study 2. Negative associations between support and all dimensions of ER difficulties found in this study suggest that high levels of perceived support in the adolescent-mother relationship may assist the adolescent in developing adaptive independent emotion regulatory skills. By contrast, heightened levels of perceived negative interaction in the mother-adolescent relationship were strongly positively related to difficulties in all aspects of ER studied here, suggesting that negative interactions enhance the development of emotional difficulties in adolescents. It is likely that the basis for these associations lies in the early relationship between mother and child, as indicated in the introduction. According to attachment theorists (e.g., Kobak & Sceery, 1988), during the course of development,
the child needs to learn how to regulate his or her emotions in increasingly independent ways, and the relationship between caregiving and child becomes more and more mutual. Child ER, which has been shaped by sensitive caregiving, now in turn shapes the relationship between the child and the caregiver in important ways. And, though the basis for this relationship might date back to early infancy and childhood, it appears that the adolescent’s current perception of his/her relationship with his/her mother remains associated with ER difficulties in adolescence. Several mechanisms might be relevant here, for instance the creation of a context in which it is safe to express emotions, or that mothers who have formed a warm and supportive relationship with their children are more likely than mothers who have not, to be good models of adaptive ER.

Taken together, results of Study 1 and Study 2 suggest that mothers remain powerful agents and partners, in their adolescent child’s emotional development. Though no direct test was possible in this study, it seems that associations between parenting and adolescent ER difficulties are less strong than between social relations and ER difficulties, which is consistent with suggestions that in adolescence, less direct influences on child ER, such as the general quality of the parent-adolescent relationship become relatively more important (Zahn-Waxler et al., 1998).

Consistent with arguments that the interpersonal nature of emotions is more salient to females than to males (Shields, 1995), it appears that mother-adolescent relationship quality is related to female adolescents’ ER difficulties much more profoundly than to male adolescents’. In addition, behavioral control was associated with female adolescents’ ER difficulties only. The effects found here might be the result of gender-typical emotion socialization in childhood. Parents generally teach their daughters more relationship-oriented ER strategies, whereas they tend to teach their sons more active and instrumental ER strategies (Eisenberg et al., 1998). One needs to keep in mind that females reported on same-sex relationship, while males reported on an other-sex relationship. Links between relationship quality and ER difficulties might have been different for boys, if they had reported on their relationship with their father.

A limitation of the present study is that it relies solely on self-report data. This may have resulted in an overestimation of the links between ER difficulties and the mother-adolescent relationship due to shared-method variance (e.g., Fergusson & Horwood, 1987). Another limitation of the present study is its purely cross-sectional nature, which prevents any conclusions regarding the directionality of effects. It seems highly likely that associations between the mother-adolescent relationship and ER difficulties are bidirectional: interactions in the relationship provide a context for adolescents to develop ER skills, while at the same time their ER capabilities impact the nature of these interactions. Similarly, links between psychological and behavioral control and ER difficulties might also be bidirectional. Mothers of adolescents, who have difficulties with the regulation of emotions, might be more inclined than mothers of adolescents with better emotion regulation skills, to interfere with their son’s or daughters’ psychological world and to try and control their behavior. Future longitudinal studies are needed to shed more light on these associations. Finally, it would be informative to investigate fathers’ roles in their children’s emotional development, too.
Despite these limitations, the studies reported here support the notion that aspects of parenting and the adolescent-mother relationship are salient in adolescents’ emotional capabilities. Future studies may demonstrate the influence of parenting and relationship quality on increases and decreases in ER difficulties during adolescence, potential influences of adolescents’ ER difficulties on the development of the relationship with parents, as well as the potential role played by adolescent ER difficulties in the widely acknowledged association between parenting/relationship qualities and the development of behavioral and emotional problems in adolescence.
Developmental Associations Between the Parent-Adolescent Relationship and Adolescent Negative Affect and Psychopathology

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Manuscript submitted for publication
Negative family-relationships are among the strongest predictors of adolescent externalizing and internalizing problems (e.g., Lahey, 2008; Weems & Silverman, 2007). Nevertheless, relatively little is known about the co-development of family relationships and psychopathology in adolescence. Little is also known about potential mechanisms that link the quality of family relationships to the development of adolescent psychopathology. A likely candidate for such a mechanism are adolescents’ experiences of negative affect. Adolescent negative affect is intrinsically related to many forms of psychopathology (American Psychiatric Association [APA], 1994), and often originates from conflictual parent-adolescent social interactions (Laursen, Coy, & Collins, 1998). The main goals of this study are (1) to explore the co-development of the quality of the adolescent-father, and the adolescent-mother relationship, and adolescents’ externalizing and internalizing problems, and (2), to examine individual differences in adolescents’ experiences of negative affect as an explanatory mechanism in the links between the quality of the parent-adolescent relationship and the development of internalizing and externalizing problems.

The Parent-Adolescent Relationship and Internalizing and Externalizing Problems

During adolescence, parental support and negative interactions with parents have been emphasized as influencing adolescent development. Support and negative interactions represent two central features of close social relationships (e.g., Collins & Laursen, 2004). Attachment theory stresses the importance of parental support, including instrumental assistance, companionship, and affection, as a secure base from which to explore the outside world (Bowlby, 1969; Bugental, 2000). Negative interactions, including getting on each others’ nerves, disagreements and conflicts, are regarded as central in close social relationships, because these relationships require the integration of differing opinions and goals (Laursen & Collins, 1994), which frequently is not accomplished without conflicts, especially in the adolescent period.

Longitudinal studies of the parent-adolescent relationship show that negative interactions between parents and adolescents increase from early to mid adolescence (de Goede, Branje, & Meeus, 2009), while support decreases (de Goede, et al., 2009; Hafen & Laursen, 2009). Although the developing parent-child relationship during adolescence may be a field of research in itself, one of the prime reasons that it is studied is because of its hypothesized links with the development of psychopathology in adolescents. In fact, adolescence is a developmental period in which prevalence rates of both internalizing and externalizing problems may increase (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996; Wight, Sepúlveda, & Aneshensel, 2004), and the developing parent-child relationship is theorized to – in part – underlie this increase. For instance, coercion theory predicts close and progressive associations between parent-child interactions and externalizing problems (Reid & Patterson, 1989) in interactions in which parents and offspring match or even surpass each other’s arousal levels. As a consequence of this process, conflicts will escalate (Snyder, Edwards, McGraw, Kilgore, & Holton, 1994; Snyder, Schrepfermann, & St. Peter, 1997). Theoretical notions based on attachment theory hypothesize that attachment insecurity is related to both externalizing and internalizing symptoms through increased feelings of threat and loss and difficulties in
regulating these emotions (e.g., Brumaria & Kerns, 2010; Cummings, Schermerhorn, Davies, Goeke-Morey, & Cummings, 2006).

Despite the theoretical notions, the empirical evidence on developmental links between support from, and conflicts with parents with psychopathology is still limited. Numerous studies report contemporaneous associations between characteristics of the parent-child and parent-adolescent relationship and youth psychopathology (for a review see Collins & Steinberg, 2006). Though several studies (e.g., Branje, Hale, Frijns, & Meeus, 2010; Buist, Deković, Meeus, & van Aken, 2004; Hale, van der Valk, Akse, & Meeus, 2008) studied longitudinal associations between the parent-adolescent relationship and adolescent psychopathology using path models, few have investigated parallel growth between the quality of parent-adolescent relationships and adolescent development. One study, in which adolescents were followed from age 13 to age 15, reported significant associated change between parent-adolescent conflict, and youth internalizing problems, such that increases in conflict were associated with increases in problems (Rueter, Scaramella, Wallace, & Conger, 1999). To our knowledge, parallel growth between parent-adolescent conflict and youth externalizing problems has not been studied. Regarding adolescent perceived support from parents, one study found associated change between perceived support and adolescent internalizing and externalizing problems from age 11 to age 13 years, such that waning support went along with rising problems (Hafen & Laursen, 2009). Taken together, there is some evidence from the limited empirical studies available suggesting that parent-adolescent conflict and the development of psychopathology co-develop over time during adolescence. The first objective of the present study then, was to further explore the co-development of negative interactions and support in the parent-adolescent relationship and adolescent internalizing and externalizing problems.

Adolescents’ Negative Affect, Psychopathology and Parent-Adolescent Relationship

While there is suggestive evidence that the parent-adolescent relationship context is important for adolescent psychopathology development, very little is known on the possible processes underlying the association between the two. Theories propose negative and dysregulated affect as a possible explanatory mechanism (Brumaria & Kerns, 2010; Cummings et al., 2006; Morris, Silk, Steinberg, Myers, & Robinson, 2007). Experienced affect can be regarded as appraisals of events that are meaningful to the individual (Frijda, 1988). Whether an event elicits positive or negative affect depends on how the event relates to an individual’s goals. An event that leads (closer) to the achievement of one’s goal will induce positive affect, while an event that endangers or hinders goal achievement will lead to negative affect (anger, anxiety) (Frijda, 1988). Adolescent-parent conflicts may induce negative affect in adolescents. Conflict with parents may result from the fact that parents do not approve of a goal the adolescent wants to achieve (e.g., staying out late), thereby inducing negative affect. In contrast perceived support may help goal-achievement and may thus be linked to less negative affect. Further, the maintenance of a positive relationship itself is a fundamental human goal (Baumeister & Leary, 1995).
adolescent conflicts or decreases in parental support may lead to experienced threat of loss of the relationship, which may further increase feelings of anxiety, anger and depression.

High levels of prolonged negative affect, in turn, may impede adaptive coping, and, as a consequence, lead to increases in internalizing and externalizing problems. The link between negative affect and internalizing and externalizing problems in adolescence has been theoretically emphasized (e.g., Bradley, 2000; Cole & Hall, 2008). According to the functional approach to emotions, emotions have an important function in organizing behavior (e.g., Thompson, 1994). When emotion regulation becomes constrained, high levels of negative affect may result, which may organize behavior in ways that are problematic (e.g., withdrawal, aggression), ultimately leading to symptoms of psychopathology (Cole & Hall, 2008).

Although frequent parent-adolescent conflicts are thus likely to lead to increased negative affect, perceived parental support is likely to diminish it, and in turn, negative and dysregulated affect is likely to go hand in hand with increased psychopathology, the empirical evidence on this process is limited. Cross-sectional studies suggest that negative affect increases in adolescence (Larson & Lampman-Petraidis, 1989), and there is evidence that intense emotions arise within the parent-adolescent relationship (Collins & Laursen, 2006). Other cross-sectional (Larson, Raffaelli, Richards, Ham & Jewel, 1990; Silk Steinberg, & Morris, 2003) and longitudinal studies (Neumann, van Lier, Frijns, Meeus, & Koot, 2010) showed positive links between high levels of negative affect and psychopathology. Thus, although some evidence exists for each of the proposed links, this evidence mostly comes from cross-sectional studies and no study has addressed all three processes simultaneously. The second objective of this study is therefore to test the longitudinal link between adolescents’ experiences of negative affect with parent-adolescent conflict and parental support, and the role of negative affect in the link between parent-adolescent relationship quality and changes in psychopathology.

**Parent and Adolescent Gender**

The links between parent-adolescent relationship quality, affect, and psychopathology may differ as a function of adolescent and parent gender. With respect to the adolescent gender, it is well known that levels of externalizing problems, such as physical aggression are higher for males than for females across adolescence (Karriker-Jaffe, Foshee, Ennett, & Suchindran, 2008), and female adolescents generally report more internalizing symptoms than males (e.g., Hale, Raaijmakers, Muris, van Hoof, & Meeus, 2008). Further, levels of conflict with parents and perceived support may differ by gender. For instance, conflict has been reported to be higher for girls than for boys and higher with mothers as compared to fathers (Laursen, 1995), while at the same time females perceive their parents as more supportive than males (De Goede et al., 2009). In addition to level differences, gender differences in the postulated developmental links may arise. For instance, although the quality of the parent-adolescent relationship has been associated in similar ways to male and female externalizing behavior (e.g., Moffitt, Caspi, Rutter, & Silva, 2001), there is some
evidence that parent-adolescent relationship quality is a stronger predictor of internalizing problems for female as opposed to male adolescents (Compton, Snyder, Schrepferman, & Shortt, 2003).

Further, regarding parental gender, adolescents’ reports that their mothers are more involved in their emotional lives than their fathers, point to potentially different roles of mothers and fathers in adolescents’ affective experience (Stocker, Richmond, & Rhoades, 2007). Thus, both parental and adolescent gender need to be considered in the study.

The Present Study

The present study investigated the co-development of the adolescent-parent relationship, and adolescent negative affect and psychopathology (symptoms of generalized anxiety disorder [GAD] and physical aggression) from age 13 to age 15. Mothers and fathers were included, and the parent-adolescent relationship was assessed using adolescent-, mother- and father-report, thus incorporating the dyadic nature of relationships (Patterson & Bank, 1989). Adolescent negative affect was assessed across six five-day periods, during which adolescents reported on their emotions every day.

We hypothesized that (1) increases in negative interactions with both parents and decreases in support would be associated with increases in adolescent physical aggression and GAD symptoms, and that (2) changes in relationship quality predict changes in adolescent psychopathology through changes in adolescent negative affect. Parental and adolescent gender differences in the prediction of associated changes in the parent-adolescent relationship, adolescent affect and psychopathology were tested.

METHOD

Participants

The sample used in the present study constitutes the RADAR (Research on Adolescent Development and Relationships) study sample (see van Lier, Frijns, Neumann, den Exter Blokland, Koot, & Meeus, 2010 for an extensive description of the sample). Target adolescents were approached in grade 6 at 230 schools in the west and central parts of the Netherlands. A two step inclusion phase (teacher screen, followed by parent interviews) was used to include 497 families (target adolescent, mother, father, and sibling) as well as a friend of the target adolescent in an intensive longitudinal study. In the first step of the inclusion process, adolescents at increased risk for developing externalizing symptoms were oversampled, because of a specific focus of the RADAR study on delinquency development. High risk was determined by teacher ratings of the externalizing scales of the Teacher’s Report Form (TRF; Achenbach, 1991; Verhulst, van der Ende, & Koot, 1997). Youths who received a T-score ≥ 60 were labeled as high risk. Teacher information was available on 5,150 children. Due to the full family design and intense data collection, which requires a firm grip of the Dutch language, only adolescents who were identified by teachers as
being of Dutch origin were selected ($N = 3,237$). Due to logistical reasons, 1,544 of these children were included in the second sample selection phase. In this phase, youths’ parents were approached by phone. 463 adolescents were excluded, due to incorrect phone details, or because they did not meet additional requirements for inclusion in the study (both parents present, and presence of a sibling aged $\geq 10$ years), and 583 actively refused to participate or failed to provide written consent, resulting in the inclusion of 497 families (291 target adolescents at average risk and 206 at high risk for externalizing problems). Family socioeconomic status was low in 11% of the sample, and the majority of participants (97%) had a Dutch-Caucasian background.

For the present study, adolescent, mother and father ratings were used. Participants were included if data from the adolescent and both parents was available for at least 2 of the 3 annual assessments ($N = 452$, 55.3% male). Adolescents’ mean age at Time 1 was $13.37$ ($SD = .61$). Male and female adolescents did not differ regarding risk group status, $\chi^2(1) = .20$, $p > .05$, or age, $t(485) = .65$, $p > .05$.

Procedure

Data on adolescent psychopathology and the parent-adolescent relationship were collected during three annual home visits (time[T]1, T5, and T9), involving the adolescent and his/her parents, and six internet assessments (T2, T3, T4 during the first year and T6, T7, T8 during the second year) in which the adolescent reported on his/her daily mood. All assessments were separated by three months. During the three home visits, adolescents and their parents completed the questionnaires used in the present study as part of larger questionnaire packages under the supervision of trained interviewers. Interviewers assured that family members did not share their answers with each other and explained questions when needed. During the internet assessments, adolescents filled out their experiences of different emotions during that day, via internet. These assessments were completed for five consecutive days (Monday to Friday). Each day, participants received an invitation via e-mail to participate (at approximately 5.30 p.m.) and logged in to the RADAR website for data collection. Reminder e-mails were sent to participants who had not completed their assessment 1.5 hours after the initial invitation was sent, followed by text messages and phone calls to those who had still not completed their assessment 1.5 hours after the reminder had been sent.

Measures

*Generalized Anxiety Disorder Symptoms* were assessed with the Generalized Anxiety scale of the Screen for Child Anxiety Related Emotional Disorders (SCARED; Birmaher, Ketharpal, Brent, Cully, Balach, Kaufman, et al., 1997). The scale comprises 8 items (e.g., “I worry about the future” and “I worry about being as good as other kids”), which are rated on a three-point scale, with 0 (almost never), 1 (sometimes), and 2 (often). The Dutch SCARED has demonstrated acceptable validity (Hale, Raaijmakers, Muris, & Meeus, 2005). Cronbach’s $\alpha$s in the present sample are .84, .85, and .87 for Generalized Anxiety for T1, T5, and T9 respectively.
Physical Aggression was assessed through the 6 items of the Physical Aggression scale of the Proactive/Reactive aggression questionnaire (Linder, Crick, & Collins, 2002). Three items each assess proactive (e.g., “I try to get my own way by physically intimidating others”) and reactive physical aggression (e.g., “When I have been provoked by something a person has said or done, I have retaliated by threatening to physically harm that person.”). Items are scored on a seven-point scale ranging from 1 (not at all true) to 7 (very true). Scores on these items were summed to calculate a Total Physical Aggression score. Cronbach’s alphas ranged from .85 to .88 for Total Physical Aggression.

Negative Affect. Adolescents completed the Daily Mood Scale (an internet version of the Electronic Mood Device; Hoeksma, Sep, Vester, Groot, Sijmons, & de Vries, 2003) at the six one-week internet assessments (T2, T3, T4, T6, T7, T8) on five consecutive days. Each day, adolescents rated the intensity of happiness, anger, and anxiety on a 9-point Likert scale, ranging from ‘not at all’ to ‘very much’ during the daily internet sessions. Each emotion was indicated by three items (glad, happy and cheerful for happiness; angry, cross, and short-tempered for anger; afraid, anxious, and worried for anxiety). These three items per emotion were summed into a total score per day. Cronbach’s as ranged from .86 to .94 for happiness, from .87 to .97 for anger, and from .72 to .94 for anxiety. For each emotion a mean intensity score was computed by summing emotion scores of all days within each week, divided by the number of valid assessments in that week. Then, because discrete emotions have been shown to relate to adolescent psychopathology in general rather than specific ways (Neumann, van Lier, Frijns et al., 2010), and correlations between the discrete emotions ranged from .50 to .83, we summed the scores for happiness (reversed), anger, and anxiety to create a general Negative Affect score for each week.

Adolescent and parent reports of negative interactions and support were assessed with the corresponding short versions of subscales of the Network of Relationship Inventory (NRI; Furman & Buhrmester, 1985). Respondents completed the 6-item Negative Interaction and the 8-item Support subscale of the NRI. Adolescents completed both subscales twice at each annual assessment (T1, T5, T9): once reporting on their relationship with their mother, once reporting on their relationship with their father. Adolescents’ mothers and fathers completed the same subscales regarding their relationship with the adolescent. Examples of items are “Do you and your mother/father/child get on each others’ nerves?” and “How much do you and your mother/father/child get upset with or mad at each other?” for Negative Interactions, and “How much does your mother/father/child really care about you?” and “Does your mother/father/child like or approve of the things you do?”. All items are scored on a 5-point Likert scale ranging from 1 (a little or not at all) to 5 (more is impossible). The NRI has shown adequate construct validity (Edens, Cavell, & Hughes, 1999). The original factor structure has been replicated for the Dutch version (De Goede et al., 2009). In the present sample, Cronbach’s alphas ranged from .86 to .93 for Negative Interactions and from .73 to .86 for Support. Parents’ and adolescents’ support and negative interaction scores were summed to create dyadic mother-adolescent and father-adolescent Support and Negative Interaction scores.
CHAPTER 5

Adolescent sex, parental sex and risk status were dummy coded (0 = male, 1 = female, and 0 = low/average risk, 1 = at-risk).

Attrition and Missing Data

Complete data for Physical Aggression and GAD symptoms was available for 99% of the sample included in the present study for T1, for 95% at T5, and for 93% at T9. Complete adolescent-reported data on Negative Interactions and Support with mothers and fathers was available for 100%, 95%, and 92% at T1, T5, and T9 respectively. Mother- and father-reports of their relationship with the adolescent were complete for 100%/99% (mothers/fathers) of the sample at T1, for 94%/93% at T5, and for 93%/91% at T9. Percentage of complete data for the six 5-day internet assessments ranged from a high of 93% at T2 to a low of 74% at T7. Youths missing data on the internet assessments did not differ from those with complete data with regard to gender, socioeconomic status, and T1 GAD and Physical Aggression scores.

Statistical Analyses

Growth curve models were run in Mplus5 (Muthén & Muthén, 2007). In these models, the growth in each of the study variables was reflected by the latent growth parameters intercept and slope. The intercept reflects mean level differences over the studied period, while the linear slope reflects mean time-related changes in each of these measures. Individual variation around the means of the latent parameters is captured by the estimation of the variances of the growth parameters. The intercept was parameterized at the initial assessment (age 13) in all models. All models were corrected for non-normal distributions by maximum likelihood estimation with robust standard errors (MLR). Missing data were accounted for by full information maximum likelihood estimation. To determine model fit, the comparative fit index and Tucker Lewis Index (CFI and TLI; acceptable fit values > .90; Bentler & Bonett, 1990) and the root mean squared error of approximation (RMSEA; acceptable value < .08; Browne & Cudeck, 1993) were used. All models were controlled for risk status, and all but the multigroup models were controlled for adolescent gender, because the latter appeared to affect the course of GAD symptoms.

RESULTS

Descriptive Statistics

Descriptive statistics of the main study variables for male and female adolescents are in Table 5.1. Male adolescents reported significantly higher Physical Aggression and lower GAD Symptoms at all assessments than females. The score for Negative Interaction between mothers and daughters was higher than the score for Negative Interactions between mothers and sons at T1 (Age 13) and the Score for Mother-Daughter Support was higher than the score for Mother-Son Support.
at T9 (Age 15). No gender differences were found for adolescents’ relationship with fathers, and for Negative Affect (though there were non-significant trends for females to score higher on Negative Affect than males at T6 and T9).

Linear growth curves were fitted to the data for all study variables individually to determine the development of Physical Aggression, GAD Symptoms, Negative Affect, and Negative Interactions and Support in the adolescent-parent relationship. Model fit indices and parameter estimates of mean levels and variances of the individual growth models are in Table 5.2. All growth models with an intercept and linear term had a good fit to the data and showed statistically significant variance in mean levels (intercepts) and development (slopes), showing individual variation in level and growth with age was present.

Q1: Developmental Associations Between the Parent-Adolescent Relationship and Psychopathology

We tested whether the growth parameters of Negative Interactions and Support in the adolescent’s relationship to his/her mother and father predicted growth parameters of adolescent Physical Aggression and GAD symptoms. Eight models were estimated: four for Physical Aggression (for Negative Interactions between adolescents and mothers, and adolescents and fathers, and for Support in the adolescent-mother, and Support in the adolescent-father relationship) and four for GAD symptoms. In these models, the intercepts of Physical Aggression and GAD Symptoms respectively, were regressed on the intercept of the adolescent-parent relationship variable, and the slopes of Physical Aggression and GAD Symptoms were regressed on the slopes of the parent-adolescent relationship variable. The slopes of Physical Aggression and GAD Symptoms were also regressed on the intercepts of the relationship variables.
### Table 5.1
Means and Standard Deviations for the Main Study Variables for Male and Female Adolescents

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>SD</th>
<th>M</th>
<th>SD</th>
<th>F</th>
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</thead>
<tbody>
<tr>
<td><strong>Physical Aggression</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>T1</td>
<td>10.36</td>
<td>5.46</td>
<td>7.67</td>
<td>3.71</td>
<td>38.21***</td>
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<tr>
<td>T5</td>
<td>10.09</td>
<td>5.55</td>
<td>7.39</td>
<td>3.19</td>
<td>37.84***</td>
</tr>
<tr>
<td>T9</td>
<td>9.91</td>
<td>5.37</td>
<td>7.41</td>
<td>3.26</td>
<td>32.20***</td>
</tr>
<tr>
<td><strong>GAD Symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1</td>
<td>10.56</td>
<td>2.59</td>
<td>11.50</td>
<td>3.35</td>
<td>11.02***</td>
</tr>
<tr>
<td>T5</td>
<td>9.88</td>
<td>2.36</td>
<td>11.38</td>
<td>3.52</td>
<td>27.74***</td>
</tr>
<tr>
<td>T9</td>
<td>9.80</td>
<td>2.23</td>
<td>12.07</td>
<td>4.11</td>
<td>51.05***</td>
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<tr>
<td>Negative Interaction T1</td>
<td>15.32</td>
<td>4.13</td>
<td>16.19</td>
<td>4.82</td>
<td>3.95*</td>
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<td>4.92</td>
<td>16.42</td>
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<td>.92</td>
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<td>1.30</td>
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<td>36.69</td>
<td>4.08</td>
<td>2.92</td>
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<tr>
<td>Support T9</td>
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<td>3.78</td>
<td>36.41</td>
<td>4.53</td>
<td>7.71**</td>
</tr>
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<td>34.16</td>
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</tr>
<tr>
<td>Support T5</td>
<td>34.07</td>
<td>4.22</td>
<td>33.34</td>
<td>4.63</td>
<td>2.72</td>
</tr>
<tr>
<td>Support T9</td>
<td>33.70</td>
<td>3.99</td>
<td>33.27</td>
<td>4.77</td>
<td>.80</td>
</tr>
<tr>
<td><strong>Negative Affect</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T2</td>
<td>16.64</td>
<td>8.94</td>
<td>17.63</td>
<td>9.76</td>
<td>1.03</td>
</tr>
<tr>
<td>T3</td>
<td>16.98</td>
<td>9.61</td>
<td>17.78</td>
<td>11.13</td>
<td>.54</td>
</tr>
<tr>
<td>T4</td>
<td>18.31</td>
<td>10.60</td>
<td>18.47</td>
<td>11.36</td>
<td>.00</td>
</tr>
<tr>
<td>T6</td>
<td>17.97</td>
<td>9.88</td>
<td>20.08</td>
<td>12.20</td>
<td>3.20+</td>
</tr>
<tr>
<td>T7</td>
<td>18.19</td>
<td>10.80</td>
<td>18.92</td>
<td>10.06</td>
<td>.54</td>
</tr>
<tr>
<td>T8</td>
<td>19.26</td>
<td>10.70</td>
<td>21.51</td>
<td>11.60</td>
<td>3.74+</td>
</tr>
</tbody>
</table>

Note. GAD = Generalized Anxiety Disorder. F-tests as obtained from univariate analyses of variance, controlling for risk group. *** p < .001. ** p < .01. * p < .05. + p < .10.
Table 5.2

Results of Individual Latent Growth Curve Analyses of Study Variables

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Variance</th>
<th>Covariance</th>
<th>Model fit</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I SE S SE</td>
<td>I SE S SE</td>
<td>S * I SE</td>
<td>χ²(df) CFI TLI RMSEA</td>
</tr>
<tr>
<td>Phys Agg</td>
<td>9.14*** .21 -.14 .13</td>
<td>18.*** 3.45 3.19* .53</td>
<td>-4.02* 1.82</td>
<td>.00(1) 1.00 1.00 .00</td>
</tr>
<tr>
<td>GAD</td>
<td>10.85*** .14 -.08 .08</td>
<td>5.46*** .91 1.03* .48</td>
<td>-.23 .49</td>
<td>7.99**(1) .98 .93 .13</td>
</tr>
<tr>
<td>NegIntM</td>
<td>3.15*** .04 .05* .02</td>
<td>.70*** .09 .14*** .03</td>
<td>-.11** .04</td>
<td>1.95(1) 1.00 .99 .05</td>
</tr>
<tr>
<td>NegIntF</td>
<td>3.02*** .04 .10*** .02</td>
<td>.78*** .14 .10** .04</td>
<td>-.10 .06</td>
<td>.75(1) 1.00 1.00 .00</td>
</tr>
<tr>
<td>SupportM</td>
<td>7.40*** .04 -.12*** .02</td>
<td>.48*** .05 .07*** .02</td>
<td>-.05 .02</td>
<td>1.34(1) 1.00 1.00 .03</td>
</tr>
<tr>
<td>SupportF</td>
<td>6.88*** .04 -.10*** .02</td>
<td>.58*** .07 .09** .03</td>
<td>-.07* .04</td>
<td>1.80(1) 1.00 .99 .04</td>
</tr>
<tr>
<td>NegAffect</td>
<td>16.71*** .48 1.77*** .33</td>
<td>79.42*** 10.47 28.65*** 4.04</td>
<td>-15.09** 4.74</td>
<td>29.61*(16) .98 .98 .04</td>
</tr>
</tbody>
</table>

CHAPTER 5

The results of these eight models are in Table 5.3. Given our focus on developmental associations, only results of slope associations will be discussed. Intercept association are reported in the tables. The slope of Negative Interactions with mothers and fathers was positively related with the slope of Physical Aggression but not of GAD Symptoms. Slopes of Support were not significantly associated with the slopes of Physical Aggression or with those of GAD Symptoms.

In order to determine whether associations between the growth parameters differed for males and females, multigroup models were specified by gender. Models in which all parameters were constrained to be equal for males and females were compared to models in which individual regressions of growth parameters were allowed to vary for male and female adolescents. No sex differences in slope associations were found: Associations between slopes of adolescent-mother Negative Interactions, $\Delta\chi^2(1) = -.05$, and adolescent-father Negative Interactions, $\Delta\chi^2(1) = .35$, and Physical Aggression; slope of adolescent-mother Negative Interactions, $\Delta\chi^2(1) = -.28$, and adolescent-father Negative Interactions, $\Delta\chi^2(1) = -.49$, and GAD.

Q 2: Role of Negative Affect in the Link Between the Parent-Adolescent Relationship and Adolescent Psychopathology

Because the only significant associations between slope parameters of relationship quality and adolescent psychopathology pertained to Negative Interactions and adolescent Physical Aggression, whereas no significant associations were found for the slopes of Support and GAD Symptoms, the following analyses focus exclusively on associations between adolescent-mother and adolescent-father Negative Interactions, and adolescent Physical Aggression.

Before testing for the role of variation in Negative Affect in the developmental associations between Negative Interactions with parents and adolescent Physical Aggression, associations between Negative Interactions and Negative Affect were established using bivariate parallel growth models, which were run separately for Negative Interactions with mothers and fathers. Negative Interactions were regressed on growths parameters of Negative Affect. Increases in Negative Interactions with mothers (but not with fathers), significantly predicted increases in adolescent Negative Affect (see Table 5.4). Comparison of multigroup models by gender showed that associations between slopes of adolescent-mother Negative Interactions, $\Delta\chi^2(1) = .07$, and adolescent-father Negative Interactions, $\Delta\chi^2(1) = .67$, did not differ significantly for male and female adolescents, both $ps > .05$. 
## Table 5.3

**Main Results of Parallel Growth Curve Analyses of the Association between Parent-Adolescent Relationship and Adolescent Psychopathology**

### Physical Aggression

<table>
<thead>
<tr>
<th>Model</th>
<th>Predictor</th>
<th>(b)</th>
<th>(SE)</th>
<th>(b)</th>
<th>(SE)</th>
<th>(\chi^2(\text{df}))</th>
<th>CFI</th>
<th>TLI</th>
<th>RMSEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>NegM</td>
<td>1.26***</td>
<td>.24</td>
<td>.77*</td>
<td>.39</td>
<td>8.46(13)</td>
<td>1.00</td>
<td>1.00</td>
<td>.00</td>
</tr>
<tr>
<td>2</td>
<td>NegF</td>
<td>1.23***</td>
<td>.28</td>
<td>2.27*</td>
<td>.85</td>
<td>9.79(13)</td>
<td>1.00</td>
<td>1.00</td>
<td>.00</td>
</tr>
<tr>
<td>3</td>
<td>SupM</td>
<td>-1.39***</td>
<td>.32</td>
<td>-.65</td>
<td>.69</td>
<td>11.73(13)</td>
<td>1.00</td>
<td>1.00</td>
<td>.00</td>
</tr>
<tr>
<td>4</td>
<td>SupF</td>
<td>-1.25***</td>
<td>.29</td>
<td>-1.42</td>
<td>.80</td>
<td>9.89(13)</td>
<td>1.00</td>
<td>1.00</td>
<td>.00</td>
</tr>
</tbody>
</table>

### Generalized Anxiety Disorder Symptoms

<table>
<thead>
<tr>
<th>Model</th>
<th>Predictor</th>
<th>(b)</th>
<th>(SE)</th>
<th>(b)</th>
<th>(SE)</th>
<th>(\chi^2(\text{df}))</th>
<th>CFI</th>
<th>TLI</th>
<th>RMSEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>NegM</td>
<td>1.33***</td>
<td>.32</td>
<td>.37</td>
<td>.39</td>
<td>28.95(13)</td>
<td>.98</td>
<td>.96</td>
<td>.05</td>
</tr>
<tr>
<td>2</td>
<td>NegF</td>
<td>1.06***</td>
<td>.27</td>
<td>1.11</td>
<td>.71</td>
<td>31.02(13)</td>
<td>.98</td>
<td>.96</td>
<td>.06</td>
</tr>
<tr>
<td>3</td>
<td>SupM</td>
<td>-.94**</td>
<td>.33</td>
<td>.27</td>
<td>.59</td>
<td>25.69(13)</td>
<td>.99</td>
<td>.97</td>
<td>.05</td>
</tr>
<tr>
<td>4</td>
<td>SupF</td>
<td>-1.39***</td>
<td>.32</td>
<td>-.16</td>
<td>.59</td>
<td>32.26(13)</td>
<td>.98</td>
<td>.96</td>
<td>.06</td>
</tr>
</tbody>
</table>

*Note. NegM = Mother-Adolescent Negative Interaction. NegF = Father-Adolescent Negative Interaction. SupM = Mother-Adolescent Support. SupF = Father-Adolescent Support. The intercepts of Support and Negative Interaction with parents never significantly predicted the slope of Physical Aggression or GAD symptoms.*** \(p < .001\). ** \(p < .01\). * \(p < .05\).*

Next, we tested for a possible role of Negative Affect in the link between Negative Interactions and Physical Aggression. A graphical representation of these models is shown in Figure 5.1. Latent growth parameters of Physical Aggression were regressed on the growth parameters of Negative Interactions, and of Negative Affect, and growth parameters of Negative Affect were regressed on growth parameters of Negative Interactions. Significance of indirect effects of Negative Affect in associations between Negative Interactions and Physical Aggression were tested for using the IND command in Mplus (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). Adolescent Negative Affect played a significant indirect role in associations between adolescent-mother Negative Affect and adolescent Physical Aggression, and in this indirect effects model, the formerly significant association between adolescent-mother Negative Interactions and adolescent Physical Aggression failed to reach statistical significance. Changes in adolescent Negative Affect did not play a significant indirect role in associations between the slopes of adolescent-father Negative Interactions and Physical Aggression (Table 5.4).
## Table 5.4

*Estimates from Direct (and Mediation) Models for Physical Aggression*

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Direct effects</th>
<th>Indirect effect</th>
<th>Indirect effect</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NegInt → Phys Agg</td>
<td>NegInt → NegAff</td>
<td>NegAff → PhysAgg</td>
</tr>
<tr>
<td></td>
<td>B   SE  β</td>
<td>B   SE  β</td>
<td>B   SE  β</td>
</tr>
<tr>
<td>NegInt Mother</td>
<td>1.26 .24 .26***</td>
<td>3.83 .65 .36***</td>
<td>(.13) (.04) (.29***)</td>
</tr>
<tr>
<td>(.87) (.30) (.18**)</td>
<td>(3.75) (.64) (.36***)</td>
<td></td>
<td>(.55) (.17) (.10**)</td>
</tr>
<tr>
<td>NegInt Father</td>
<td>1.23 .28 .26***</td>
<td>3.33 .81 .32***</td>
<td>(.14) (.04) (.30***)</td>
</tr>
<tr>
<td>(.72) (.34) (.15*)</td>
<td>(3.34) (.80) (.32***)</td>
<td></td>
<td>(.47) (.16) (.10**)</td>
</tr>
<tr>
<td>NegInt Mother</td>
<td>.77 .39 .18*</td>
<td>4.20 1.31 .29**</td>
<td>(.06) (.03) (.19*)</td>
</tr>
<tr>
<td>(.47) (.41) (.11)</td>
<td>(4.12) (1.23) (.30**)</td>
<td></td>
<td>(.25) (.12) (.06*)</td>
</tr>
<tr>
<td>NegInt Father</td>
<td>2.27 .85 .41*</td>
<td>7.35 4.26 .30</td>
<td>(.03) (.09) (.09)</td>
</tr>
<tr>
<td>(2.54) (1.49) (.40*)</td>
<td>(7.12) (3.73) .40</td>
<td></td>
<td>(.19) (.21) (.03)</td>
</tr>
</tbody>
</table>

**Note.** NegInt = Negative Interactions. PhysAgg = Physical Aggression. NegAff = Negative Affect. *** $p < .001$. ** $p < .01$. * $p < .05$. **
CHAPTER 5

Finally, we tested for possible gender differences in indirect pathways using multigroup models. No significant gender differences were found; for the path from adolescent-mother Negative Interactions via Negative Affect to Physical Aggression: $\Delta \chi^2(2) = .03$, for the path from adolescent-father Negative Interactions via Negative Affect to Physical Aggression: $\Delta \chi^2(2) = 1.91$, both $p$s > .05.

DISCUSSION

The present study examined the co-development of the parent-adolescent relationship, adolescent negative affect and internalizing and externalizing problems from age 13 to age 15, using parallel growth curve models. Significant developmental associations were found between negative interactions with mothers and fathers and adolescent physical aggression, but not between negative interactions and GAD symptoms. Changes in support in the relationship with parents were never associated with changes in adolescent psychopathology. Developmental associations between adolescent-mother negative interactions and adolescent physical aggression were fully mediated by adolescent negative affect, whereas slopes of adolescent-father negative interactions and adolescent physical aggression were direct and not mediated by adolescent negative affect. Some of the studied associations thus differed depending on parent-gender; however, changes in relationship quality, negative affect and psychopathology were associated in much the same way for male and female adolescents.

As expected, changes in negative interactions in parent-adolescent relationships were significantly associated with intraindividual change in physical aggression from age 13 to age 15, such that increasing levels of aggression were accompanied by increasing levels of negative interactions. Associated change between negative interaction with parents and adolescent aggressive behavior points to synchronous, interdependent, and possibly circular associations between family relationships and adolescent adjustment (Kuczynski, 2003; Patterson & Bank, 1989). While physical aggression is probably rarely displayed against, or directly in front of parents, parents’ knowledge of physical aggression displayed against siblings or peers is very likely to be a subject of conflict between parents and adolescents (e.g., Laursen, 1993). Further, it is likely that adolescents who display physical aggression against peers and/or siblings may not have the most peaceful conflict resolution strategies at their disposal (Rubenstein & Feldman, 1993), even in situations when they are not resorting to physical aggression, including in conflicts with parents. In turn, frequent negative interactions may impede the learning of more socially adequate ways to dealing with conflict, making future physical aggression more likely (Snyder et al., 1997). In addition, or instead of circular interaction patterns, associated change may also stem from common underlying factors, such as a genetic liability, that underlie the development of family relationships as well as aggressive behavior.

Unexpectedly, no direct longitudinal associations were found between negative interactions with parents and GAD symptoms, and between support in the adolescent-mother and the adolescent-father relationship and adolescent physical aggression and GAD symptoms. Our
findings stand in contrast to what has been reported previously, regarding both support (Hafen & Laursen, 2009) and conflict (Rueter et al., 1995) in the relationship. Diverging findings may be due to different conceptualizations of adolescent psychopathology, and different raters. While we studied physical aggression, Hafen & Laursen (2009) focused on general externalizing problems. As mentioned before, it is unlikely that physical aggression is displayed directly in front of or against parents, while general externalizing problems, which include behaviors such as talking too much, being stubborn or irritable, and swearing (Achenbach, 1991), are likely to be more visible to parents. A more direct relationship between parent-child support and general externalizing problems may thus be expected. Further, while we focused on GAD symptoms, the other studies used items covering the entire spectrum of internalizing symptoms, including items pertaining to hostility (Rueter et al., 1999) which may show some content overlap with parent-child disagreements, and items such as “I feel that no one loves me” (Achenbach, 1991), which show content overlap with low support. Finally, the study by Hafen and Laursen (2009) depended heavily on adolescent self-report, and associations between changes in support and changes in externalizing behavior may have been inflated by shared source variance. In contrast, a major strength of the present study is that the quality of the parent-adolescent relationship was rated by both the adolescent and the parent, representing the dyadic nature of this relationship (Patterson & Bank, 1989), and reducing the problem of shared source variance. It is also possible that the parent-adolescent relationship is related to the development of psychopathology mostly in those cases, in which the quality of the relationship is extremely poor. For instance, decreasing support in the relationship to parents is a normative phenomenon in adolescence, and a certain threshold of low support may have to be reached before it relates to the development of adolescent psychopathology.

Changes in mother-adolescent negative interactions were associated with changes in adolescent physical aggression through changes in adolescents’ negative affect. Thus, continuing negative interactions with mothers seem to contribute to the development of mental health problems through adding to pathogenic processes (high levels of negative emotions that may be difficult to regulate) within the adolescent. Increasing negative interactions between adolescents and their parents are normative in early adolescence and, for identity and autonomy development important developmental changes in adolescence (e.g., Goossens, 2006). However, if levels of negative interactions with mothers are high, they may lead to concurrent increases in the experience of negative affect, and thereby add to the development of psychopathology.

In contrast, developmental associations between father-adolescent negative interactions and adolescent physical aggression were not mediated by adolescent negative affect. In fact, changes in negative interactions with fathers were not associated with changes in adolescent negative affect. Adolescent gender played an important role in levels of physical aggression (with males scoring much higher than their female peers) and of GAD symptoms (with females consistently scoring higher than males), but figured less prominently in associations between developmental changes. The fact that adolescent-mother, but not adolescent-father negative interactions were related to the developmental course of adolescent negative affect is in line with the notion of mothers as the ‘emotional brokers’ in families (Patterson, 1980), and with cross-sectional findings that adolescents
perceive their mothers as more involved in their emotional lives than their fathers (Stocker et al., 2007).

Two additional findings with regard to adolescent negative affect are noteworthy. First, of all, the present study provides strong support to cross-sectional findings (Larson & Lampman-Petraidis, 1989) that negative affect increases in adolescence, and secondly, the study shows that the course of adolescent negative affect is closely intertwined with the course of adolescent physical aggression. This finding lends strong longitudinal support to the notion that negative and dysregulated affect underlies the development of psychopathology (e.g., APA, 1994; Bradley, 2000) and is in line with earlier cross-sectional (Larson et al., 1990; Neumann, van Lier, Gratz, & Koot, 2010; Silk et al., 2003) and longitudinal studies (Neumann, van Lier, Frijns et al., 2010) of links between emotions and psychopathology.

Strengths of the present study include its longitudinal design, and the fact that adolescent affect was measured using six five-day long periods of daily assessments of experienced affect. These intensive assessments minimize recall bias and maximize ecological validity (Shiffman, Stone, & Hufford, 2008). Also, the assessment of the adolescent-mother and adolescent-father relationship quality was based on combined ratings of the adolescent and his/her parents’ scores, reflecting the dyadic nature of relationships. Nevertheless, results of the present study should be interpreted in the light of its limitations. First, the sample consisted of white, mostly middle-class Dutch adolescents from (mainly) two-parent families; thus caution should be exercised in generalizing the results to the general adolescent population. It is also unclear how the present results generalize to clinical populations. Further, although the sample size was reasonably large, and we used repeated measures, the statistical power of this study may have been too limited to detect potential sex differences in associations. Another limitation is that emotions and internalizing and externalizing problems were assessed by adolescent self-report only, thus associations between negative emotions and psychopathology might have been inflated by shared source variance.

Nonetheless, the present study clearly underscores the importance of studying potential mediating processes in links between family relations and the development of adolescent mental health problems. It is obvious that further study on the precise mechanisms underlying these associations is warranted. Since few developmental associations between the adolescent-parent relationship and adolescent psychopathology were found, future studies may also want to focus on factors that potentially moderate associations between relationship quality and adolescent developmental outcomes. Candidate variables for such moderator effects might be attachment security (securely attached adolescents may find it easier to regard decreases in relationship quality as temporary, and less threatening to the continuity of the relationship), and emotion regulation difficulties.
The Role of Contextual Risk, Impulsivity, and Parental Knowledge in the Development of Adolescent Antisocial Behavior

Anna Neumann
Edward D. Barker
Hans M. Koot
Barbara Maughan


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A multitude of individual and contextual risk factors have been associated with the development of antisocial behavior (ASB) in adolescence (e.g., Loeber, 1990). According to ecological (Bronfenbrenner, 1977) and transactional (Sameroff, 2000) models, these factors interact in determining human development. Although recent research on the development of ASB has increasingly paid attention to interrelations between individual and contextual characteristics (e.g., Meier, Slutske, Arndt, & Cadoret, 2008; Trentacosta, Hyde, Shaw, & Cheong, 2009; Vazsonyi, Cleveland, & Wiebe, 2006) studies of this kind are still relatively scarce. The present study will focus on the development of ASB in an age period in which ASB shows a sharp increase, i.e., from early to mid adolescence, and consider the roles of individual, and parenting, family, and neighborhood contextual factors.

Neighborhood Risk, Family Risk, Impulsivity, and the Development of ASB

Neighborhood, family, and individual factors have consistently been shown to pose risk for the development of ASB (e.g., Loeber & Stouthamer-Loeber 1986; Lipsey & Derzon, 1998; Leventhal & Brooks-Gunn, 2000). Neighborhood risk can be defined in terms of neighborhood structure, and neighborhood social processes. Neighborhood structure refers to compositional characteristics of communities, such as rates of unemployment (e.g., Vazsonyi et al., 2006). Neighborhood social processes may include measures of informal social control (“the capacity of a group to regulate its member according to desired principles – to realize collective […] goals”; Sampson, Raudenbush, & Earls, 1997), or perceived neighborhood danger (e.g., Ingoldsby & Shaw, 2002). Both neighborhood structural characteristics and social processes have been linked with ASB in youth (e.g., Jencks & Mayer, 1990; Leventhal & Brooks-Gunn, 2000; Sampson et al., 1997; Wilson, 1987). Early adolescence is an important developmental period for the study of neighborhood effects, as adolescents spend increasingly more time in their communities. At this age, youth have greater autonomy and increased opportunity for unsupervised activities (e.g., Leventhal & Brooks-Gunn, 2000).

In addition to high-risk neighborhoods, adverse family situations may pose a risk for the development of ASB. Notably, youths who live with a single mother or father (e.g., Breivik & Olweus, 2006; Loeber & Stouthamer-Loeber, 1986) are at increased risk for the development of ASB. As past studies have also documented, however, family and neighborhood risk are highly likely to co-occur. In general, family socioeconomic status (SES) is lower in single parent families, as compared to two-caregiver households (Bulanda, 2008). Accordingly, single parent families may be over-represented in disadvantaged neighborhoods, and the children of these families are likely exposed to additional risk factors. Moreover, single parents who reside in disadvantaged neighborhoods may experience more difficulties in parenting their adolescent child than those in more advantaged neighborhoods, e.g., because these parents are also exposed to more stressors (e.g., Wilson, 1987).

Ultimately however, the influence of neighborhood structural and social processes, and of family risk, may be conditional on the child’s individual vulnerability to engage in ASB. One of the
most important individual risk factors for the development of ASB is high levels of impulsivity (e.g., Loeber, 1990). Impulsivity has been defined as “a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others” (Moeller, Barrett, Dougherty, Schmitz, & Swann, 2001, p. 1784), and has been linked to persistent delinquency across the adolescent years (Carrasco, Barker, Tremblay, & Vitaro, 2006). Consistent with expectations from Bronfenbrenner’s biococological model (1977), as well as Sameroff’s transactional model (2000), the link between impulsivity and ASB has been shown to be stronger for children living in more disadvantaged neighborhoods (Lynam, Caspi, Moffitt, Wikström, Loeber, & Novak, 2000; Meier et al., 2008), though not all studies have found such interactive effects (see, e.g., Vazsonyi et al., 2006). To our knowledge, no studies have yet focused on interactions between family risk and child impulsivity. We hypothesize here, that neighborhood structural and social process risk factors, single parenthood, and impulsivity put early adolescents at risk for the development of ASB. In addition to main effects of these risks, we also expect to find that the effect of impulsivity on ASB is stronger in high risk as opposed to low risk neighborhoods, and in single parent as opposed to two-caregiver families.

Parental Monitoring as a Protective Factor

Even if exposed to all these risk factors, not all early adolescents actually develop ASB problems. Some contextual factors may provide protection rather than put the adolescent at risk. One of the strongest protective factors against early adolescent ASB is parental monitoring (“a set of correlated parenting behaviors involving attention to and tracking of the child’s whereabouts, activities, and adaptations”; Dishion & McMahon, 1998, p. 61; Burke, Pardini, & Loeber, 2008; Hoeve, Dubas, Eichelsheim, van der Laan, Smeerik, & Gerris, 2009; Lahey, van Hulle, D’Onofrio, Rodgers, & Waldman, 2008; Loeber & Dishion, 1983). Parental monitoring is thought to enable parents to effectively control their child’s behavior, resulting in less involvement in risk behaviors (e.g., Rai, Stanton, Wu, Li, Galbraith, Cottrell et al., 2003; Ramirez, Crano, Quist, Burgoon, Alvaro, & Grandpre, 2004). Parental monitoring has been shown to protect against the development of ASB for male as well as female adolescents (e.g., Hoeve et al., 2009; Lahey et al., 2008; Laird, Criss, Pettit, Dodge, & Bates, 2008), and across different ethnic groups (Forehand, Miller, Dutra, & Watts Chance, 1997). Although levels of parental monitoring typically decrease across adolescence (e.g., Burke et al., 2008), evidence suggests that it remains important for adolescent adjustment until at least mid-adolescence (e.g., Lahey et al., 2008; Laird et al., 2008).

Research has not only linked parental monitoring to ASB, but evidence is also accumulating that parental monitoring itself is associated with, and affected by other risk factors, including neighborhood, family, and child individual factors. Neighborhood structural risk may affect parental monitoring in different ways. Fauth, Leventhal, and Brooks-Gunn (2007) showed that parental monitoring decreased in families who moved out of poor neighborhoods (by way of a court-ordered neighborhood desegregation program). This decrease may reflect the fact that parents
adjust their level of monitoring according to the level of neighborhood risk. That said, neighborhood stress has also been shown to decrease parental monitoring through increasing parents’ psychological distress (Kotchick, Dorsey, & Heller, 2005). Low levels of informal social control in the neighborhood are likely to decrease parental monitoring, for instance because in neighborhoods with low levels of informal social control, residents are less likely to share their observations of youth behavior in the neighborhood with the parents. Further, single parents generally know less about their child’s whereabouts than coupled parents (Pettit, Laird, Dodge, Bates, & Criss, 2001; Pardini, Fite, & Burke 2008). Thus, single parents may find it harder to counter neighborhood risk than coupled parents. Finally, child impulsivity correlates negatively with parental monitoring (Flannery, Vazsonyi, Torquati, & Fridrich, 1994). Importantly, although not studied in a longitudinal framework to date, reciprocal effects between parental monitoring and child characteristics are likely. That is, impulsive children may be especially hard to monitor without the help of neighbors or a partner. Given these potential combined and cumulative effects of environmental risk on parental monitoring, it is important to address them simultaneously and across time.

Recently, support for the notion that monitoring might constitute one of the more proximal pathways through which other risks have their effects on the development of ASB is beginning to show. Some recent studies have shown that neighborhood risk affects male adolescents’ ASB (Chung & Steinberg, 2006; Tolan, Gorman-Smith, & Henry, 2003) and child externalizing problems (Kohen, Leventhal, Dahinten, & McIntosh, 2008; Mrug & Windle, 2009) through poor parenting. Further, it has been shown that single parent households increase the risk for adolescent ASB, through low levels of parental monitoring (Breivik, Olweus, & Endresen, 2009). These findings suggest that contextual risk on ASB is (partly) mediated by poor parental monitoring. Accordingly, we hypothesize, that the influence of neighborhood risk, single parenthood, and impulsivity, as well as their possible interactions increase ASB in part through decreasing parental monitoring.

We chose a specific operationalization of parental monitoring. It has been shown that parents may overestimate their knowledge of young people’s whereabouts, and that adolescent reports reflect actual parental knowledge more accurately (Laird, Pettit, Bates, & Dodge, 2003). Accordingly, as in other studies (Dishion & McMahon, 1998; Laird et al. 2008; Pettit et al., 2001; Trentacosta et al., 2009) parental monitoring was operationalized here as adolescents’ reports of parental knowledge of youth whereabouts, rather than as active parental surveillance.

Finally, in the present study we test for the possibility of gender differences in these various processes. Male and female youth differ not only in levels of impulsivity (Else-Quest, Hyde, Goldsmith, & van Hulle, 2006), parental knowledge (Kim, Hetherington & Reiss, 1999), and ASB (e.g., Moffitt, Caspi, Rutter, & Silva, 2001), but there also exists some evidence that neighborhood and family risk may differentially impact male and female development (e.g., Zahn-Waxler, Shirtcliff, & Marceau, 2008). For instance, neighborhood risk is thought to be more important for male adolescents, compared to females (e.g., Kroneman, Loeber & Hipwell, 2004), whereas family factors are sometimes found to be more strongly linked to female than to male ASB (see Zahn-
Therefore, we tested whether interrelations between the variables studied depend on the adolescent’s gender. We hypothesize that neighborhood risk will have a stronger effect on male ASB, whereas living with a single parent is relatively more important for female ASB.

METHODS

Sample

Participants were from the Edinburgh Study of Youth Transitions and Crime (ESYTC; Smith & McVie, 2003), a large-scale, representative cohort of children (4,597; 51% male) constituted at age 12 and studied annually to age 17. The initial recruiting sample ($N = 4469$) at wave 1 included 92% of the total population of youths, who were enrolled as first year pupils at Edinburgh secondary schools in autumn of 1998. Of these, 156 opted out, and 13 could not be surveyed due to logistic reasons ($n = 8$) or difficulties understanding the questionnaires ($n = 5$), resulting in a response rate of 96.2% at wave 1 (McVie, 2001). At the following waves, students, who transferred to participating schools, were also asked to participate; this resulted in the sample of 4,597. For a full description of response rates per wave, see McAra & McVie (2007). The majority of participants (94.2%) were Caucasian; 1.6% were of Pakistani origin, 1.1% Chinese, 0.7% African, 0.7% Indian, 0.3% Bangladeshi, and 1.4% from other ethnic groups.

Procedure

Parental consent was obtained for all children who participated in the study. Trained researchers administered the self-report questionnaires to study members in classrooms. Absent students, at each data collection wave, were captured via follow-up visits to the school and by home visitation. To reassure participants about reporting sensitive information and to encourage honest reporting, particularly about their own behavior, a complete guarantee of confidentiality was given to each child.

Measures

The frequency of ASB in the previous year was assessed using self-reports at ages 13 and 15 years with the following nine items (response scale 0 = never to 7 = most days): 1) shoplifting, 2) breaking into a house/building, 3) joyriding, 4) disturbance of peace, 5) vandalize property, 6) arson, 7) break into car to steal something, 8) carried a knife or weapon for protection or in case it was needed in a fight, and 9) used force, threats, or a weapon to get money or something else from somebody. We created a composite score, ranging from 0 to 63 at each age available, by summing scores on all items. Coefficient alphas were .74 and .76 for age 13 and 15 respectively. Because the measure of ASB was extremely skewed, due to a preponderance of scores at the scale-minimum
(i.e., over-dispersion of zeros), the ASB scores at ages 13 and 15 were log-transformed. On transformation, the skewness improved (see Table 6.1).

**Neighborhoods** in Edinburgh were defined using a geographic information system (GIS) and census level information (Smith & McVie, 2003). Postcode data were collected from the school records of all cohort members at sweep 1, thereby allowing individuals to be allocated to one of these neighborhoods. Postcode information was available for 3,972 (92%) of all eligible participants at sweep 1. A small proportion of these (291) were not resident within the city of Edinburgh, therefore, a total of 3,681 (85%) of cohort members at sweep 1 were allocated to one of the 91 Edinburgh neighborhoods at age 12. The average neighborhood cluster size was 112 (range 36 – 462).

**Neighborhood economic deprivation** was determined by combining the neighborhood allocation with data from the 2001 census, which provided a range of demographic, housing, health, education and cultural information about the population as a whole, and per neighborhood. The four variables used as indicators of neighborhood economic deprivation were 1) % of population consisting of lone parent with dependent children; 2) % of households with more than one person per room; 3) % of population in local authority (public) housing; 4) % of population who are unemployed. For each adolescent a neighborhood economic deprivation score was computed by summing the values of these four indicators applicable to his/her allocated neighborhood. For some eligible participants no postcode information was available in the school records, while for others postcode data were present, but they were not resident within the city of Edinburgh. For these participants no deprivation score was computed. The measure showed satisfactory fit in a CFA (CFI = 0.99; TLI = 0.97; RMSEA = 0.15) and the standardized loadings of the four indicators were all greater than 0.80. Because it is the truly disadvantaged who are most at risk of being affected (cf. Crane, 1991), we identified the top 10% of the neighborhood economic deprivation score to indicate those most at risk (cf. Ingoldsby, Shaw, Winslow, Schonberg, Gilliom & Criss, 2006; Wikström & Loeber, 2000; Wilson, 1987). This resulted in a dichotomized measure of extreme neighborhood economic deprivation scored 0 = low economic deprivation and 1 = high economic deprivation. Accordingly, 204 male and 191 female adolescents lived in deprived neighborhoods versus 1,658 male and 1,624 female adolescents lived in non-deprived neighborhoods.

**Informal social control** was measured via a modified version of an existing scale (Sampson, Raudenbush, & Earls, 1997). The self-report items comprised two questions (‘would adults try to stop’ and ‘would someone call the police’) for three scenarios: 1) “if someone was spray painting a wall in your neighborhood”, 2) “if someone was trying to steal a car in your neighborhood” and 3) “if teenagers were fighting in the street in your neighborhood” (6 items: Cronbach’s alpha = .58). Due to the low alpha, we estimated a confirmatory factor analysis to help discriminate the acceptability of the measure. The analysis showed adequate model fit (CFI = 0.96; TLI = 0.87; RMSEA = 0.08) and the standardized loadings varied from .30 to .62. As for the measure of neighborhood economic deprivation, we used scale scores to identify the 10% of neighborhoods lowest in informal social control.
Family type was derived from participant reports and was coded as 0 = two-caregiver family (64%), and 1 = single parent family (29%). Information on family type was missing for the remaining 7% of the sample.

Low family income was derived from school and Education Department records of eligibility for free school meals; 13% of the sample was eligible (Smith & McVie, 2003).

Impulsivity was measured via a modified version of the Eysenck Impulsivity Scale (Eysenck, Easting, & Pearson, 1984) through self-reports at age 12 by the following six items: 1) Planning takes the fun out of things, 2) I get into trouble because I do things without thinking, 3) I put down the first answer that comes into my head on a test, and often forget to check it later, 4) I get involved in things that I later wish I could get out of, 5) I sometimes break rules because I do things without thinking, 6) I get so excited, about doing new things that I forget to think about problems that might happen (Cronbach’s alpha = .86). This scale discriminates children with conduct problems from controls, and predicts future conduct problems (Luengo, Carrillo-de-la-Peña, Otero, & Romero, 1994).

Parental knowledge was assessed using adolescent report at ages 13 and 15 (both Cronbach’s αs = .72) with the following three items: “In the last year, how often did your parents know 1) where you were going, 2) who you were with, and 3) what time you would be home” on a four point scale (1 = most days, 2 = at least once a week, 3 = less than once a week, 4 = never). Scores were recoded so that higher scores indicate higher levels of parental knowledge.

Attrition and Missing Data

Complete data for ASB was available for 94% of the original sample at age 13 and 90% at age 15. Complete data for parental knowledge were available for 93% of the original sample at age 13, and for 89% at age 15. Self-reported risk data were missing for small proportions of the sample at age 12, ranging from 7% (family type) to 11% (low informal social control). Children missing census-based neighborhood characteristics (20% of the total sample; N = 917) were more likely to be entitled to free school meals (24% vs. 16%), χ^2 (DF = 1, N = 3,706) = 17.57, p < .001, and scored slightly higher on self-reported impulsivity at age 12, t(4185) = 2.11, p < .05. They did not differ from the rest of the sample on any of the other study variables.

Analyses

Path analysis models were run in Mplus5 (Muthén & Muthén, 2007). Analyses proceeded in three major steps. In Step 1, an autoregressive cross-lagged model of age 13 and age 15 parental knowledge, and adolescent ASB was defined. In this model, we tested for main effects of the age 12 predictors of neighborhood economic deprivation, low informal social control, single parent family, and adolescent impulsivity. To ascertain that the neighborhood economic deprivation variable captured a neighborhood level risk and was not confounded with family level economic deprivation, all pathways were controlled for low family income. Age 13 and age 15 variables were
allowed to correlate. To control for non-independence of the data, youth were clustered within neighborhoods defined by the GIS scores. Using this baseline model, we added two-way interactions between the age 12 variables as predictors in Step 2. Each of the five two-way interactions (Neighborhood economic deprivation X Impulsivity, Low informal social control X Impulsivity, Single parent family X Impulsivity, Neighborhood economic deprivation X Single parent family, Low informal social control X Single parent family) was always tested alone, i.e., without the other interaction terms in the same model, to avoid a model with interactions depending upon other interactions, yielding non-interpretable results. Finally, we tested for indirect influences of age 12 variables via age 13 parental knowledge on age 15 ASB using the IND statement in Mplus. Because male and female adolescents differed on levels of all continuous study variables (see Table 6.1), and because there are reasons to expect gender differences in links between risk factors and male and female ASB (e.g., Kroneman et al., 2004), separate models were run for males and females.

All models were corrected for non-normal distributions by maximum likelihood estimation with robust standard errors (MLR). Missing data were accounted for by full information maximum likelihood estimation. Model fit was determined through the Comparative Fit Index and Tucker-Lewis Index (CFI & TLI; exact fit = 1.00, close fit 0.95 - 0.99, acceptable fit 0.90 – 0.95; Bentler & Bonett, 1980) and root mean square error of approximation (RMSEA; exact fit = 0.00, close fit 0.06 – 0.01, acceptable fit 0.08 – 0.06; Browne & Cudeck, 1993).

RESULTS

Descriptive Statistics

Descriptive statistics for all continuous study variables are presented in Table 6.1 by gender. Male and female adolescents differed significantly on levels of all variables, with males reporting higher levels of ASB, and impulsivity, and lower levels of parental knowledge than females.
### Table 6.1
Descriptive Statistics of the Main Continuous Study Variables for Male and Female Adolescents

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>SD</th>
<th>Skew</th>
<th>Kurtosis</th>
<th>M</th>
<th>SD</th>
<th>Skew</th>
<th>Kurtosis</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Male adolescents</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASB Age 13</td>
<td>4.59</td>
<td>7.09</td>
<td>2.46</td>
<td>7.61</td>
<td>2.49</td>
<td>4.50</td>
<td>2.96</td>
<td>11.19</td>
<td>11.62</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Log transformed ASB Age 13</td>
<td>.48</td>
<td>.48</td>
<td>.49</td>
<td>-1.05</td>
<td>.32</td>
<td>.40</td>
<td>.92</td>
<td>-.35</td>
<td>11.36</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>ASB Age 15</td>
<td>6.09</td>
<td>8.79</td>
<td>2.13</td>
<td>5.45</td>
<td>3.72</td>
<td>6.24</td>
<td>2.66</td>
<td>8.37</td>
<td>10.17</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Log transformed ASB Age 15</td>
<td>.55</td>
<td>.52</td>
<td>.35</td>
<td>-1.25</td>
<td>.41</td>
<td>.45</td>
<td>.68</td>
<td>-.80</td>
<td>9.49</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Impulsivity Age 12</td>
<td>14.02</td>
<td>5.49</td>
<td>-.47</td>
<td>-.22</td>
<td>12.53</td>
<td>5.62</td>
<td>-.26</td>
<td>-.54</td>
<td>8.69</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Parental Knowledge Age 13</td>
<td>9.52</td>
<td>2.07</td>
<td>-.70</td>
<td>.02</td>
<td>9.94</td>
<td>1.94</td>
<td>-.88</td>
<td>.28</td>
<td>-6.82</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Parental Knowledge Age 15</td>
<td>9.01</td>
<td>1.99</td>
<td>-.41</td>
<td>-.06</td>
<td>9.38</td>
<td>1.91</td>
<td>-.47</td>
<td>-.29</td>
<td>-6.05</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><strong>Female adolescents</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* ASB = antisocial behavior. T-values as obtained from independent samples t-tests.
### Table 6.2
Zero-order Correlations Between the Study Variables for Male and Female Adolescents

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Neighborhood economic deprivation</td>
<td>--</td>
<td>-0.06</td>
<td>0.14</td>
<td>0.28</td>
<td>0.05</td>
<td>-0.01</td>
<td>0.05</td>
<td>0.06</td>
<td>0.06</td>
</tr>
<tr>
<td>2 Neighborhood low informal social control</td>
<td>0.13</td>
<td>--</td>
<td>0.06</td>
<td>0.04</td>
<td>0.09</td>
<td>-0.04</td>
<td>0.00</td>
<td>0.08</td>
<td>0.06</td>
</tr>
<tr>
<td>3 Single parent family</td>
<td>0.15</td>
<td>0.07</td>
<td>--</td>
<td>0.27</td>
<td>0.07</td>
<td>-0.10</td>
<td>-0.07</td>
<td>0.13</td>
<td>0.15</td>
</tr>
<tr>
<td>4 Free meals</td>
<td>0.29</td>
<td>0.07</td>
<td>0.30</td>
<td>--</td>
<td>0.08</td>
<td>0.02</td>
<td>0.05</td>
<td>0.08</td>
<td>0.10</td>
</tr>
<tr>
<td>5 Impulsivity</td>
<td>0.07</td>
<td>0.07</td>
<td>0.10</td>
<td>0.10</td>
<td>--</td>
<td>-0.26</td>
<td>-0.19</td>
<td>0.32</td>
<td>0.32</td>
</tr>
<tr>
<td>6 Parental knowledge age 13</td>
<td>-0.06</td>
<td>-0.13</td>
<td>-0.09</td>
<td>-0.01</td>
<td>-0.22</td>
<td>--</td>
<td>0.45</td>
<td>-0.44</td>
<td>-0.40</td>
</tr>
<tr>
<td>7 Parental knowledge age 15</td>
<td>-0.05</td>
<td>-0.07</td>
<td>-0.02</td>
<td>-0.00</td>
<td>-0.16</td>
<td>0.42</td>
<td>--</td>
<td>-0.26</td>
<td>-0.35</td>
</tr>
<tr>
<td>8 Antisocial behavior age 13</td>
<td>0.09</td>
<td>0.09</td>
<td>0.13</td>
<td>0.06</td>
<td>0.31</td>
<td>-0.42</td>
<td>-0.31</td>
<td>--</td>
<td>0.63</td>
</tr>
<tr>
<td>9 Antisocial behavior age 15</td>
<td>0.05</td>
<td>0.07</td>
<td>0.11</td>
<td>0.05</td>
<td>0.31</td>
<td>-0.31</td>
<td>-0.33</td>
<td>0.65</td>
<td>--</td>
</tr>
</tbody>
</table>

*Note.* All correlations are significant with \( p \) at least < .05, one-tailed, unless marked with subscript \( a \).

Correlations for male adolescents are below, correlations for female adolescents are above the diagonal.
Zero-order correlations between all study variables for male and female adolescents are in Table 6.2. Correlations between neighborhood economic deprivation and low informal social control on the one hand, and ASB on the other were generally small and positive for both males and females. As expected, single parent family and impulsivity correlated positively with ASB, and the risk factors also correlated significantly with each other: Neighborhood economic deprivation and low social control were positively linked with single parent family (for male and female adolescents) and with impulsivity (for male adolescents only). Single parent family and impulsivity also correlated positively. Associations between risk factors and parental knowledge generally also showed the expected pattern, with neighborhood risk, single parent family status, and impulsivity all correlating negatively with parental knowledge (though not all correlations between neighborhood risk and parental knowledge reached statistical significance).

**Step 1: Main Effects of Neighborhood Risk, Family Risk, and Impulsivity on the Simultaneous Development of Parental Knowledge and Adolescent ASB**

The main effects model for male adolescents (see Figure 6.1) fit the data well: $\chi^2(8) = 51.00$, $p < .001$; CFI = .99, TLI = .93, RMSEA=.05 (90% CI = .04 - .07). The model revealed significant effects for each of the age 12 predictors on levels of ASB one year later. Low informal social control, single parent family, and impulsivity were also negatively linked with parental knowledge at age 13. The autoregressive cross-lagged part of the model showed moderate stability of parental knowledge, and high stability of ASB between the ages of 13 and 15 years. In addition, parental knowledge at age 13 was negatively linked to age 15 ASB, and age 13 ASB was negatively related to age 15 parental knowledge.

The main effects model for female adolescents (see Figure 6.2) also showed good model fit: $\chi^2(8)=41.34$, $p < .001$; CFI = .98, TLI=.94; RMSEA=.05 (90% CI = .03 - .06). For females, the main effects of neighborhood economic deprivation and low informal social control on age 13 parental knowledge and ASB failed to reach significance. However, as for male participants, single parent family and impulsivity were positively related to ASB. Parental knowledge was predicted by single parent family and by impulsivity (negative associations). Similar to results for male adolescents, the autoregressive cross-lagged part showed moderate stability of parental knowledge, and high stability of ASB. Parental knowledge at age 13 predicted age 15 ASB, and age 13 ASB predicted age 15 parental knowledge (negative associations).

In order to test for possible gender differences in path estimates in the main effects model, multigroup models were specified by gender. A fully constrained model (i.e., a model in which all estimates were constrained to be equal for male and females adolescents) was compared to models in which individual path estimates were freed one at a time (i.e., allowed to vary across gender). Comparison of these models revealed significant differences between male and female adolescents in the link from low informal social control to parental knowledge at age 13 (which was only
significant for males) \( \Delta \chi^2 (1) = 7.16, p < .01 \), and in the link from parental knowledge at age 13 to ASB at age 15, \( \Delta \chi^2 (1) = 7.80, p < .01 \) (which is significant for both genders, but stronger for females).

In the next step of the analyses, two-way interactions were added one by one to the main effects model. For male participants, none of the interactions was significant. For girls, two significant interaction effects were found: (1) impulsivity interacted with neighborhood economic deprivation in the prediction of parental knowledge, and (2) impulsivity interacted with family type in the prediction of parental knowledge. Model fit for the model including the impulsivity \( \times \) neighborhood economic deprivation interaction was good: \( \chi^2(10) = 43.64, p < .001; \) CFI= .98, TLI= .94; RMSEA=.04 (90% CI = .03 - .06), and the same was true for the model including the impulsivity \( \times \) family type interaction: \( \chi^2(10) = 45.55, p < .001; \) CFI= .98, TLI= .94; RMSEA=.05 (90% CI = .03 - .06). Plots displaying standardized mean scores of parental knowledge for girls with low (1 SD below the mean) or high (1 SD above the mean) impulsivity in economically deprived versus economically non-deprived neighborhoods (Figure 6.3) and from two-caregiver versus single parent families (see Figure 6.4), illustrate the nature of these interactions. Figure 6.3 illustrates that the link between impulsivity and parental knowledge is weaker in disadvantaged as compared to more advantaged neighborhoods. Tests of simple slopes support this conclusion (\( \beta = -.17, p < .05 \) for high neighborhood economic deprivation; \( \beta = -.28, p < .001 \) for low neighborhood economic deprivation). Figure 6.4 illustrates that the link between impulsivity and parental knowledge is stronger in two-caregiver (simple slope: \( \beta = -.30, p < .001 \)) as compared to single parent families (simple slope: \( \beta = -.15, p < .05 \)).

Follow-up tests for gender differences in the path estimates of age 12 interaction terms and age 13 variables were conducted. The path estimates did not differ significantly for male and female adolescents, which may indicate that similar interaction effects may be at work for male adolescents, but that these failed to reach statistical significance in the present sample.
Figure 6.1. Standardized path estimates from the structural model on neighborhood and family risk, and impulsivity on parental knowledge, and adolescent antisocial behavior (ASB) for male adolescents. All paths are controlled for low family income. All interaction terms were tested alone, i.e., without the other interaction terms in the model. Only statistically significant (p at least < .05) paths are shown. EcoDep = Neighborhood Economic Deprivation. Imp = Impulsivity. LowSocCon = Low Informal Social Control. SingPar = Single Parent Family.
Figure 6.2. Standardized path estimates from the structural model on neighborhood and family risk, and impulsivity on parental knowledge, and adolescent antisocial behavior (ASB) for female adolescents. All paths are controlled for low family income. All interaction terms were tested alone, i.e., without the other interaction terms in the model. Only statistically significant ($p$ at least < .05) paths are shown. EcoDep = Neighborhood Economic Deprivation. Imp = Impulsivity. LowSocCon = Low Informal Social Control. SingPar = Single Parent Family.
Figure 6.3. Graphic representation of the interaction between neighborhood economic deprivation and impulsivity in the prediction of parental knowledge for female adolescents.
Figure 6.4. Graphic representation of the interaction between family type and impulsivity in the prediction of parental knowledge for female adolescents.
Table 6.3

**Significant Indirect Effects of Parental Knowledge in Links Between Risk and Male and Female Antisocial Behavior**

<table>
<thead>
<tr>
<th>Age 12</th>
<th>Age 13</th>
<th>Age 15</th>
<th>B</th>
<th>SE</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male adolescents</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neighborhood Low Informal Social Control</td>
<td>Knowledge</td>
<td>ASB</td>
<td>.008</td>
<td>.003</td>
<td>.006*</td>
</tr>
<tr>
<td>Impulsivity</td>
<td>Knowledge</td>
<td>ASB</td>
<td>.001</td>
<td>.001</td>
<td>.013*</td>
</tr>
<tr>
<td>Female adolescents</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single Parent Family</td>
<td>Knowledge</td>
<td>ASB</td>
<td>.012</td>
<td>.004</td>
<td>.012**</td>
</tr>
<tr>
<td>Impulsivity</td>
<td>Knowledge</td>
<td>ASB</td>
<td>.003</td>
<td>.001</td>
<td>.039***</td>
</tr>
<tr>
<td>Neighborhood Economic Deprivation X Impulsivity</td>
<td>Knowledge</td>
<td>ASB</td>
<td>-.002</td>
<td>.001</td>
<td>-.017*</td>
</tr>
<tr>
<td>Single Parent Family X Impulsivity</td>
<td>Knowledge</td>
<td>ASB</td>
<td>.002</td>
<td>.001</td>
<td>.027**</td>
</tr>
</tbody>
</table>

*Note. ASB = Antisocial Behavior. **p < .001. ***p < .01. *p < .05.*

**Step 3: Indirect Effects of Parental Knowledge on Associations Between Risk Factors and ASB**

Several significant indirect pathways were identified for both male and female adolescents (Table 6.2), suggesting that effects of other risks were partially mediated through effects on parental knowledge. For male adolescents, both age 12 low informal social control and impulsivity predicted higher age 15 ASB via changes in age 13 parental knowledge. For female adolescents, four significant indirect pathways were found: age 12 single parent family predicted higher age 15 ASB via changes in age 13 parental knowledge, and age 12 impulsivity predicted higher ASB at age 15 via changes in parental knowledge at age 13. In addition, the age 12 interactions between neighborhood economic deprivation and impulsivity, and between single-parent family and impulsivity predicted age 15 ASB via age 13 parental knowledge.

**DISCUSSION**

In this study, we investigated main and interactive effects of neighborhood and family risk, and adolescent impulsivity on early-mid adolescent ASB. In addition, we examined effects of these risk factors on parental knowledge, and the intervening role of parental knowledge in links between risk factors and ASB. Employing data from a large-scale representative sample of adolescents in Scotland, we showed (1) main effects of the risk factors under investigation on adolescent ASB and parental knowledge, (2) interactive effects of impulsivity with economic deprivation and family
CHAPTER 6

Several results of the present study support previous research on risk factors for the development of ASB in adolescence. As in earlier studies, neighborhood economic deprivation and low informal social control (e.g., Loeber & Wikström, 1993; Sampson et al., 1997), and living in a single parent household (e.g., Breivik & Olweus, 2006), were each associated with increased levels of ASB. Similarly, in agreement with earlier research, adolescent impulsivity and ASB were strongly associated (e.g., Beauchaine & Neuhaus, 2008; Carrasco et al., 2006). Further, the results corroborate earlier research on the role of parental knowledge in youth ASB (e.g., Lahey et al., 2008; Laird et al., 2008), showing that parental knowledge early in adolescence is linked with subsequent decreases in ASB, while earlier involvement in ASB is also associated with subsequent decreases in parental knowledge. A novelty of the current study is that we examined the complex interplay of neighborhood, family, and personal characteristics influencing parental knowledge and antisocial behavior in one model using longitudinal data. Importantly, in doing so the present study extends what has been reported previously, by (1) demonstrating main and interactive effects of neighborhood, family, and individual risk factors on the development of ASB in male and female adolescents separately, and (2) demonstrating that risk factors from all levels partly affect ASB through parental knowledge.

Neighborhood Risk, Family Risk, Impulsivity and the Development of ASB in Male and Female Adolescents

The first question of the present study concerned main and interactive effects of contextual risk factors including neighborhood economic deprivation, neighborhood low informal social control, and living in a single parent-family, together with child impulsivity on the development of ASB. When all four risk factors were evaluated in one model, we found main effects only for all risk factors for male participants. For females, there were no significant main or interaction effects of neighborhood risks on ASB. The finding that neighborhood risks affect male adolescents’ development, but not female adolescents’ development directly, is consistent with the hypothesis that neighborhood factors are more influential for boys’ than for girls’ development (Kroneman et al., 2004). In contrast, impulsivity was related to higher levels of male and female adolescent ASB alike, and so was living with a single parent. Thus, considering only main effects, it appears that individual and family risk contribute to the development of ASB in adolescents of both genders, but that male adolescents are additionally affected by the effects of neighborhood risk in the form of high economic deprivation and low informal social control.

The Intervening Role of Parental Knowledge

The second main goal of this study was to evaluate the hypothesis that both contextual risk and impulsivity partly affect adolescent ASB through their effects on parental knowledge. We
confirmed this hypothesis: neighborhood risk, family risk, and impulsivity were not only linked with the development of youth ASB directly, but also indirectly, via decreasing levels of parental knowledge. For male adolescents, age 12 impulsivity and low informal social control in the neighborhood increased age 15 ASB via decreasing parental knowledge at age 13. For female adolescents, main and interaction effects of impulsivity and living with a single parent increased ASB at age 15 through decreases in parental knowledge. Additionally, for females the interaction between neighborhood economic deprivation and impulsivity also affected age 15 ASB through changes in age 13 parental knowledge. Though the effects may seem small (standardized betas of indirect pathways range from .006 to .039), they are in line with what has been reported previously. For instance, Kohen and colleagues reported an indirect effect from neighborhood cohesion via family functioning and consistent parenting on verbal ability with a beta of .001, and Mrug and Windle (2009) reported an indirect effect of neighborhood poverty through neighborhood disorganization and parenting on children’s externalizing problems of .04. Thus, like interaction effects, indirect effects are generally small. Nevertheless, the indirect effects found in the present study add to our insight into potential influences on early adolescent ASB, despite its strong stability (betas of .63 and .57 for males and females, respectively, in the present study). As such the effects found offer pointers for potential ‘down-stream’ effects if a certain risk was targeted by an indicated prevention effort. Findings of the present study replicate and extend the findings of two previous studies of adolescent antisocial behavior. A cross-sectional study showed that weak neighborhood social organization is linked to ASB partly through parenting for male adolescents (Chung & Steinberg, 2006). Another, longitudinal one showed that parenting practices partially mediate the effects of neighborhood social processes on gang membership across the 11 – 14 year period in male youths from poor urban communities (Tolan et al., 2003). Importantly, the present study increased extant knowledge through demonstrating the importance of impulsivity as a moderator of contextual risk for females.

Of interest, the differential importance of neighborhood and family factors found for the development of male versus female adolescents’ ASB is also reflected in effects on parental knowledge. Low informal social control decreased parental knowledge for males but not females. This finding may reflect that male adolescents tend to spend more time in the community than their female peers (Kroneman et al., 2004), making parents of sons more dependent on neighbors’ support in keeping track of their child. In neighborhoods with higher levels of social control, parental knowledge may stem in part from neighbors, who care what youngsters are up to in the streets, and share their observations with these youngsters’ parents. Watchful neighbors may also contribute to youth disclosure (“I’ll tell them who I hang out with, because otherwise they might hear it from someone else”). In addition, standards of parenting may differ in neighborhoods with high versus low levels of informal social control.

In our study, for female adolescents, neighborhood economic deprivation affected parental knowledge in conjunction with female adolescents’ impulsivity, i.e. impulsivity had a stronger impact in decreasing parental knowledge in non-deprived neighborhoods. Parents in low risk neighborhoods may feel safer to let their daughter do as she pleases, whereas parents in high risk
neighborhoods may try to remain knowledgeable even if their daughter is highly impulsive. Living with a single parent was associated with a decrease in parental knowledge for male and female youths. For females, however, this effect was also dependent upon their impulsivity levels. The link between high impulsivity and decreased parental knowledge appears stronger in two-caregiver families, indicating that two-caregivers, more than single parents, benefit from low levels of their daughter’s impulsivity. This effect also shows that highly impulsive female adolescents are always harder to parent, even with the help of a partner, than less impulsive females. These interaction effects can be interpreted as showing that female adolescents’ personality affects parenting more in low (low neighborhood economic deprivation and two-caregiver families) as compared to high risk contexts (high neighborhood economic deprivation and single parent families).

Risk factors were not only differentially important for the developmental course of parental knowledge for male and female adolescents, but the protective effect of parental knowledge on adolescent ASB is also significantly stronger for female as compared to male adolescents. This finding is consistent with research showing that family support predicts less delinquency among girls but not boys (Windle, 1992), and thus with suggestions that females benefit more from positive parenting behaviors than males.

The Role of Impulsivity

We did not find a neighborhood risk by impulsivity interaction in the prediction of male ASB, which is in line with results reported by Vazsonyi and colleagues (2006), but may seem to contradict results of two other studies (Lynam et al., 2000; Meier et al., 2008). It needs to be considered, however, that results of the present study are not directly comparable with those of earlier studies testing interactions between impulsivity and neighborhood risk, for instance because we additionally included parental knowledge in our model. We did find, however, that impulsivity interacted significantly with neighborhood economic deprivation for female adolescents in the prediction of parental knowledge, which in turn lead to changes in ASB two years later. In addition, impulsivity interacted with contextual risk in the form of living with a single parent for female adolescents in the prediction of parental knowledge. Effects of the risk factors studied here (on both the development of ASB and on parental knowledge), are in line with the ecological systems model of influences on risk development, which distinguishes between different environmental layers of influence, that form nested structures (cf. Bronfenbrenner 1977). In accordance with this model, (1) the proximal factor adolescent impulsivity appeared as the main player in the prediction of ASB as well as parental knowledge development, and (2) it was shown that the impact of impulsivity is moderated in part by the more distal family and neighborhood risk factors (at least for females), and may be attenuated by adequate levels of parental monitoring for both male and female adolescents. The moderating role of child impulsivity on parenting factors in predicting antisocial outcomes for girls has also been demonstrated in earlier studies (e.g., Leve, Kim, & Pears, 2005), but these were not directly comparable using different risk factors and different age periods for study.
As such, findings provide support for the statement that “temperamental impulsivity is usually not enough – except in perhaps the most extreme cases – to result in psychopathology in the absence of additional vulnerabilities and/or risk factors” (Beauchaine & Neuhaus, 2008, p. 140). In this context, it should be pointed out that, while some effect sizes, particularly for effects of neighborhood risk and parental knowledge, seem rather small in comparison to those for child impulsivity, these appeared (1) with other variables known to affect ASB in the same model, (2) while controlling for low family SES, and (3) while clustering within neighborhoods to account for the non-independence of data. For future research, these findings imply that it is important to consider child factors in conjunction with contextual factors from both the family and the community level. For a better understanding of the development of youth ASB, it is also recommended to consider possible gender differences in the impact of (contextual) risk factors.

Study Limitations and Future Directions

A number of limitations need to be acknowledged. First, the measures of impulsivity, neighborhood informal social control, parental knowledge, and antisocial behavior were all obtained via self-reports, raising the possibility of shared method variance. Nevertheless, self-reports were chosen for good reason: Adolescents are likely to be well-aware of informal social control in their neighborhoods, and of their own impulsivity and their parents’ knowledge on their whereabouts. For relatively mild delinquency that will lead to only few convictions, self-reported measures are considered as most suitable and reliable (Jolliffe, Farrington, Hawkins, Catalano, Hill, & Kosterman, 2003), since adolescents are unsupervised by teachers and parents during the times in which they are most likely to be involved in aggressive and delinquent behaviors (U.S. Department of Education, 2000). Further, self-reports of ASB show significant association to official court records of aggressive acts (see Loeber & Farrington, 2000). Nevertheless, replications in samples where these constructs are assessed independently would be of considerable value. Second, the present results may be sample dependent, as more than 90% of the adolescents in the present study were white and from an urban community in Scotland, limiting the generalizability of our findings. Third, as Kerr and Stattin (2000; Kerr, Stattin, & Trost, 1999) pointed out, parental knowledge may be derived from youth disclosure, or active parental surveillance. As our measure of parental knowledge did not distinguish its source, our findings may not be directly comparable to those of studies that did. And although recent research supports the importance of parental knowledge, independently of whether derived from active surveillance or child disclosure (Lahey et al., 2008; Hoeve et al., 2009), it might be informative to investigate models such as the one tested here, with measures which distinguish the source of parental knowledge. Such distinctions could have important implications for the interpretation of the results, for instance, whether negative associations between adolescent impulsivity and parental knowledge are mainly due to the fact that impulsive children disclose less of their activities to their parents, or whether parents have a harder time monitoring highly impulsive children. Similarly, since recent research has supported the usefulness of a distinction between aggressive and non-aggressive forms of ASB, both in factor
analytic (e.g., Tackett, Krueger, Iacono, & McGue, 2005) and longitudinal research designs (e.g.,
Barker, Séguin, Raskin White, Bates, Lacourse, Carbonneau et al., 2007), it would be interesting to
see if the contextual risk and protective factors in the present study affect different forms of ASB in
different ways.

Taken together, results of the present study clearly demonstrate that while prevention and
intervention efforts at the individual level may be helpful, efforts may also usefully be aimed at the
family and community levels. As the present study shows, parenting early adolescent children may
be harder for some parents than for others, namely those living in neighborhoods with low levels of
informal social control, and single parents. Further, parent-support groups may be offered,
especially for single parents in the community. Even simply providing a meeting point for single
parents may enhance mutual social support, either emotionally or functionally by teaming up in
parenting activities (see Connell & Kubisch, 2001). At the community level, increasing social
control in the community by fostering a sense of community and shared responsibility, for instance
through neighborhood social activities, may decrease youth antisocial behavior not only directly but
also indirectly, by increasing neighborhood support for parents.
Adolescent Affect Dysregulation
- General Discussion
CHAPTER 7

Work in the present thesis is grounded in the notions that affect dysregulation is a core aspect of almost all forms of psychopathology, and that close social relationships provide a context for affective development and regulation. In the last two decades, there has been an increased interest in the role of affect, its regulation and dysregulation as related to different forms of psychopathology, with a range of studies showing associations between affect regulation deficits and diverse mental health problems (see Gratz & Roemer, 2004). At the same time, many studies have shown that the parent-child relationship may undergird as well as undermine the development of adaptive affect regulation (see Morris et al., 2007). Despite a generally increasing interest in the sources and consequences of affect dysregulation, there is a relative lack of studies on affect dysregulation focusing on adolescence, which is regrettable, given that adolescence is a time of important developmental changes regarding social relationships, affective experiences, and prevalence of several forms of psychopathology.

The studies reported in this thesis addressed this void of research on affective dysregulation in adolescence. Main goals were: (1) to evaluate measures for the assessment of affect dysregulation in adolescence, thereby also shedding more light on the concept of affect dysregulation in adolescence in general, (2) to establish how different forms of affect dysregulation relate to diverse forms of psychopathology in adolescents, (3) to study the parent-adolescent relationship as a context for the development of affect dysregulation and internalizing and externalizing psychopathology in adolescents, and (4) to establish how adolescent affect dysregulation and parent-child interactions jointly impact adolescent behavioral development depending on the broader social context of the family (single parent versus two-caregiver family, neighborhood characteristics).

Results of the in the present thesis clearly show that affect dysregulation can be assessed reliably in adolescence, on the level of emotion dynamics, as well as at the meta-cognitive level (chapters 2 and 3). Further, affect dysregulation appears to be associated with adolescent psychopathology cross-sectionally and longitudinally. Specifically, dimensions of affect dysregulation at the meta-cognitive level relate to anxiety, depression, aggressive and delinquent behavior in rather specific ways (chapter 2) and emotion dynamics underlie the continuity of anxiety disorder, depressive symptoms, and aggressive behavior from age 13 to age 14 (chapter 3). While it is clear that affective dynamics are related to the development of psychopathology, there is little evidence of specific associations of discrete emotions with psychopathology (with the exception of a more prominent role of anger compared to other emotions in relation to aggressive behavior; chapter 3). Moreover, parent-adolescent interactions evidently play an important role for adolescent affective development: parenting and relationship quality are associated with adolescent affect regulation difficulties at the meta-cognitive level (chapter 4), and parent-adolescent interactions are associated directly, and indirectly, via affective dysregulation with externalizing problems (chapter 5). Notably, parenting may also protect against some of the risks that high levels of adolescent behavioral dysregulation pose for the development of antisocial behavior in adolescents, and the same applies for the protection of potential risk at the family (single parenthood), and neighborhood (low informal social control, economic deprivation) level (chapter...
adolescent affect dysregulation: discussion

6). Adolescent gender played a role in several associations between social context and adolescent development (chapter 4 and 6), while only few and inconsistent gender differences were found in associations between affective dysregulation and adolescent psychopathology (chapters 3, 5, and 6).

In this chapter I will first discuss results relating to the conceptualization and assessment of affect dysregulation in adolescence. Next, I turn to links between different forms of affect dysregulation and psychopathology. Then, the focus will be on the role of the parent-adolescent relationship for adolescent affect dysregulation and psychopathology, and finally, effects of the broader social context will be discussed. Though not an explicit goal of the present thesis, adolescent gender emerged as a moderating variable for associations between the studied variables and these gender effects will be discussed where relevant. Finally, implications of the present studies for clinicians and policy makers are presented, limitations of the studies are discussed, and suggestions for future research are offered.

Affect Dysregulation and its Assessment in Adolescence

Despite the increasing interest in and research on affect dysregulation, it is still an ill-defined concept. This is likely due to the fact that affect, and consequently its regulation, are affected by a number of different ‘systems’, including psychophysiological, cognitive, and behavioral ones. One consequence of the equivocality of affect dysregulation as a concept is that little is known about its normative development, especially beyond the childhood years. Approaching affect dysregulation from different levels of experience (emotion dynamics and meta-cognitive), the present thesis adds to the understanding of what constitute important aspects of affective dysregulation in adolescence.

Inspired by cross-sectional reports that adolescents, compared to both children and adults experience heightened levels of negative emotions (Larson & Lampman-Petraidis, 1989) and heightened emotional variability (Larson, Csikszentmihalyi, & Graef, 1980), we studied levels and variability of happiness, anger, anxiety, and sadness longitudinally. In support of the notion of high emotional variability as an indication of affective dysregulation, it was shown that correlations between the day-to-day variability and intensity of negative emotions are large and positive, but large and negative between variability and intensity of happiness. Importantly, longitudinal analyses provided support for general developmental trends, as well as for intraindividual stability of emotion dynamics. Specifically, results of the present thesis are the first to show that slopes of negative affect increase from age 13 to age 15 (chapter 5), and to show moderate intraindividual stability of emotional variability over the course of three and six months (chapter 3), supporting the notion of emotional variability as an individual difference characteristic (Larsen & Diener, 1987).

Because we are interested in affect dysregulation as a risk factor for psychopathology, we also approached affect dysregulation from the meta-cognitive level, inspired by work that identified clinically important dimensions of affective dysregulation in adults (Gratz & Roemer, 2004). These dimensions include the awareness and clarity of perceived emotions, the ability to control attention and behavioral impulses when distressed, the non-acceptance of emotional responses, and low emotional self-efficacy. The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer,
CHAPTER 7

2004) was developed to assess these dimensions in adults. Results of our study of the DERS with samples of adolescents show that these dimensions are important and can be reliably distinguished in adolescents (chapter 2). Significant positive correlations between emotional variability and adolescent self-reported difficulties with emotional clarity and impulse control were found, as well as with the nonacceptance of emotional responses, low confidence in one’s ability to modulate negative emotional responses, and difficulties engaging in goal-directed behavior when distressed (Neumann, van Lier, Frijns, Meeus, & Koot, unpublished data) This finding supports the notion that emotion dynamics and perceived difficulties in emotion regulation represent different parts of one underlying concept in adolescence, namely affective dysregulation.

Gender emerged as an important variable in the study of affect dysregulation in adolescence. Though only few gender differences were found in emotion dynamics (females scored slightly higher on happiness variability and on sadness intensity and variability; chapter 3), female participants reported significantly higher levels of emotion regulation difficulties on four of the six DERS subscales, including Lack of Emotional Clarity, Difficulties Engaging in Goal-Directed Behavior when Distressed, Nonacceptance of Emotional Responses, and Limited Access to Emotion Regulation Strategies, while males reported more difficulties with emotional awareness. Observed gender differences on some of the DERS subscales should be interpreted with caution, as they may reflect gender based differences/bias in the ratings of emotion-related items in addition to true gender differences in emotion regulation difficulties (chapter 2). Nevertheless, taken together, results of the present thesis pertaining to gender differences in levels of affect dysregulation suggest that, while male and female adolescents may not experience very different levels and variability of emotions, their meta-cognitive analyses of experienced emotions differs to a good extent.

In sum, results of the present thesis add to our understanding of affect dysregulation in adolescence by showing intraindividual stability and normative developmental change in emotion dynamics, and by identifying clinically relevant dimensions of meta-cognitive affect dysregulation and gender differences in affect dysregulation in adolescents. In addition, the daily web-based ratings of experienced emotions used in our studies and the DERS are identified as reliable ways to assess different dimensions of affect dysregulation in adolescence, that relate in predictable ways to adolescent psychopathology and the family context, as will be further discussed below. Given their comparatively easy, time-effective and inexpensive procedures, these measures are well suited for the use with larger samples, and in longitudinal studies of emotional development in adolescence.

Affect Dysregulation and Psychopathology in Adolescence

Two main issues were identified as important for the study of affect dysregulation and psychopathology in adolescence. First, there is a lack of longitudinal studies on associations between affect dysregulation and psychopathology. Secondly, it is not clear how discrete emotions and specific dimensions of affect dysregulation relate to different forms of psychopathology. Below, contributions of the present thesis to these issues will be discussed in turn.
Longitudinal Associations Between Affect Dysregulation and Psychopathology

Cross-time associations between affect dysregulation and psychopathology were demonstrated in chapters 3 and 5 of the present thesis, with a focus on emotion dynamics. Chapter 3 shows that individual differences in the dynamics of happiness, anger, anxiety, and sadness partly account for changes in adolescent internalizing symptoms, while dynamics of negative emotions, but not of happiness, play a role in the development of aggressive behavior from age 13 to age 14. Parallel growth curves described in chapter 5 indicate parallel change between dynamics of adolescent negative affect and physical aggression from age 13 to age 15. In general, these results are consistent with theory, proposing that emotion dysregulation underlies the development of psychopathology (Bradley, 2000), and add to a growing body of evidence from studies showing longitudinal associations between affect dysregulation and internalizing problems in children and adolescents (Beauchaine, Gatzke-Kopp, & Mead, 2007; Bosquet & Egeland, 2006; Yap, Allen & Ladouceur, 2008).

The results of the studied longitudinal associations between affect dysregulation and adolescent psychopathology, suggest that by adolescence the development of affect dysregulation and psychopathology is so intertwined, that each constitutes both a source and a consequence of the other. That is, negative affect and heightened affective variability add to increases in symptoms of psychopathology, which in turn lead to higher levels of negative emotions and heightened affective variability. This suggests that several adolescent strategies used to deal with affect are not effective, that is they do not alleviate the distress, and may even exacerbate it in the long run. Maladaptive coping strategies may exacerbate negative affect in more or less direct ways. For instance, rumination is likely to increase negative affect directly, whereas giving in to aggressive impulses may alleviate distress in the short-term, but increase it in the long run, because it interferes with one’s social relationships.

General and Specific Associations Between Affect Dysregulation and Different Forms of Psychopathology

It has been clearly demonstrated that affect dysregulation is a risk factor for many forms of psychopathology (e.g., Cole & Hall, 2008). To move the field further, studies investigating the general and specific ways in which diverse forms of affect dysregulation relate to different forms of psychopathology have been called for (Cole & Deater-Deckard, 2009). Results of the present thesis show both general and specific associations between affect dysregulation and psychopathology. Levels of discrete experienced emotions (low happiness, anger, anxiety and sadness) related in mostly general ways to symptoms of internalizing problems (anxiety, depression), and aggressive behavior (chapter 3), suggesting that the notion of functional continuity (i.e., the notion that discrete emotions are specifically associated with the development of different forms of psychopathology; Malatesta & Wilson, 1988) does not hold for experiential data in adolescence. On the other hand, two specific aspects of affect dynamics, the intensity and variability of emotions, related in specific ways to symptoms of psychopathology: while depressive symptoms were uniquely associated with the level of emotions, but not their variability, the reverse was true for symptoms of anxiety.
disorders (chapter 3). In addition, though anxiety and sadness also played a role in aggressive behavior in some analyses, associations between anger and aggressive behavior were found more consistently. Dimensions of affect regulation difficulties at the metacognitive level also related in rather specific ways to anxiety, depression, aggressive and delinquent behavior (chapter 2).

The finding that (dynamics of) discrete emotions are related to psychopathology in rather nonspecific ways, is in line with some earlier cross-sectional studies on the same subject (e.g., Silk et al., 2003), but diverges from at least one other. A study on the facial expression of emotions and psychopathology in male adolescents (Keltner et al., 1995) reports specific links between the expression of discrete emotions and internalizing and externalizing problems. Taken together, the results of the present thesis and these of earlier studies suggest that, at the experiential level, there is little or no specificity of links between discrete emotions and different forms of psychopathology, while at the level of emotional expression, there is. This raises the possibility that different operationalizations of emotions and their dynamics may lead to different results regarding their relation with psychopathology. It is well known that measures of emotion from different levels generally show very little convergence (e.g., physiological, experiential, and behavioral measurements; e.g., Hubbard, Parker, Ramsden, Flanagan, Relyea, Dearing et al., 2004). This may be because they are not experienced, not labeled correctly, experienced but behaviorally suppressed etc. In addition, if expressed they may be falsely interpreted by raters. For example, some adolescents may feel emotional arousal but denote it as dysphoria without labeling it as sadness, fear, or remorse. Some individuals may experience various, but express only some emotions (e.g., express anger, but suppress the expression of fear). Alternatively, some may express all negative emotions in similar ways (e.g., express fear by attacking, making it look more like anger than fear). However, each of these conditions may be related to psychopathology. The question thus arises is: What determines which emotion is experienced, which is expressed, and how it is expressed? Moreover, many questions remain on how dimensions of emotions interact in their relation with the development of psychopathology.

Part of the answer to the questions posed above may lie in the meta-cognitive experience of affect and affect regulation. Dimensions of perceived emotion regulation difficulties were associated in divergent ways with aggressive and delinquent behavior and with internalizing symptoms. Specifically, in line with earlier findings it was found that difficulties in impulse control and engaging in goal-directed behavior when distressed associated with aggressive behavior (e.g., Beauchaine & Neuhaus, 2008), and a lack of emotional awareness with delinquency (e.g., Herpertz et al., 2001), while a lack of emotional clarity, nonacceptance of emotional responses and a limited access to emotion regulation strategies associated with symptoms of anxiety as well as depression, but not with externalizing problems (chapter 2).

Taken together, the results suggest that, whereas levels of discrete emotions relate in rather general ways to diverse forms of psychopathology, different forms of psychopathology may still be characterized by unique profiles of affect dysregulation (see Table 7.1 for an overview of dimensions of affect dysregulation as associated with different forms of psychopathology in the present thesis). For instance, while both depressive symptoms and anxiety disorder symptoms are
associated with increased negative affect and decreased positive affect, and by decreased emotional clarity, ineffective emotion regulation strategies, and the nonacceptance of emotional responses, only anxiety symptoms are additionally associated with high levels of emotional variability. Patterns of affect dysregulation underlying aggressive behavior may be better characterized by a combination of negative affectivity and impulse control difficulties, including difficulties in focusing attention to tasks at hand.

Future research may undertake efforts to combine diverse indices of affect dysregulation and study their unique and interactive effects on the development of psychopathology, thereby delineating the unique affect dysregulation profiles underlying different forms of psychopathology. These profiles might then be used to tailor effective prevention and intervention efforts.
Table 7.1
Overview of Dimensions of Affect Dysregulation Associated with Different Forms of Psychopathology in the Present Thesis

<table>
<thead>
<tr>
<th></th>
<th>Anxiety Disorder Symptoms</th>
<th>Depressive Symptoms</th>
<th>Aggressive Behavior (incl. Physical Agg &amp; ASB)</th>
<th>Delinquency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perceived Emotion</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regulation Difficulties</td>
<td>- Lack of Emotional Clarity</td>
<td></td>
<td>- Lack of Emotional Clarity</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Nonacceptance of Emotional Responses</td>
<td></td>
<td>- Nonacceptance of Emotional Responses</td>
<td></td>
</tr>
<tr>
<td>Emotion Dynamics</td>
<td>- High Variability of positive and negative emotions</td>
<td></td>
<td>- High levels of negative and low levels of positive emotions</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- High negative emotions, especially high anger</td>
<td></td>
</tr>
</tbody>
</table>

*Note. ASB = Antisocial Behavior.*
Parent-Adolescent Interactions as a Context for the Development of Affect Dysregulation in Adolescence

The parent-child relationship has long been recognized as an important context for emotional development during the childhood years. However, it is less clear, if and how interactions with parents continue to be associated with affect development in adolescence. Results of the studies described in the present thesis demonstrate the continuing importance of parents for adolescents’ affective lives by showing significant associations between the parent-adolescent relationship and adolescent affective dysregulation across different conceptualizations of parent-adolescent interactions (parenting, parent-adolescent relationship quality) and affect dysregulation (emotion dynamics, perceived difficulties in emotion regulation, impulsivity). Chapter 3 shows that maternal psychological control (and to a lesser extent maternal behavioral control) is significantly and consistently related to adolescent perceived emotion regulation difficulties. Results of chapter 4 demonstrate that both support and negative interactions in the adolescent-mother relationship are also associated with adolescents’ perception of their own affect regulation competencies. Results in chapter 5 extend these results to negative interactions in the adolescent-mother (cross-sectionally and longitudinally) as well as in the adolescent-father relationship (at least cross-sectionally), and to affective dysregulation assessed in terms of heightened negative affect. Two findings stand out: (1) the bidirectional nature of links between parent-adolescent interactions and adolescent affect dysregulation underscored by the longitudinal study in chapter 5, and (2) the fact that associations between parent-adolescent interactions and perceived affect regulation difficulties depend on adolescent gender.

In accordance with transactional models of development (e.g., Sameroff, 2010), and with the notion of affect (regulation) as both output and inputs of social interactions (e.g., Bell & Calkins, 2000), results of parallel growth curves, mapping the development of the adolescent-parent relationship and adolescent negative affect from age 13 to 15 years, clearly illustrate the bidirectional nature of associations between adolescent-parent relationship quality and adolescent affect dysregulation. Specifically, longitudinal associations between adolescent negative affect and relationships with mothers point to parallel change between affect and relationship quality (i.e., slope-slope associations), but no evidence for prediction of one by the other (i.e., intercept-slope associations) was found (chapter 5). Low quality parent-child interactions (i.e., low warmth/support, high levels of conflict) thus add to adolescent affect dysregulation, by inducing negative affect and by providing little support for handling negative affect. In turn, adolescent negative affectivity, difficulties in handling negative emotions and associated behavioral impulses, all add to less positive evaluations of the relationship by both the adolescent and the parent. Adolescent affect dysregulation likely increases both the frequency as well as the intensity of conflicts. In the worst case, parents and their adolescent children will find themselves in a continuous circle of negative affect and conflictual relationships that is increasingly hard to break. It will be vital for future research to determine which factors differentiate between parent-adolescent dyads that get stuck in this cycle, from those who do not.
Findings of the present thesis suggest that adolescent gender acts as a powerful moderator of associations between parent-child relationship quality and affect dysregulation, at least on the meta-cognitive level. Results reported in chapter 4 demonstrate that the mother-adolescent relationship quality and maternal behavioral control is more strongly associated with perceived emotion regulation difficulties for female than male adolescents. These findings are in line with the notion that the interpersonal nature of emotions is more salient for females than males (Shields, 1995). This might be due to gender-typical emotion socialization in childhood (e.g., parents teach girls more relationship-oriented emotion regulation strategies and their sons more active and instrumental emotion regulation strategies [Eisenberg et al., 1998]). However, associations between growth of the adolescent-mother relationship quality and adolescent negative affect (chapter 5), were gender-invariant. Combined, the results of the present thesis suggest that relationship influences are stronger for female adolescents for affect dysregulation at the meta-cognitive level, while relationship quality affects more “basic” emotional experiences (levels of negative affect) in similar ways for male and female adolescents. It might be that negative affectivity is more biologically determined than (meta-) cognitive aspects of affect dysregulation, which are more easily influenced by socialization experiences.

In sum, results regarding associations between parent-adolescent interactions and adolescent affect dysregulation suggest that (1) parenting behaviors, especially psychological control, as well as relationship characteristics, especially high levels of negative interaction, but also low levels of support, are associated with adolescent affect dysregulation in adolescence, (2) these associations are best described as bidirectional, and (3) links between parent-adolescent relationship quality and adolescent affect dysregulation at the meta-cognitive level are stronger for females, while relationship quality relates to direct emotional experiences in similar ways for male and female adolescents.

Results of the present thesis pertaining to the joint effects of adolescent affect dysregulation, parent-adolescent interactions, and adolescent psychopathology, illustrate that parent-adolescent interactions may either present a risk for adolescent affect dysregulation and subsequent psychopathology development (chapter 5), or protect adolescents from the risks associated with high levels of dysregulation (chapter 6).

Parallel growth between the mother-adolescent relationship quality and adolescent physical aggression was in part explained by growth in adolescent negative affect (chapter 5). Normative developmental increases in negative interactions and decreases in support in the mother -adolescent relationship are thus associated with increases in adolescent psychopathology, because they go hand in hand with adolescent negative affect. Thus, it seems that developmental changes in the mother -adolescent relationship form a risk factor for the development of psychopathology, because they interfere with adolescent affect regulation. It might be that these developments are of a temporary nature (e.g., van Doorn, Branje, & Meeus, 2010) for many adolescents: decreased quality of
relationship with parents will make adolescents less likely to seek their parents’ help to deal with emotional situations. At the same time, they may not yet have acquired the skills to deal with their affective experiences in adaptive ways on their own, nor have yet formed intimate relationships with friends and intimate partners that allow for the adaptive co-regulation of emotions. Future studies may investigate whether by late adolescence increases in relationship quality have a more supportive impact on adolescent affect regulation again. Alternatively, associations between the parent-adolescent relationship and adolescent affect dysregulation, be they positive or negative in nature, may be diminished by late adolescence, either because the adolescent has acquired better individual regulation strategies or has other partners to help him/her deal with affective experiences.

Of course, adolescent-parent interactions are not the only source of affect dysregulation. On the contrary, several aspects of affect dysregulation are likely to be genetically determined (Hariri & Forbes, 2007). One aspect of affect dysregulation that is at least partially genetically determined is heightened impulsivity (Forbes, Brown, Kimak, Ferrell, Manuck, & Hariri, 2009). In the context of the present thesis, heightened levels of impulsivity represent a dimension of affect dysregulation, which is closely related to behavioral self-regulation (Carver, 2004), and is thought to be genetically determined to a greater extent than for instance meta-cognitive emotion regulation difficulties. In accordance with Bronfenbrenner’s (e.g., 1986) bioecological model of development, which postulates a greater impact of more proximal as opposed to more distal risk factors, Chapter 6 identifies impulsivity as the main predictor of adolescent antisocial behavior, in a model which also includes family and neighborhood risk factors. Importantly, and also in line with predictions made by the bioecological and other current dynamic transactional models of development (Sameroff, 2010), the impact of impulsivity was moderated by family (single parent) and neighborhood risk (economic disadvantage), at least for female adolescents. Most important for the current discussion, the study also demonstrates the protective influence of parenting. In particular, it was demonstrated that parental monitoring acts as a buffer against some of the risk that high levels of impulsivity (and neighborhood and family risk factors) pose for the development of both male and female antisocial behavior. Thereby, this study points to the importance for parents to stay knowledgeable about and engaged with their child through mid adolescence, especially if the child’s behavioral control is low and is jeopardized additionally by environmental risk.

In sum, the parent-child relationship seems to affect both affect regulation and behavioral regulation of adolescents and thus to influence the development of psychopathology in the child. Reducing negative interactions, and maintaining support and monitoring when needed seem important ways to influence the development of both internalizing and externalizing adolescent problem behavior.

Clinical and Policy Implications

Never mind the rather basic nature of most of the work reported in the present thesis, I do believe that the present results have several important implications for clinical and policy work. Concerning clinical work, a number of points are important. First, studies in the present thesis
clearly show that emotion regulation related meta-cognitions can be assessed reliably in adolescents, and given that they are differentially associated with different forms of psychopathology, may be used as an additional diagnostic tool. Further, they may offer useful starting points for treatment (e.g., working on increasing emotional clarity and acceptance). Secondly, work in the present thesis underscores the usefulness of day-to-day registration of emotion intensity as a tool for diagnosis and treatment. That is, daily emotional experiences may be added to patient-diaries, for instance as a means to uncover sources of emotional instability. Regarding daily emotional experiences, it is necessary to include a diversity of discrete emotions, no matter the seemingly predominant psychopathological problem. Studies in this thesis show that, regarding clinical disorders, there may not be one predominant emotion that reigns the development of each specific type of psychopathology. A possible exception might be the role of anger in the development of aggressive behavior. Nevertheless, heightened anxiety and sadness were also implicated in aggression. Anxiety disorders are not only associated with increased feelings of anxiety and fear, but also with decreased positive affect and increased sadness. Despite that, there may still be specific patterns of affect dysregulation associated with the development of the anxiety disorders, as for instance it was found that heightened emotional variability, and diminished emotional clarity, low emotional self-efficacy, and nonacceptance of own emotional responses were all related to anxiety disorders. Consequently, clinicians should be aware that dysregulation in several discrete emotions may play a role in any given behavioral or emotional disorder, or that the presence of any given emotion, may not come to expression or be expressed as a different emotion. If one misses out on potential underlying feelings of fear, worry, and sadness, and focuses on anger, important opportunities for diagnosis and intervention may be missed. A final result of the present thesis, which has ramification for clinical work, is that symptoms of psychopathology fuel affect dysregulation, just as affect dysregulation fuels psychopathology development. Consequently, working on adaptive affect regulation is highlighted as an effective means of preventing further development of psychopathology, and on the other side of the coin, one cannot expect psychopathology to improve, as long as affect regulation does not.

An important message for clinicians and policy makers alike is that context matters, and that the parent-adolescent relationship continues to provide a central context in adolescence, but is not the only one. For clinicians, this implies that adolescent behavior should be interpreted in light of the relationship, family, and neighborhood context, and the possibility of including parents into treatment, or helping adolescents to deal differently with conflicts with their parents, should be assessed carefully. For policy makers, it means that they need to be aware of the fact that, while intervention efforts at the individual level may be helpful, efforts may also usefully be aimed at the family (e.g., support groups for single parents) and community levels (e.g., increasing informal social control through neighborhood activities that foster a sense of community).
ADOLESCENT AFFECT DYSREGULATION: DISCUSSION

Limitations and Future Directions

Results of the present thesis must be interpreted in light of its limitations. The first limitation concerns the samples used in the present thesis. Although the studies in the present thesis used several different samples from the Netherlands, Germany, and Scotland, all these samples contained mostly white adolescents (and their families), limiting the generalizability of our findings to adolescents from other ethnic background. In addition, it would also be interesting to see to what extent our findings can be replicated in samples of adolescents with clinical levels of internalizing and externalizing problems, as problems with affect regulation seem to have a significant impact in this group. For example, Tromp and Koot (2010) showed that affective instability of adolescents referred for mental health services was related to affective disorders, but also to self-harm behavior, and the ability to observe rules in the home and perform age-appropriate tasks.

Another main limitation of the studies presented in the present thesis is the heavy reliance on adolescent self-reports which may be influenced by the individual’s willingness or ability to report accurately on his or her behaviors. Though when possible, we combined self- and other reports (chapter 5), included census data (chapter 6), and made use of the experience sampling method (chapter 3 and 5) in addition to traditional self-report questionnaires, the main use of self-reports may have resulted in an overestimation of links between parent-adolescent interactions, adolescent affect dysregulation, and adolescent psychopathology due to shared-method variance (e.g., Fergusson & Horwood, 1987).

Though not a specific focus of the present thesis (since its focus is on dimensions of affective dysregulation), it is still regrettable that not more of a differentiation between affect and its regulation could be made. Because affect is modulated as or even before it is generated, and not only afterwards, neurobiological perspectives suggest that affect regulation should be investigated as part of affect itself (Thompson et al., 2008). The notion of affect and its regulation as undividable also receives support from functional approaches to emotion research (Campos, Frankel, & Camras, 2004). Nevertheless, there exists quite some agreement that both science and clinical work may profit from concepts that describe how individuals modulate their affective experiences (e.g., Cole & Deater-Deckard, 2009; Goldsmith, Pollak, & Davidson, 2008). Successful approaches to do so should combine different methods and levels of analyses as well as samples of typically and atypically developing children and adolescents (Cole & Deater-Deckard, 2009).

A final limitation of the present dissertation is the fact that not every topic addressed in this thesis could be studied longitudinally. For instance, affect dysregulation at the meta-cognitive level was only studied cross-sectionally. This is unfortunate, because cross-sectional studies do not allow any conclusions about the direction of effects, and because we cannot draw any conclusions about potential developmental changes of affect dysregulation at the meta-cognitive level throughout adolescence. Fortunately, longitudinal data on affective dysregulation at the meta-cognitive level obtained in the RADAR study (Eichelsheim, Buist, Deković, Wissink, Frijns, van Lier et al., 2010) are ready for analysis.
CHAPTER 7

As studies in the present thesis are among the few that focus on affective dysregulation, its consequences and sources in adolescence, it offers several ideas for potential research regarding the concept and development of affect dysregulation in adolescence, the foundations of adolescent affect dysregulation, the role of affect dysregulation in adolescent psychopathology, and potential moderators of its effects. Longitudinal studies of affect dysregulation in adolescence, that have the potential to show both general developmental trends and the emergence of intraindividual differences, will be important to further our understanding of affect dysregulation in this important developmental period. The present thesis shows that negative affect increases from age 13 to age 15 years. Important follow-up questions are: Is this increase paralleled by, or does it follow or is it followed by increases in other aspects of affect dysregulation? How does puberty impact affect dysregulation? What happens towards late adolescence: Does affect dysregulation level off, or decrease? In addition to the study of normative developmental changes in affect and indicators of affect dysregulation, affect regulation strategies should be studied. It would be highly informative to see studies assessing the development of several indices of affective development from late childhood to young adulthood, including experiential and meta-cognitive aspects of affect dysregulation, but also psychophysiological measures, and observational data. In addition, it may be worthwhile to design studies that identify changes in affective reactivity as well as changes in affect regulation strategies across adolescence.

Ultimately, studies of the normative developmental changes regarding affect and its regulation may prove of great use for the study of associations between affect dysregulation and psychopathology. Once we have a better picture of how affect regulation and dysregulation develops normally across adolescence, we can try to find early markers of affect dysregulation that indicate ‘when things turn to go wrong’. In addition, person-centered analyses may be used to differentiate youths low in affective reactivity from those that are high in affective reactivity but are able to regulate their affect in adaptive ways, from those that are at double jeopardy, because they are highly reactive and use inefficient affect regulation strategies. By including different aspects of affect, affect regulation and dysregulation in one study, one may also identify unique, additive and interactive effects of different dimensions of affective dysregulation on the development of distinct forms of psychopathology. For instance, using frequent assessments of emotions (several ratings daily over a course of 3-4 days) and a Stop Signal Reaction Task, Hoeksma and colleagues (Hoeksma, Oosterlaan, Schipper, and Koot, 2007) showed that anger accelerates less fast as inhibitory control increases, and that high anger variability is associated with low inhibitory control in a sample of preadolescent boys and girls. It would be interesting to see how anger (and other emotions) and inhibitory control develop across adolescence, and how they, and their interaction relate to the development of psychopathology.

Although in the present thesis, little evidence was found for specific associations between discrete emotions and different forms of psychopathology, it may still be worthwhile to study discrete emotions. Thereby, one may find answers to questions such as: What is it that leads from the experience of one emotion to its expression, possibly masked as another emotion? In this context, it would also be interesting to compare clinical and non-clinical samples. While our data
imply that the experience of discrete emotions is not linked with different forms of psychopathology in specific ways, this may differ at extreme ends of the continuum, i.e., for clinically high symptom levels.

Regarding sources of affect dysregulation, the present thesis shows that negative affect arises in the context of negative parent-adolescent interactions. For future research, it would be interesting to see whether and how other family relationships, for instance relationships with siblings or the marital relationship are associated with adolescent affective dysregulation. Since the social context outside the family gains in importance in adolescence, future studies may also focus on links between peer- and intimate partner relationships and affect (dys)regulation. Because of the high variability of findings relating environmental influences to child development (e.g., Rothbaum & Weisz, 1994), an especially promising avenue for future research is the study of combined genetic and environmental influences on affect regulation (e.g., Zimmermann, Mohr, & Spangler, 2009). Amongst others, variations in genes related to the dopamine (e.g., Drabant, Hariri, Meyer-Lindenberg, Munoz, Mattay, Kolachana et al., 2006) and serotonin systems (e.g., Canli & Lesch, 2007) have been associated with interindividual differences in affect regulation. It is well established however, that individuals carrying vulnerability genes may only develop mental health problems, if they are additionally exposed to significant environmental stressors (e.g., Caspi, McClay, Moffitt, Mill, Martin, Craig et al., 2002; Hariri & Forbes, 2007). One interesting question for developmental studies might be whether affectivity is mostly genetically determined, whereas regulation of affective responses and coping are more influenced by socialization experiences. If that was the case, positive socialization experiences may ameliorate the influence of risk genes. Given that genetic risk might be better conceptualized as genetic susceptibility to environmental influences (Belsky & Pluess, 2009), both positive and negative aspects of the environment and both positive and negative developmental outcomes should be studied.

Conclusions

The present thesis clearly demonstrates that affect dysregulation is a risk factor for the development of mental health problems in adolescence. It may both mediate the effects of social experiences on the development of psychopathology, or be moderated by environmental influences. The present thesis points out that affect dysregulation is a multidimensional construct, and offers ways to assess several of these dimensions reliably in research with adolescents. The need to approach affect dysregulation at diverse levels of experience is stressed by the fact that the precise nature of associations between affect dysregulation and psychopathology depends on the conceptualization of affect dysregulation and the form of psychopathology in question. Results of the present thesis show that in this regard, the form of dysregulation may be more important than the discrete emotion under study, at least when it comes to the conscious experience of emotions. Consequently, scientists and clinicians alike should be aware that diverse emotional experiences may underlie any form of psychopathology.
CHAPTER 7

Further, the present study clearly demonstrates that parent-adolescent interactions form an important context for adolescent mental health and affect dysregulation, and that associations between parent-adolescent interactions and adolescent outcomes are best characterized as bi-directional or closely co-developing. It appears that the development of parent-adolescent interactions may not always be associated with the development of internalizing and externalizing problems directly, but indirectly through adolescent affective dysregulation. This underscores the necessity to include potential intervening variables in studies of associations between contextual factors and developing psychopathology.

Finally, a factor that should not be underestimated when studying adolescent affect dysregulation is adolescent gender. This might be especially true for studies focusing on affect dysregulation in the context of social relationships: Results of the present thesis suggest that adolescent gender affects links between the social relations and the broader social context and adolescent development, whereas links between affect dysregulation and psychopathology might be more gender-invariant. One interesting exception to the latter may be a role of internalizing emotions (anxiety, sadness) in the development of aggressive behavior for female, but not for male adolescents. However, this finding clearly needs to be replicated in different samples.

All in all, the studies reported in this thesis evidence that early adolescent affect dysregulation is an important construct that deserves ample attention from researchers and clinicians alike. Simultaneously, they only scratch the surface of this phenomenon, and indicate that much more work needs to be done to understand its nature and influence.
Dysregulatie van Affect en Psychopathologie bij Adolescenten in de Gezinscontext

(Samenvatting)
Affect heeft een belangrijke functie in de organisatie van gedrag. Affect moet en kan in principe gereguleerd worden, als dit voor iemands doelen van belang is. Dysregulatie van affect verwijst naar maladaptieve patronen van de regulatie van affect, dat wil zeggen naar patronen die iemands lange termijn doelen ondermijnen. Dysregulatie van affect is onderliggend aan veel vormen van psychopathologie. Dit geldt zowel voor internaliserende vormen van psychopathologie, zoals angst en depressie, als ook voor externaliserende vormen, zoals agressief en crimineel gedrag. Hoewel affect een reflectie van neurologische activiteit is, wordt het tegelijkertijd ook beschreven als een sociaal fenomeen: als input voor en uitkomst van sociale interacties. Bovendien gaan veel onderzoekers op het gebied van de socialisatie van affect ervan uit, dat de regulatie van affect in sociale interacties met belangrijke partners wordt aangeleerd, en dat in de kindertijd en jeugd de ouder-kind relatie hierbij van bijzonder belang is. Affect regulatie vaardigheden, op hun beurt, kleuren ook de sociale interacties die iemand heeft. Belangrijke ontwikkelingen op sociaal en emotioneel gebied maken de adolescentie een belangrijke periode voor de studie van associaties tussen affect dysregulatie en psychopathologie in de sociale context: met de aanvang van de adolescentie nemen de ervaring van negatief affect en de variabiliteit van affect toe, beginnen psychische problemen zich vaker voor te doen, en verandert bovendien de relatie tussen ouders en hun adolescente kinderen. Tenminste tijdelijk nemen conflicten tussen ouder en kinderen in deze periode toe en wordt ouderlijke steun minder.

Ondanks het feit dat de adolescentie dus een periode van belangrijke sociale, emotionele en psychische veranderingen is, bestaat er nog Weinig onderzoek op het gebied van affect dysregulatie en de oorzaken en consequenties ervan in de adolescentie. Het voorliggende proefschrift beoogt het fenomeen affectieve dysregulatie in de vroege tot midden adolescentie nader te beschrijven, en gevolgen voor de ontwikkeling van psychische problemen en mogelijke invloeden van ouder-kind interacties te bestuderen. Specifieke doelen zijn:

1. Associaties tussen verschillende vormen van affect dysregulatie en verschillende vormen van psychopathologie te bestuderen. Omdat affect dysregulatie zich op verschillende manieren kan manifesteren, werd rekening gehouden met verschillende vormen van affect dysregulatie. Specifiek werd gekeken naar affect dysregulatie op het meta-cognitief gebied (zelf-ervaren problemen met de regulatie van affect), en affect dysregulatie zoals tot uiting komt in de dynamiek (de intensiteit en variabiliteit) van ervaren emoties. Op het gebied van de dynamiek van emoties werd aandacht besteed aan de vraag of dysregulatie van specifieke discrete emoties verbonden is aan specifieke vormen van psychopathologie (zoals verdriet met depressie of boosheid met agressief gedrag) of dat associaties tussen dysregulatie van affect en verschillende psychologische problemen eerder aspecifiek van aard is.

2. De betekenis van de ouder-adolescent relatie voor affectieve dysregulatie en de ontwikkeling van psychopathologie bij de adolescent te bestuderen. Specifieke vragen zijn:
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a. Is affectieve dysregulatie geassocieerd met de kwaliteit van de ouder-adolescent relatie?
b. Is de kwaliteit van de ouder-adolescent relatie direct of indirect (via de dysregulatie van affect) aan de ontwikkeling van internaliserende en externaliserende problemen verbonden?


De studies die het voorliggende proefschrift uitmaken, zijn gebaseerd op vier steekproeven van adolescenten (en hun ouders) uit drie verschillende landen (Nederland, Duitsland en Schotland). De studies in hoofdstuk 2 en 3 beschrijven associaties tussen affect dysregulatie en psychopathologie. In hoofdstuk 4 worden associaties tussen moeder-kind interacties en problemen met affect dysregulatie van de adolescent beschreven. De studies in hoofdstukken 5 en 6 zijn gericht op het samenspel van affect dysregulatie en de kwaliteit van ouder-adolescent interacties in de ontwikkeling van psychische problemen van de adolescent. In hoofdstuk 6 worden deze associaties onderzocht rekening houdend met gezins- en buurtkenmerken.

Hoofdstuk 2: Psychometrische kenmerken van de Difficulties in Emotion Regulation Scale in een steekproef adolescenten

In hoofdstuk 2 werden cross-sectionele associaties tussen zelf waargenomen problemen met affect regulatie en internaliserende (angst en depressie) en externaliserende (agressief en delinquent gedrag) vormen van psychopathologie onderzocht in een steekproef van meer dan 700 Nederlandse scholieren. Hiervoor werd gebruik gemaakt van een vragenlijst, die voor onderzoek van affect regulatie problemen met volwassenen werd ontwikkeld, de Difficulties in Emotion Regulation Scale (DERS). Een hoofdresultaat van de studie is dat de dimensies van affect regulatie problemen, die de DERS bij volwassenen met, ook bij adolescenten goed kunnen worden onderscheiden. Deze conclusie wordt ondersteund uit resultaten van confirmatieve factoranalyses, maar ook door het feit dat verschillende dimensies van waargenomen affect regulatie problemen op specifieke manier met vormen van psychopathologie samenhangen. Zo spelen een lage emotionele helderheid (d.w.z. het niet goed weten wat men eigenlijk voelt), het niet accepteren van emotionele reacties en een lage ervaren effectiviteit op het gebied van de emotie regulatie een rol bij symptomen van angststoornissen en depressie. Agressief gedrag is verbonden aan problemen met impulscontrole en met moeite met doelgericht gedrag in emotionele situaties. Delinquent gedrag is alleen verbonden aan het hebben van weinig aandacht voor emoties. Verder laat deze studie zien dat vrouwelijke adolescenten doorgaans meer problemen met affect regulatie rapporteren dan
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mannelijke adolescenten. Hierbij moet men echter rekening houden met het feit dat sommige van deze verschillen gedeeltelijk toe te schrijven zijn aan verschillende interpretaties van de items van de DERS door vrouwelijke en mannelijke adolescenten.

Hoofdstuk 3: De dynamiek van emoties en psychopathologie bij adolescenten

Net zoals in hoofdstuk 2 worden in hoofdstuk 3 associaties tussen affect dysregulatie en psychopathologie onderzocht. In hoofdstuk 3 gaat het echter niet om affect dysregulatie in de vorm van waargenomen affect regulatie problemen, maar in de vorm van de intensiteit en variabiliteit van vier discrete emoties (vreugde, woede, angst en verdriet) en hun longitudinale associaties met symptomen van angststoornissen en depressie en agressief gedrag. Resultaten van padmodellen, die de rol van de intensiteit en variabiliteit van de vier emoties in de ontwikkeling van psychopathologie in de tijdspanne tussen 13 en 14 jaar bestuderen, laten zien dat alle vier emoties een rol spelen in veranderingen in symptomen van angststoornissen en depressie. Met betrekking tot de ontwikkeling van agressief gedrag werd gevonden dat, hoewel angst en verdriet ook een rol spelen, boosheid zeker de hoofdrol speelt. De variabiliteit van emoties speelde een groter rol dan hun intensiteit in de ontwikkeling van symptomen van angststoornissen, terwijl de intensiteit van emoties een grotere rol speelde dan hun variabiliteit in de ontwikkeling van depressieve symptomen en agressief gedrag. De associaties tussen affect dysregulatie en psychopathologie waren in het algemeen hetzelfde voor vrouwelijke en mannelijke adolescenten. Uitzonderingen hierop zijn het feit dat angst en verdriet een rol speelden in de ontwikkeling van agressief gedrag bij meisjes, maar niet bij jongens.

Bij elkaar genomen laten de studies in hoofdstukken 2 en 3 zien dat verschillende dimensies van affect dysregulatie inderdaad verbonden zijn aan internaliserende en externaliserende problemen bij jongeren. De studies wijzen erop dat specifieke vormen van affect dysregulatie onderliggend zijn aan verschillende vormen voor psychopathologie. Zo lijkt het bijvoorbeeld, dat symptomen van angststoornissen en depressie verbonden zijn aan weinig emotionele helderheid, het niet-accepteren van emotionele responsen en een lage ervaren effectiviteit op het gebied van de emotie regulatie, maar dat symptomen van angststoornissen verder verbonden zijn met een hoge variabiliteit van emoties, terwijl symptomen van depressie verbonden zijn aan hoge niveaus van negatieve en lage niveaus van positieve emoties. Er werd weinig evidentie gevonden voor de hypothese dat discrete emoties specifiek verbonden zijn aan verschillende vormen van psychopathologie. Geslachtsverschillen in de mate van affect dysregulatie bestaan vooral op het gebied van zelf-waargenomen affect regulatie problemen, en niet op het gebied van de intensiteit en variabiliteit van gevoelens. Associaties tussen affect dysregulatie en psychopathologie verschillen weinig voor mannelijke en vrouwelijke adolescenten, maar het lijkt belangrijk om in toekomstige studies aandacht te besteden aan mogelijke geslachtsverschillen in associaties tussen affect dysregulatie en psychopathologie.
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Hoofdstuk 4: Emotieregulatieproblemen en moeder-adolescent interacties

In hoofdstuk 4 werden cross-sectionele associaties tussen moeder-adolescent interacties en waargenomen problemen met affect regulatie, zoals gemeten met de DERS, in steekproeven van Duitse scholieren bestudeerd. In studie 1 in dit hoofdstuk werden associaties tussen opvoeding en affect regulatie problemen bestudeerd, terwijl 2 gericht was op associaties tussen negatieve interacties en steun in de ouder-kind interactie. Resultaten van studie 1 tonen aan dat hoge ervaren controle door de moeder positief samenhangt met affect regulatie problemen. Dit geldt vooral voor psychologische controle, in mindere mate echter ook voor gedragscontrole. Resultaten van studie 2 laten zien dat negatieve interacties tussen moeder en adolescent positief samenhangen met affect regulatie problemen, terwijl ervaren steun door de moeder negatief samenhangt met affect regulatie problemen. Associaties tussen gedragscontrole, negatieve moeder-adolescent interacties en steun van de moeder aan de ene kant en affect regulatie problemen aan de andere, verschillen voor mannelijke en vrouwelijke adolescenten, met sterkere associaties voor vrouwelijke dan mannelijke adolescenten. De twee studies geven dus aanwijzingen dat interacties tussen moeders en kinderen ook in de leeftijd van 12 tot 19 jaar nog van betekenis zijn voor de affect regulatie van de kinderen. Geslachtsverschillen in associaties tussen moeder-kind interacties en affect regulatie problemen zouden de uitkomst kunnen zijn van geslachtspecifieke socialisatie van emoties, waarbij ouderen hun dochters eerder relatie-oriënteerde emotie regulatie strategieën laten zien, en hun zoons meer actieve en instrumentele strategieën.

Hoofdstuk 5: Parallele ontwikkelingen in de ouder-adolescent relatie, negatieve emoties en psychopathologie

In de studies in hoofdstuk 5 werd onderzocht (1) in hoeverre de ontwikkeling van negatieve interacties en steun in de relatie tussen adolescenten en hun moeders en vaders aan de ene kant en van internaliserende en externaliserende vormen van psychopathologie aan de andere kanten (2) na te gaan in hoeverre samenhangen tussen ontwikkelingen in relatie kwaliteit en psychopathologie direct met elkaar verbonden zijn, of indirect verlopen, via ontwikkelingen in de ervaring van negatief affect door de adolescent. Daartoe werden gegevens van 452 Nederlandse adolescenten en hun moeders en vaders, gevolgd van 13 tot 15 jaar, met behulp van latente groei curve modellen geanalyseerd. Veranderingen in ouderlijke steun waren niet verbonden aan veranderingen in internaliserende of externaliserende psychopathologie van de adolescent. Echter, er werden significante associaties gevonden tussen ontwikkelingen in negatieve interacties van de adolescent met moeders en vaders en de ontwikkeling van fysieke agressie, maar niet van symptomen van gegeneraliseerde angststoornis. Het bleek dat als negatieve interacties toenemen, neemt ook fysiek agressief gedrag toe. Voor negatieve interacties tussen moeder en adolescent werd deze associatie volledig gemedieerd door toename in negatief affect van de adolescent. De associatie tussen ontwikkelingen in de vader-adolescent relatie en de ontwikkeling van fysieke agressie was echter direct, d.w.z. er was geen mediërende rol van negatief affect van de adolescent in deze associatie.
De studie benadrukt dus het belang van het bestuderen van de rol van de moeder-adolescent als ook de vader-adolescent relatie in de ontwikkeling van jongeren en de potentiële verschillende paden via welke zij de ontwikkeling van hun adolescente zoon of dochter mogelijk beïnvloeden.

Bij elkaar genomen laten de studies in de hoofdstukken 4 en 5 zien dat ouder-adolescent interacties ook bij jongeren een belangrijke context voor affect regulatie en dysregulatie vormen, en dat affect dysregulatie een mogelijke weg is via welke de ouder-adolescent relatie samenhangt met de ontwikkeling van psychopathologie in de adolescence. Verder wordt het belang van het geslacht van jongeren zowel als ouders in associaties tussen sociale interacties en de psychische ontwikkeling van jongeren geaccentueerd.

**Hoofdstuk 6: Impulsiviteit, ouderlijke toezicht en antisociaal gedrag van adolescenten in de bredere sociale context**

De studie in hoofdstuk 6 beschrijft het samenspel van dysregulatie van gedrag (hoge impulsiviteit) en toezicht door de ouders (parental knowledge) op de ontwikkeling van antisociaal gedrag in de leeftijd van 12 tot 15 jaar in de context van het gezin (een- versus twee-ouder gezinnen) en de buurt (economische benadeeld, en lage informele sociale controle). Hiervoor werden gegevens van een Schotse steekproef van meer dan 4000 jongeren gebruikt. Het leven in een een-ouder gezin en hoge impulsiviteit op leeftijd 12 voorspelden significant het niveau van antisociaal gedrag en ouderlijk toezicht op leeftijd 14 voor zowel mannelijke als vrouwelijke adolescenten. Buurtkenmerken voorspelden antisociaal gedrag en ouderlijk toezicht voor mannelijke adolescenten. Voor vrouwelijke adolescenten werden significante interacties tussen impulsiviteit en economische nadeel van de buurt en impulsiviteit en gezinstype op de ontwikkeling van ouderlijk toezicht gevonden. Deze interactie effecten kunnen worden geïnterpreteerd als steun voor de hypothese dat de impulsiviteit van vrouwelijke adolescenten de mate van ouderlijk toezicht sterker beïnvloedt in omgevingen met laag risico (twee-ouder gezinnen, geen economische achterstand in de buurt) dan omgevingen met een hoog risico (een-ouder gezinnen, wel economische achterstand in de buurt). Effecten van impulsiviteit en contextuele risicofactoren op de ontwikkeling van antisociaal gedrag werden gedeeltelijk verklaard door hun negatieve samenhang met ouderlijk toezicht. Vrouwelijke adolescenten profiteerden significant sterker dan mannelijke adolescenten van hoge niveaus van ouderlijk toezicht.

Bij elkaar laten de studies in hoofdstukken 5 en 6 zien dat ouder-adolescent interacties zowel een risico voor affectieve dysregulatie van de adolescent en daarmee verbonden ontwikkeling van psychopathologie kunnen zijn, als ook een protectieve factor in associaties tussen affectieve en gedragsdysregulatie van de adolescent en psychopathologie.

**Discussie**

Implicaties voor theorie, praktijk en beleidsvorming, en suggesties voor verder onderzoek worden in hoofdstuk 7 gepresenteerd. Resultaten van dit proefschrift dragen bij aan ons begrip van
affect dysregulatie in de adolescentie door intraindividuele stabiliteit en normatieve ontwikkelingen in de dynamiek van emoties te laten zien en door de identificatie van klinisch relevante dimensies van waargenomen affect regulatie problemen bij adolescenten. Bovendien wordt aangetoond dat de DERS en dagelijkse beoordelingen van affect via internet betrouwbare manieren zijn om affect dysregulatie bij adolescent te meten en dat deze op voorspelbare wijze met psychopathologie en ouder-adolescent interacties samenhangen.

Dit proefschrift toont aan dat affect dysregulatie en psychopathologie in de adolescentie ook longitudinaal samenhangen. De resultaten wijzen erop dat affect dysregulatie en psychopathologie sterk met elkaar verbonden zijn, zodat beide zowel een bron als een uitkomst voor de ander zijn. Dit geeft aan dat verbetering van affectregulatie kan bijdragen aan verbetering van psychopathologie bij adolescenten. Verder laat dit proefschrift zien dat, hoewel discrete emoties weinig specifiek met vormen van psychopathologie samen hangen, verschillende vormen van psychopathologie toch door verschillende profielen van problemen met affect dysregulatie gekenmerkt zijn. Toekomstig onderzoek zou verschillende vormen van affect dysregulatie en hun samenhang met de ontwikkeling van psychopathologie in één studie kunnen combineren, om de verschillende affect dysregulatie profielen, die aan verschillende vormen van psychopathologie onderliggend zijn beter te beschrijven. Deze profielen zouden uiteindelijk voor het samenstellen van effectieve preventieve en interventie programma’s gebruikt kunnen worden.

De ouder-kind relatie werd vaak als belangrijke context voor de ontwikkeling van affect regulatie en dysregulatie in de kindertijd beschreven. Dit proefschrift toont aan dat dit ook in de vroege tot midden adolescentie het geval is en dat (1) de associaties tussen de ouder-adolescent relatie en affect regulatie het best als bidirectioneel kunnen worden beschreven, en (2) dat sommige van deze associaties sterker zijn voor vrouwelijke dan voor mannelijke adolescenten. Verder lieten we zien dat affect dysregulatie een potentiële mediator is in associaties tussen ouder-adolescent interacties en de ontwikkeling van psychopathologie. Tegelijkertijd moet benadrukt worden dat positieve ouder-adolescent interacties ook als protectieve factor in associaties tussen individuele en contextuele risicofactoren en de ontwikkeling van psychopathologie kunnen werken. Deze bevindingen impliceren dat clinici het gedrag van de adolescent ook in het licht van relatie-, gezins- en buurtfactoren moeten beoordelen en dat het includeren van de ouders in de behandeling zou moeten worden overwogen. Beleidsmakers moeten zich ervan bewust zijn, dat, hoewel individuele interventies kunnen helpen, interventies gericht op het niveau van gezin en buurt ook behulpzaam kunnen zijn.

Een beperking van dit proefschrift bestaat in de gebruikte steekproeven, die doorgaans uit Europese middenklasse adolescenten (en hun ouders) bestonden, waardoor de conclusie uit de verschillende studies niet naar adolescenten met andere achtergronden en met klinische niveaus van psychische stoornissen gegenereerd kunnen worden. Een andere beperking is dat er veel gebruikt gemaakt werd van zelf-rapportage vragenlijsten, die beïnvloed kunnen worden door iemands bereidheid en vermogen om accuraat zijn gedrag en gevoelens te beschrijven. Verder zou hierdoor de sterkte van gevonden associaties overschat kunnen zijn. Verder is het spijtig, dat niet alle studies in deze proefschrift longitudinaal zijn, waardoor wat betreft sommige constructen, geen
uitspraken over oorzaak en gevolg kunnen worden gedaan. Hoewel ook het geen specifieke doelstelling van dit proefschrift was, is het toch ook een beperking dat weinig onderscheid werd gemaakt tussen affect en de regulatie ervan. Toekomstig onderzoek zou verschillende maten van affect en affect regulatie kunnen combineren om unieke, additieve en interactie-effecten tussen affect en affect regulatie op de ontwikkeling van psychopathologie in de adolescentie te onderzoeken. Verder onderzoek zou ook nog gedaan moeten worden met betrekking tot normatieve ontwikkeling van affect en affect regulatie en dysregulatie in de adolescentie. Als we een beter beeld hebben van de normatieve ontwikkeling, zijn vroege indicatoren van ontwikkelingen, die echt problematisch zijn, makkelijker te vinden. Op het gebied van bronnen van affect dysregulatie, zou het bijzonder productief kunnen zijn om interacties tussen gen- en omgevingsinvloeden te bestuderen.
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Dankwoord

Dankwort

Acknowledgments
Bedankt Hans, voor je neverending enthousiasme, jouw overzicht, je bereidheid om steeds weer met mij in discussie te gaan, altijd met plezier en altijd inspirerend; voor jouw Verstand und Verständnis.

Bedankt Pol, voor jouw kritische blik, “Geschäftssinn”, gepaard met enthousiasme, jouw altijd open deur en oor, jouw grappen en verhalen.

Bedankt Hans en Pol. Ik had het niet beter kunnen treffen.


Dank aan mijn collega’s van RADAR voor de mogelijkheid om aan dit project mee te werken. Wim, Tom, Endy, Pieter-Bas: ik heb niet alleen heel veel geleerd, het was ook nog erg gezellig!

Dank aan de (oud-)collega’s van de VU voor de gezelligheid. Dank aan Agnes, Cristina, Evelien, Maartje, Miranda en Noor voor de fantastische tijden in Boston, New York, Florence, Chicago, Würzburg en Denver.

Thanks to Barbara Maughan, for giving me the opportunity to work with the ESYTC data reported in chapter 6. It was great to be in London and profit from your expertise.

Danke an meine neuen Kollegen an der Bergischen Universität Wuppertal für die freundliche Aufnahme. Danke Peter, für die Möglichkeit zurück ins Rheinland zu gehen und dort die Arbeit an dieser Dissertation zu beenden. Auf zu neuen Ufern!

Dank aan vrienden in Nederland, Deutschland and Berkeley: für verquatschte Tage und durchtanzte Nächte und erklommene Gipfel / voor eindeloos kletsen en dansen / for endless talking at day and dancing at night.


Special thanks to Ted Barker. For companionship throughout a large part of this journey.