

TOO MUCH, TOO LITTLE?
THE ROLES OF AFFECT DYSREGULATION AND DEFICIENT AFFECT IN
YOUTH VIOLENCE

by

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ABSTRACT

Children with high levels of dysregulated affect and negative reactivity experience a range of emotional and behavioral problems, including low levels of prosocial behavior, aggression and delinquency. Alongside this body of literature, research has also shown that abnormally low levels of affect and emotional reactivity – commonly termed ‘deficient affect’ – is also associated with aggression and violence. These diverging lines of research call attention to the complex role of emotion in aggressive behavior, and appear to support opposing hypotheses (i.e., dysregulated versus deficient affect as risk factors for aggressive behavior). The goal of this study was to clarify the contributions of affect dysregulation and deficient affect in predicting acts of aggression, violence and non-violent delinquency in high-risk youth. Structural equation modeling (SEM) was used to investigate the direct and interaction effects of affect dysregulation and deficient affect in a prospective study of aggression and antisocial behavior in a sample of 179 adolescents. Results support the notion that there are two separate routes to problem behaviors, and highlight the importance of identifying two “faces” of affective experience that give rise to aggressive behaviors among adolescents.

I would like to dedicate this dissertation to my parents who have provided constant and unconditional support of each and all of my endeavors.

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INTRODUCTION

Research has shown that there exists a significant amount of heterogeneity within groups of aggressive and antisocial youth. Children differ not only in terms of the types of aggression they manifest (e.g., overt versus covert behaviors; Coie & Dodge, 1998; Loeber & Schmaling, 1985), but also according to the age of onset and chronicity of their antisocial behaviors (e.g., life course persistent versus adolescent limited; Moffitt, 1993, 2006). Importantly, children and adolescents also differ with respect to the risk factors and etiological mechanisms that contribute to the onset of problem behaviors, and which are thought to shape the developmental course and manifestation of aggressive, violent, and antisocial behaviors (Burke, Loeber, & Birmaher, 2002; Loeber & Stouthamer-Loeber, 1998).

Affect regulation has received consistent empirical support as a salient risk factor in aggression and other behavioral problems among children and adolescents. Much of this research has investigated the relationship between under-regulated emotions (e.g., manifesting as high emotional arousal and reactivity) and negative outcomes (e.g., externalized behavior problems such as aggression). For instance, high levels of negative reactivity, defined as the “tendency to react strongly and consistently to environmental events with emotions of negative valence” (Frick & Morris, 2004, p. 58), have been associated with aggressive conduct problems among children (Caspi, 2000; Eisenberg et al., 1997, 2001; Hubbard et al., 2002) and are thought to emerge from basic difficulties with regulating affect.

Interestingly, however, alongside studies demonstrating a positive association between affect dysregulation, negative reactivity, and aggression is a growing body of research demonstrating both concurrent (Frick, Cornell, Bodin, et al., 2003; Loney, Frick, Clements, Ellis, & Kerlin, 2003) and prospective (Frick, Cornell, Barry, Bodin, & Dane, 2003) relationships between extremely *low* levels of emotional reactivity and aggressive behaviors in children and adolescents. In fact, abnormally low resting levels of arousal and emotional reactivity have been linked to aggressive behaviors in several studies (e.g., Lahey, Hart, Pliszka, Applegate, & McBurnett, 1993; Raine, 2002), and have also been implicated in the development of psychopathy – a personality syndrome that encompasses a constellation of affective, interpersonal, and behavioral characteristics such as a callous disregard for others, a lack of empathy, and a propensity toward highly impulsive and irresponsible behavior (Hare, 2003). In particular, the affective characteristics of psychopathy (i.e., callousness, remorselessness, and superficial emotions; collectively referred to as “deficient affect”; Cooke & Michie, 2001) are hypothesized to develop as a result of a distinct temperamental style characterized by low emotional reactivity and relative fearlessness to aversive stimuli and novel or threatening cues in the environment (Frick & Ellis, 1999; Frick, Cornell, Bodin et al., 2003).

The current research examines the roles of affect dysregulation and deficient affect in predicting aggression, violence and non-violent delinquency in a sample of high-risk youth. In light of the finding that heterogeneous risk factors along multiple pathways can lead to the development of aggression and antisocial behavior among children (Cicchetti & Cohen, 1995), a central goal of this research was to explore two significant risk factors for aggression (i.e., affect dysregulation and deficient affect) that appear to

signal the presence of distinct and perhaps diverging developmental trajectories towards aggressive and antisocial behaviors. Specifically, this research evaluates the possibility that there exist two separate routes to problem behaviors – one that occurs through a developmental failure to achieve adequate modulation of affective states and another which arises through the lack of sufficient affective arousal to provoke inhibition of aggressive and antisocial behaviors.

What is Affect Regulation and How is it Measured?

The emergence of affect regulation is regarded as a major developmental task that cuts across childhood and adolescence and has significant implications for psychological adjustment and emotional competence (Cicchetti, Ganiban, & Baret, 1991; Cole, Michel, & Teti, 1994). Developmental approaches to behavioral disorders involving aggression, violence, and non-violent delinquency have commonly emphasized the role of emotions and associated regulatory abilities. Similarly, in the adult literature, difficulties in regulating affect have been implicated in a range of psychological disorders, including the majority of non-substance related Axis I disorders and virtually all of the personality disorders on Axis II (Cicchetti & Cohen, 1995; Gross & Levenson, 1997).

Despite the prominence of affect regulation in theories of adult and child psychopathology, there has yet to be an agreed upon definition of affect regulation that cuts across all research in the area. Unlike behaviors, affective processes are more difficult to operationalize and must often be inferred from behavioral indicators. Furthermore, despite that affective and behavioral processes are “intricately and perhaps sometimes inextricably associated” (Eisenberg, Fabes, Guthrie, & Reiser, 2000, p. 138), it is important to define and operationalize affect regulation as an underlying process that

is distinctive from its affective or behavioral consequences. For instance, Eisenberg and colleagues explicitly differentiate between the regulation of internal (e.g., affect) and external (e.g., behavior) states in their definition of affect regulation. These researchers define affect regulation as “the process of initiating, maintaining, modulating, or changing the occurrence, intensity, or duration of internal feeling states and emotion-related physiological processes, often in the service of accomplishing one’s goals” (Eisenberg et al., 2000, p. 137). Perhaps more succinct is the definition offered by Shields and Cicchetti (1998); these authors define adaptive affect regulation as “the ability to monitor and modulate one’s affective arousal such that an optimal level of engagement with the environment is fostered” (p. 382-383). Others have defined successful regulation in similar ways, for example, as the “capacity to respond flexibly and strategically in emotionally arousing situations in order to engage in goal-directed social behavior” (Pope & Bierman, 1999, p. 336), and have distinguished affect regulation from the simple inhibition of problematic behaviors. Thus, while there are some differences between the descriptions that appear in the literature, most definitions of affect regulation highlight the individual’s capacity to identify, control, and modulate affect so that an optimal level of interpersonal functioning is achieved.

With respect to measurement, there have been several efforts to develop and validate instruments that assess affect regulation in both child and adult samples (Gross & John, 2003; Mayer & Stevens, 1994; Shields & Cicchetti, 1997; Zeman, Shipman, & Penza-Clyve, 2001), and it appears that adults and children can engage in comparable types of affect regulation strategies (Harris & Lipian, 1989; Harris, Olthof, & Terwogt, 1981). A common theme that emerges across studies is the conceptualization of affect

regulation as multidimensional: there are specific strategies that fall under the rubric of “affect regulation” and these can be investigated in relation to specific types of emotional and behavioral problems. For example, results from the investigations of Gross and John (1998, 2003) suggest that distinct strategies for affect regulation can be identified and studied (e.g., cognitive reappraisal, expressive suppression), and that certain strategies are more adaptive than others with respect to emotional and psychosocial functioning. Specifically, the use of suppression (defined as “a form of response modulation that involves inhibiting ongoing emotion-expressive behavior”; Gross & John, p. 349) was associated with avoidant behavior in close relationships, decreased awareness of emotions and experiences of positive emotions, rumination, and low self-esteem. In contrast, the use of cognitive reappraisal (defined as “a form of cognitive change that involves construing a potentially emotion-eliciting situation in a way that changes its emotional impact”; Lazarus & Alfert, 1964, ctd. in Gross & John, p. 349) was related to more effective attempts at mood repair, more frequent experiences and expression of positive emotion, closer relationships, likeability, and self-esteem.

Other studies have substantiated this claim, finding that strategies such as dysregulated expression (i.e., the undercontrol of emotional experience) and suppression (i.e., the inhibition of emotional experience) are associated with negative outcomes as compared to more adaptive coping strategies (Mayer & Stevens, 1994; Shields & Cicchetti, 1997; Zeman et al., 2001). It also appears that the undercontrol versus inhibition of emotional experience show divergent patterns of relationships with measures of emotional and behavioral functioning: whereas undercontrol is often linked with aggression and externalized behaviors, inhibition is typically associated with

outcomes such as depression or other internalizing problems (Gross & John, 2003; Shields & Cicchetti, 1997). Importantly, these types of findings substantiate the discriminant validity of different affect regulation strategies, which is an especially imperative feature to demonstrate given the lack of specificity which often surrounds the construct of affect regulation (Cole, Martin, & Dennis, 2004).

Despite the growing body of literature on affect regulation in child and adult samples, the majority of research studies in the field have been conducted in normative, healthy samples of children and adults (see Shields & Cicchetti, 1997, 1998 for exceptions). Consequently, relatively less is known about how affective processes such as dysregulation contribute to aggressive and violent behaviors in high-risk populations (Cole et al., 1994). This is unfortunate given that affect regulation likely plays a key role in understanding most major forms of developmental psychopathology (Cicchetti et al., 1991; Cole et al., 1994), and is a well-established factor in aggression and other behavioral problems (Dearing et al., 2002; Lahey et al., 1999). Thus, the investigation of affect regulation needs to be extended into high-risk and other atypical populations in order to substantiate findings from normative samples, and to further demarcate the negative consequences of poor affect regulatory skills across diverse groups.

The Role of Affect Dysregulation in Aggression and Developmental Psychopathology

Recent conceptualizations of child and adolescent psychopathology have increasingly emphasized difficulties regulating emotion (Bradley, 2000; Steinberg & Avenevoli, 2000), and many leading authorities in the field have proposed a direct link between affect dysregulation and externalizing behavior problems such as childhood aggression (Dearing et al., 2002; Lahey et al., 1999). In normative samples of children,

there are numerous studies in support of the idea that affect dysregulation is a key mechanism in aggression and other externalizing conduct problems (e.g., Eisenberg et al., 1997, 2001; Frick, Cornell, Bodin, Dane, Barry & Loney, 2003; Hubbard et al., 2002), and may also represent an important mediating factor between temperament and subsequent behavioral problems (Martin & Fox, 2006). Eisenberg and colleagues (2001), for example, found that internalizing and externalizing problems among school-aged children could be differentiated in terms of patterns of dispositional negative emotionality (i.e., anger, sadness, and fear), as well as behavioral and attentional regulation; specifically, children with externalizing problems were characterized by low attentional and behavioral regulation (e.g., attention focusing, inhibitory control), as well as increased anger proneness. Similarly, a study by Cole, Zahn-Waxler and Smith (1994) found an association between children's ability to regulate their expressiveness in social situations and the level of externalizing problems exhibited. In this study, the amount of negative emotion (e.g., anger) shown during a "disappointment task" in the presence of an adult examiner was predictive of general oppositionality and disruptive behavior (Cole, Zahn-Waxler, et al., 1994).¹

In adolescent samples, Silk and colleagues (2003) demonstrated that adolescents showing intense and labile emotions, paired with ineffective regulation skills, had higher levels of depressive symptoms and problematic behaviors including aggression. In this study, the use of ineffective regulation strategies such as "involuntary engagement" (i.e.,

¹ Although these studies and others appear to support a direct link between affect dysregulation and behavioral problems, it must also be noted that the variables used may not tap affect regulation skills specifically. For instance, the study conducted by Eisenberg and colleagues (2001) assessed negative emotionality alongside overt displays of behavioral and attentional regulation (e.g., impulse control, attention focus). And, although behavioral and attentional regulatory skills likely relate to affect regulation in important ways, it is important not to confound or infer affect regulatory processes from purely behavioral indicators.

rumination) and disengagement (i.e., denial) were associated with increased levels of depressive symptomatology and externalizing behaviors, highlighting the fact that a range of problematic outcomes aside from overt aggression may be associated with ineffective affect regulation (Silk, Steinberg, & Morris, 2003). Other studies have similarly demonstrated both concurrent (Loney, Frick, Clements, Ellis, & Kerlin, 2003) and prospective (Caspi, 2000; Caspi, Henry, McGee, Moffitt, & Silva, 1995) relationships between levels of emotional reactivity, impaired affect regulation, and both internalizing disorders and externalizing behavior problems.

The more limited body of research investigating emotion regulatory processes in adolescents at-risk for aggressive and delinquent behaviors (e.g., de Castro, Merk, Koops, Veerman, & Bosch, 2005; Lochman & Dodge, 1994; Shields & Cicchetti, 1998) has produced results which are largely concordant with the above literature, showing, for example, that highly aggressive youth possess fewer adaptive affect regulation strategies and exhibit higher levels of unregulated anger. In the study conducted by de Castro and colleagues (2005), boys referred for problems with aggression mentioned less effective affect regulation strategies (e.g., using distraction, or further aggression in response to negative affect) than did their non-aggressive counterparts, and were less likely to identify any strategy that could potentially be used to regulate their emotion. Furthermore, problems with affect regulation were able to account for a significant portion of the variance even after social information processing (SIP) variables (e.g., a bias towards interpreting neutral cues as aggressive or hostile; Crick & Dodge, 1994) were accounted for.

Lochman and Dodge's (1994) investigation assessed the relative contributions of social-cognitive variables in a sample of highly aggressive and violent adolescent boys. These variables, which included a mixture of both cognitive (e.g., interpretation and processing of cues, generating solutions) and affective (e.g., affect labeling) processes, were shown to relate to patterns of aggression in expected ways. Highly aggressive boys showed significantly greater deficits in perceiving social cues accurately and generating appropriate (i.e., non-aggressive) solutions to social dilemmas. Violent boys also had greater difficulty accurately labeling emotions (e.g., more often mislabeling fear as happiness), and the authors suggested that this may reflect a larger deficit with affect regulation such that these boys use an "arousal detour" (i.e., cognitively prepare themselves by mislabeling a negative emotion as more positive) to cope with affectively charged situations. This finding is particularly interesting as it suggests that weaknesses in affect regulation skills can facilitate the development of deficits in emotional awareness and understanding. Unfortunately, however, there has not been much recognition in the literature that the consequences of affect dysregulation may be more widespread than a simple increase in negative affect due to the undercontrol of one's emotions.

The Role of Deficient Affect in Developmental Psychopathology: Psychopathy

Authorities in the field of psychopathy have long acknowledged the centrality of the affective traits which are posited to lie at the "core" of the adult syndrome (Blackburn, 1998; Hare, 1998). Cleckley (1941, 1976) theorized that the majority of psychopathic symptoms were the result of a deep-seated affective deficit, manifesting in personality traits such as callousness, a lack of empathy and remorse, and shallow

emotional responses. Empirical support for this claim lies in studies showing that the affective traits associated with psychopathy are most accurate in discriminating psychopathic from non-psychopathic individuals (Cooke & Michie, 1997), and also show the highest cross-cultural consistency (Cooke & Michie, 1999). Furthermore, individuals scoring highly on validated measures of psychopathy tend to show distinct physiological patterns in response to upsetting or aversive stimuli, suggesting that the affective deficits manifested by psychopaths may be partially mediated by biological or genetic processes. In particular, studies have found a relationship between scores on the interpersonal/affective dimension of psychopathy and deficits in physiological reactions to emotional stimuli (e.g., startle reflex, electrodermal reactivity, limbic system activity; Benning, Patrick, & Iacono, 2005; Kiehl et al., 2001; Patrick, Bradley, & Lang, 1994; Verona, Patrick, Curtin, Bradley, & Lang, 2004). Investigators have thus suggested that psychopathic individuals may experience abnormalities in the processing of emotionally relevant material in the environment, leading to the expression of affective psychopathic characteristics.

Among younger samples, there is preliminary evidence to suggest that the affective features of psychopathy may be relatively stable (Frick, Kimonis, Dandreaux, & Farrell, 2003), heritable (Viding, Blair, Moffitt, & Plomin, 2005), and show an earlier age of onset compared to the interpersonal and behavioral features of the construct (Klaver, 2006). More generally, as the construct of psychopathy has been increasingly extended to children (Frick, Bodin, & Barry, 2000; Lynam, 1996) and adolescents (Forth, Kosson, & Hare, 2003), there is now emerging evidence to suggest that the affective features of psychopathy have significant utility for refining the heterogeneous population of

aggressive and antisocial youth. Specifically, it has been suggested that youth demonstrating features consistent with the affective dimension of psychopathy represent a particularly severe subgroup with respect to behavioral and emotional problems (Barry, Frick, DeShazo, McCoy, Ellis, & Loney, 2000; Blair, 1997).

Recent studies have also shown that indicators of deficient affect and decreased emotional reactivity are associated with aggressive and antisocial behaviors among children. The work of Frick and colleagues (e.g., Christian, Frick, Hill, Tyler, & Frazer, 1997; Frick, Cornell, Barry, et al., 2003; Frick, Cornell, Bodin, et al., 2003), for example, has found that children and adolescents with elevated levels of “callous-unemotional” (CU) traits such as a lack of guilt, impaired empathy, and constricted emotions, when compared to conduct-problem children without these traits, show a greater severity and variety of antisocial behaviors, as well as a reward-dominant response style and preference for thrill and adventure seeking activities. Children with CU traits have also been found to be less sensitive to cues of punishment, and generally less reactive to threatening and emotionally distressing stimuli (Barry et al., 2000; Blair, 1999; Frick, Cornell, Bodin, et al., 2003). In contrast, conduct-disordered children without elevated levels of CU traits more often show higher levels of emotional reactivity, anxiety, and attention-related problems (Barry et al., 2000; Frick, O’Brien, Wootton, & McBurnett, 1994; Loney et al., 2003). In samples of older adolescents, features of deficient affect have demonstrated importance for predicting overt and relational forms of aggression (Penney & Moretti, 2007), as well as chronic and severe patterns of antisocial behavior (Vincent, Vitacco, Grisso, & Corrado, 2003).

Findings from the adult literature support this claim, showing that the affective features of psychopathy appear to contribute uniquely to the prediction of aggressive and violent behaviors (i.e., after the effects attributable to the behavioral features of psychopathy such as impulsivity, irresponsibility, and stimulation seeking have been accounted for; Salekin, Rogers, & Sewell, 1996). At the same time, however, other studies utilizing both adolescent and adult samples suggest that the behavioral dimension of psychopathy, rather than the interpersonal or affective components, may be primarily responsible for psychopathy's robust association with general and violent recidivism (Corrado, Vincent, Hart, & Cohen, 2004; Skeem & Mulvey, 2001). Thus, although certain studies find that the affective dimension of psychopathy accounts for a significant and unique proportion of variance in predicting common outcome variables (e.g., violence, recidivism, institutional misconduct), other studies show that these effects are substantially smaller in magnitude when compared to the behavioral indicators of psychopathy (e.g., Walters, 2003).

Affect Regulation, Deficient Affect, and Antisocial Behavior: Towards an Integrated Model

There exists a significant degree of conceptual and empirical overlap between the developmental construct of affect regulation and the deficient affect symptom cluster of psychopathy. Affect regulation is implicated in the development of a range of competencies (e.g., morality and empathic concern [Blair, 1999; Kochanska, 1997], social cognitive skills [Dodge & Pettit, 2003], successful peer relationships [Rubin, Bukowski, & Parker, 1998]), many of which are compromised in samples evidencing high levels of psychopathic personality features. Furthermore, affect dysregulation is

associated with several maladaptive outcomes (e.g., deficits in guilt and empathy) and criterion variables (e.g., aggression, delinquency) that comprise integral parts of the psychopathy construct.

Despite the relevance of research on affect regulation for understanding psychopathy (particularly the affective features of the syndrome), to date these two fields of research have progressed largely independent from each other. Consequently, we have little understanding of how affect regulation and psychopathy may be related, nor of how components of each construct may contribute separately or together to the prediction of aggressive, violent, or antisocial behaviors among adolescents. Eisenberg and colleagues' (Eisenberg et al., 1996, Eisenberg, Cumberland, & Spinrad, 1998) research on the deleterious effects of affect dysregulation on the development of empathy provides a point of departure towards addressing these issues. These investigators proposed that the association between affect dysregulation and aggression is due primarily to the negative effect of overarousal on empathic processes; specifically, Eisenberg proposes that poor affect regulation impedes the development of empathy to the extent that the vicarious experience of emotion is perceived as overwhelming and aversive. For individuals with deficient regulation skills and intense emotions, the arousal induced by emotional stimuli can lead to a self-focus that is largely incompatible with empathic responses and quality social interactions (Cohen & Strayer, 1996; Eisenberg et al., 1996, 1998). Instead, overarousal may impede a person from processing important emotional and social cues from their environment (Hoffman, 1983), and can result in low levels of social and emotional competence (Eisenberg & Fabes, 1992; Lopes, Salovey, Cote, & Beers, 2005).

This line of reasoning begins to bridge the literatures on affect regulation and psychopathy by proposing a novel developmental model whereby difficulties with affect regulation facilitate the expression of psychopathic features such as a lack of empathy and emotional awareness, which in turn may contribute to aggressive and antisocial behaviors. Alternatively, however, affect dysregulation and deficient affect may represent two separate, and perhaps even mutually exclusive, risk factors for aggression and violence. This latter model is consistent with the increasing recognition that aggressive youth represent a heterogeneous population, and the hypothesis that these youth differ with respect to the etiology of their behaviors (Dodge & Coie, 1987; Loeber & Stouthamer-Loeber, 1998). Of note, however, the view that there are different routes towards aggression is not inconsistent with the developmental model proposed by Eisenberg and colleagues (1996, 1998), since this model does not expect *all* youth to transition from manifesting dysregulated affect to showing features of deficient affect. Rather, this model proposes that only certain youth will undergo this transition, while others will continue to demonstrate difficulties with affect dysregulation into late adolescence and adulthood. Thus, regardless of the underlying developmental model that one subscribes to, the overarching expectation is that there will exist significant heterogeneity with respect to the emotional functioning of aggressive and antisocial youth.

The Current Study

There is a large body of research demonstrating that children with compromised affect regulation skills and heightened levels of negative reactivity experience a range of emotional, behavioral, and psychosocial dysfunctions (Bower, 1992; Gross & Munoz,

1995; Larsen, 2000). On the other side of the spectrum, studies have demonstrated a link between very low levels of arousal, low emotional reactivity, and aggression (Frick, Cornell, Bodin, et al., 2003). Furthermore, it has been hypothesized that features of low reactivity may signal the development of psychopathy – a construct which embodies characteristics of low or deficient emotionality (Cleckley, 1976) and shows robust associations with aggressive and violent behaviors (Forth et al., 2003; Hare, 2003). While these diverging lines of research may reflect the fact that affect dysregulation and deficient affect represent distinct pathways to aggressive and antisocial behavior, this idea has yet to receive empirical scrutiny. More broadly, the importance of identifying diverse trajectories to behavior problems which encompass distinct risk factors continues to represent an important research endeavor with implications for tailoring effective prevention and intervention efforts.

In order to begin addressing these issues, the present study investigated the joint contributions of affect dysregulation and deficient affect in predicting acts of aggression, violence, and non-violent delinquency. Two types of affect dysregulation were investigated (affect dyscontrol and affect suppression) in order to provide a test of divergent validity and to begin elucidating which specific regulatory mechanisms contribute most to aggression and antisocial behavior. Within each model, the bivariate association between affect dysregulation and deficient affect was assessed to determine whether there exists significant shared variance between these constructs in their relation to outcomes. Additionally, a wide range of dependent variables was included in order to substantiate results across diverse outcomes, as well as allow for the possibility that affect dysregulation and deficient affect may show divergent patterns of relationships across

different variables (e.g., reactive versus instrumental aggression, violent versus non-violent offending). For example, reactive aggression is typically associated with a high degree of sympathetic arousal and angry reactivity, whereas instrumental aggression is conceptualized as an offensive and methodical type of aggression (e.g., Dodge & Coie, 1987; Little, Jones, Henrich, & Hawley, 2003). Thus, affect dysregulation (particularly affect dyscontrol) may show a stronger association with reactive aggression, whereas deficient affect may be more strongly associated with instrumental aggression.

Lastly, moderation analyses were conducted in order to explore whether affect dysregulation and deficient affect interact to create a heightened (or lowered) risk to engage in aggression, violence, or non-violent delinquency. Other investigators have proposed relatively complex developmental models to trace the interaction of these constructs over time (Eisenberg et al., 1996, 1998), and it is possible that the effects of affect dysregulation and deficient affect continue to interact into adolescence and influence an adolescent's risk to engage in aggressive and antisocial behaviors. At first glance, affect dysregulation and deficient affect appear to be opposite and perhaps mutually exclusive constructs; however exploring the conditions in which they may interact can further clarify the diverse affective experiences that may give rise to aggression and antisocial behavior among adolescents. For example, it may be the case that youth with high levels of deficient affect, paired with erratic and intense surges of unregulated negative affect, will exhibit a disproportionately higher risk to engage in aggression and violence.

In sum, three central research questions were proposed: (1) Do affect dysregulation and deficient affect each relate to concurrent indices of aggression,

violence, and non-violent delinquency? (2) Do these same constructs predict *future* acts of aggression, violence, and non-violent delinquency? (3) Is there evidence that affect dysregulation and deficient affect represent two separate routes to antisocial behaviors, such that there is little to no shared variance between them? Alternatively, is there evidence that the constructs of affect dysregulation and deficient affect interact such that the effects of one construct vary across different levels of the other? Consistent with the idea that there exists significant heterogeneity within samples of aggressive and antisocial youth, it was hypothesized that both affect dysregulation and deficient affect would show significant relationships to each of the dependent variables while being marginally related to each other. Additionally, it was hypothesized that affect dyscontrol, rather than affect suppression, would show significant relationships with each of the outcome variables. Lastly, it was hypothesized that affect dysregulation (specifically affect dyscontrol) would show a stronger association with reactive aggression, whereas deficient affect would show a stronger association with instrumental aggression.

METHOD

Overview

The current research protocol was administered during the course of an ongoing research study examining gender and aggression in high-risk youth. Semi-structured interviews and self-report measures were administered to a sample of incarcerated juveniles and adolescents from a provincial mental health assessment center to examine various psychosocial factors that contribute to the prediction and development of aggression.

Participants and Procedure

Participants at Time 1 were 179 adolescents (97 males, 82 females) between the ages of 12 and 18 ($M = 15.3$, $SD = 1.5$) drawn from a maximum (28%) and minimum (25%) security custody center, a provincial assessment center (45%), and probation offices (2%) in British Columbia. The ethnic composition of the sample included 66% ($n = 118$) Caucasian, 23% ($n = 41$) Aboriginal, and 11% ($n = 20$) of youth of other ethnicity. In the offender sample, an attempt was made to approach 132 youth. Of these youth, parent/legal guardians refused consent for 28 youth (21%), 5 youth refused consent (4%), and 1 youth withdrew partway through the study (< 1%). In the non-offender sample, an attempt was made to approach 102 youth. Of these youth, 19 youth refused consent (19%) and 2 withdrew partway through the study (2%).

Attempts were made to enroll every new female admission to the custody and assessment centers who was then matched with a same aged male youth. Exclusionary criteria for this sample comprised (a) an IQ below 70, or (b) any significant Axis I psychotic symptomatology. Youth agreeing to participate in this portion of the research

study completed individual assessments comprised of semi-structured clinical interviews, computerized diagnostic assessments, and a battery of self-report measures. They were compensated either \$30 (residential and outpatient youth) or were provided with snacks during testing and \$10 upon completion of the protocol (incarcerated youth). All sessions were digitally recorded, and informed consent was obtained from both the youth and his or her legal guardian before beginning the testing sessions. Ethics approval was obtained from the university and institutional review boards prior to the start of the study.

Follow-up data collection was conducted via phone interview at least 22 months from the youth's Time 1 participation ($M = 26.0$, $SD = 3.7$). At this time, youth agreeing to participate were administered a portion of the self-report questionnaires that were administered at Time 1 (including the same self-report measure of aggression and offending, see the Form-Function Aggression Measure and the Self-Report of Offending below), in addition to supplementary questions regarding their mental and physical health. At the time of the current study, 82 youth (36 males, 46 females) had completed a follow-up phone interview (62% of youth from the original sample who were eligible for the follow-up interview; Time 2 data collection is still ongoing). Supplemental analyses using data from Time 1 did not reveal any systematic differences on demographic variables (i.e., age, gender, ethnicity), nor on the variables of interest (i.e., affect dysregulation, deficient affect, aggression, and antisocial behavior) between those youth who had versus had not completed a follow-up phone interview at Time 2.

Measures

Affect Regulation Checklist (ARC; Moretti, 2003). This 12-item self-report scale was developed to measure three components of affect regulation: Dyscontrol (e.g., "I

have a hard time controlling my feelings”; “My feelings just take over me and I can’t do anything about it”), Suppression (e.g., “I try hard not to think about my feelings”; “I try to do other things to keep my mind off of how I feel”), and Reflection (e.g., “Thinking about why I have different feelings helps me to learn about myself”). All items are scored on a 3-point scale ranging from “*not like me*” to “*a lot like me*”. A subset of items was adapted from published scales of affect regulation (Gross & John, 1998, 2003; Shields & Cicchetti, 1998) and others were developed to tap the three factors of affect regulation in adolescents outlined above. Consistent with other studies in the area, the ARC represents a multidimensional view of affect regulation that includes both maladaptive (lack of control, suppression) and adaptive (reflection) attempts at regulating affect. Furthermore, the ARC is careful to inquire about regulatory abilities that are not tied to any specific emotion so as not to confound the emotion with regulatory processes (Cole et al., 2004). The present study focused on the first two subscales of the ARC (i.e., dyscontrol and suppression), as they most clearly represent regulation strategies that are maladaptive in nature, and are also most consistent with the definitions of affect dysregulation provided in other studies (e.g., Gross & John, 1998; Shields & Cicchetti, 1998).

Results from confirmatory factor analyses supported a 3-factor solution for the ARC, CFI = .93, RMSEA = .06, over both a 1-factor (CFI = .45, RMSEA = .18) and 2-factor (CFI = .83, RMSEA = .10) solution. With respect to convergent and divergent validity, only the dyscontrol factor was positively and uniquely associated with indicators of both Oppositional Defiant Disorder ($\beta = .52, p < .01$) and Conduct Disorder ($\beta = .21, p < .05$), whereas both the dyscontrol and suppression factors were positively related to Separation Anxiety Disorder ($\beta = .20$ and $.23, p < .05$ for dyscontrol and suppression,

respectively). Additionally, both the dyscontrol and reflection factors were positively and uniquely associated with indicators of depression ($\beta = .21$ and $.22, p < .05$ for dyscontrol and reflection, respectively). Means and standard deviations for each factor are presented in Table 1.

Psychopathy Checklist: Youth Version (PCL:YV; Forth et al., 2003). The PCL:YV is a 20-item symptom construct rating scale designed to measure the same interpersonal, affective, and behavioral dispositions as does its parent measure, the PCL-R, in youth. Each item is scored on a 3-point scale, with scores of zero (consistently absent), one (inconsistent), or two (consistently present) for each component reflecting inferences about the presence and severity of a specific trait across situations. Items are summed to yield a total score ranging from 0 to 40, with higher scores reflecting the increased presence of psychopathic features. The scoring guidelines for the PCL:YV have been modified to reflect the different expressions of psychopathic characteristics in adolescents of varying ages (Kosson, Cyterski, Steuerwald, Neumann, & Walker-Matthews, 2002), and require the examiner to compare a youth's behavior to other youth of the same chronological age.

Items on the PCL:YV are purported to retain the same 2-factor structure as the PCL-R (i.e., with Factor 1 representing the interpersonal and affective features of psychopathy, and Factor 2 encompassing the antisocial behaviors), although several confirmatory factor analyses have indicated that this model does not provide an adequate fit to youth data (Kosson et al., 2002; Odgers, Reppucci, & Moretti, 2005). More recently, results from confirmatory factor analyses published in the PCL:YV manual (Forth et al., 2003) identified both the 3-factor (Cooke & Michie, 2001) and 2-factor/4-

facet (Hare, 2003) models of psychopathy as acceptable test structures in youth samples. In light of these findings, analysis for the current study was guided by the hierarchical 3-factor model of psychopathy (Cooke & Michie, 2001). This model posits a superordinate factor, Psychopathy, with three separate subfactors: Arrogant and Deceitful Interpersonal Style (ADI), Deficient Affective Experience (DAE), and Impulsive and Irresponsible Behavioral Style (IIB). More recently, a fourth factor (Antisocial) was proposed by Hare (2003) which groups together the omitted criminality items from Cooke and Michie's model. The current study focused on Factor 2 of the PCL:YV (DAE) as a measure of deficient affect. This factor is comprised of four items: Lacks Remorse, Shallow Affect, Callousness/Lacks Empathy, and Failure to Accept Responsibility.

Semi-structured interviews lasting approximately 60-90 minutes were conducted by three graduate students who had received formal training in the administration and coding of the PCL:YV.² The interview touched on a range of areas including the youth's educational history, work history and occupational goals, suicidal ideation, family and peer relationships, aggression and criminal activity, affect, and mood. Collateral sources of information, including developmental and social histories, pre-sentencing and disposition reports, and psychological assessments were coded as well. Using single-rater intraclass correlation coefficients (ICC_1) for a two-way random effects model for absolute groups (McGraw & Wong, 1996), interrater reliability was satisfactory for PCL:YV total score based on file-only training cases (.87; $n = 5$). For interview cases ($n = 28$), the ICC_1 for PCL:YV total score was .96. For the factor scores, the coefficients

²All raters underwent a PCL:YV training session with an expert in adolescent psychopathy who had experience administering the measure to offenders. The training involved a one-day workshop including an overview of psychopathic traits in adolescents, a description of the PCL:YV items, and guidelines on scoring the items. Prior to the start of data collection, between five and eight training assessments were conducted and a minimum interrater reliability of .85 for the Total score was attained.

ranged as follows: Factor 1 = .93, Factor 2 = .90, and Factor 3 = .84. The mean and standard deviation for the DAE factor is presented in Table 1.

Form-Function Aggression Measure (FFAM; Little, Jones, Heinrich, & Hawley, 2003). The Form-Function Aggression Measure is a 36-item self-report measure designed to separate and assess the forms (i.e., overt, relational) and functions (i.e., instrumental, reactive) of aggression. Items on the FFAM are based directly on other published measures of aggression (Crick, 1997; Crick & Grotpeter, 1995; Dodge & Coie, 1987), and include items which assess “pure” subtypes of aggression (e.g., overt-reactive aggression). In the current study, a modified 25-item version of the measure was used, reflecting those items that demonstrated the highest reliabilities in supplemental analyses performed by Little (T. Little, personal communication, April 2003). All items are scored on a 4-point scale ranging from “*not true at all*” to “*completely true*”. Little and colleagues (2003) reported acceptable levels of internal validity (r_{xx} ranging from .62 for pure relational aggression to .84 for overt instrumental aggression), as well as satisfactory external and criterion validity for the scale, which was shown to generalize across age, gender, and ethnicity. A separate study investigating the psychometric properties of the FFAM in the current sample (Lee, Penney, Odgers, & Moretti, 2007), supported the use of a 6-factor model representing the following six subtypes of aggression: pure overt, reactive overt, instrumental overt, pure relational, reactive relational, instrumental relational. The current study focused on reactive and instrumental forms of overt aggression (e.g., “When I am hurt by someone, I often fight back”; “I often threaten others to get what I want”). Means and standard deviations for each subtype at the two time points are presented in Table 1.

Self-Report of Offending, Revised (SRO-R). The Self-Report of Offending (Huizinga, Esbensen, & Weiher, 1991) was adapted for use in this study based on the more widely studied Self-Report of Delinquency (see Huizinga & Elliot, 1986; Piquero, MacIntosh, & Hickman, 2002). This scale has been shown to produce results consistent with official measures of delinquency (e.g., similarities on demographic features and offense type categories between self-reported offenders and officially identified offenders; Elliott, Dunford, & Huizinga, 1987). However, as is the case with most other self-report offense measures, the SRD tends to identify significantly more “career offenders” as compared to official arrest data. The SRD has also demonstrated functional invariance across gender and ethnicity (Knight, Little, Losoya & Mulvey, 2004). The current measure included 15 items, largely comparable to those found in large-scale high-risk and normative studies, assessing the youth’s lifetime (Time 1) and current (past 24 month; Time 2) involvement in violent (e.g., assault and weapons charges) and non-violent (e.g., narcotics and property crimes) offenses. Means and standard deviations for the number of different violent and non-violent offenses reported at both time points are presented in Table 1.

Table 1

Means and Standard Deviations of Measures Employed at Time 1 and Time 2

Variable	Time 1		Time 2	
	M	SD	M	SD
Dyscontrol (ARC)	3.84	2.4	--	--
Suppression (ARC)	2.87	1.9	--	--
Deficient Affect (PCL:YV)	4.17	1.9	--	--
Reactive Aggression (FFAM)	9.77	3.5	9.00	3.4
Instrumental Aggression (FFAM)	7.93	3.7	6.26	2.2
Violent Offenses (SRO-R)	2.12	2.0	1.30 [†]	1.9
Non-violent Offenses (SRO-R)	2.42	1.9	1.80 [†]	1.7

Note. ARC = Affect Regulation Checklist (Moretti, 2003); PCL:YV = Psychopathy Checklist: Youth Version (Forth et al., 2003); FFAM = Form-Function Aggression Measure (Little et al., 2003); SRO-R = Self Report of Offending, Revised (Huizinga et al., 1991). Minimum and maximum scores for each of the scales are as follows: 0 to 5 (Non-violent Offenses), 0 to 6 (Violent Offenses), 0 to 8 (Dyscontrol, Deficient Affect, Reactive Aggression), and 0 to 10 (Suppression, Instrumental Aggression).

[†] So as not to overlap with the SRO-R data collected at Time 1, these figures represents the number of different violent and non-violent offenses engaged in over the last two years only.

Analytic Strategy: Structural Equation Modeling

The current study employed a structural equation modeling (SEM) framework in order to evaluate the direct and interaction effects of affect dysregulation and deficient affect in predicting aggression, violence, and non-violent delinquency. SEM provides a confirmatory approach to data analysis in which multiple sets of regression equations can be tested simultaneously, thus allowing the researcher to test complex models involving a potentially large number of linear relationships (Tomarken & Waller, 2005).

Additionally, the relationships between latent variables and their manifest indicators (the measurement model) may be separately estimated from the hypothesized relationships among latent constructs (the structural model). Consequently, the associations among constructs are corrected for biases stemming from construct-irrelevant variance and measurement error associated with the observed variables (Bollen, 1989; Tomarken & Waller, 2005). Thus, SEM provides a test of construct-level hypotheses at the appropriate (i.e., construct) level rather than at the level of a measured variable (Ullman, 2006).

Once a model has been specified, a critical issue in SEM is that of model identification. A model meets criteria for identification only when there is a unique numerical solution for each parameter in the model (Ullman & Bentler, 2003). For this criterion to be realized, it is necessary that the number of data points in a model (i.e., the number of non-redundant sample variances and covariances) exceed the number of estimated parameters (i.e., the number of regression coefficients, variances and covariances to be estimated). When a model meets this condition, it is said to be overidentified. This may be contrasted with just identified or underidentified models in which there are equal or fewer data points than estimated parameters, and for which the

model's adequacy cannot be meaningfully tested. Another requirement for model identifiability is to establish the scale of each latent factor. This is most commonly done by fixing the regression coefficient from the factor to one of the observed indicator variables to 1. Lastly, it is advised that each latent factor have at least three or more pure indicators (i.e., indicators which load only on one latent factor) with nonzero loadings and uncorrelated error terms to ensure model identifiability (Ullman, 2006).

All models in the current study were fit to the data using Mplus Version 3.1 (Muthén & Muthén, 2004), and analyses were performed using robust weighted least-squares estimation with a mean- and variance-adjusted chi-square algorithm (robust WLS). In contrast to maximum-likelihood (ML) estimation which assumes the observed variables are continuous and normally distributed, WLS estimation is more appropriate when the data are binary or discrete (e.g., Likert items) due to its usage of polychoric correlations (Muthén, du Toit, & Spisic, 1997). Furthermore, robust WLS has been shown to perform well under conditions of minor to moderate nonnormality (Flora & Curran, 2004), and also avoids many of the pitfalls associated with full WLS estimation, particularly the requirement of extremely large sample sizes (> 2500) to obtain unbiased test statistics and parameter estimates. Under the robust WLS estimation method in Mplus, missing data are handled using a pairwise present method.³

³ This method computes polychoric correlations which are based on pairwise present data for the two variables at hand, and assumes that the data is missing completely at random (MCAR; Rubin, 1976) for each variable pair. While it is not possible to perform a direct test of MCAR as the missing values for each variable are unknown, the data were examined for evidence of systematic missingness. This was done by creating a missing data dummy variable for each exogenous variable in the subsequent models that was then entered as a dependent variable in a logistic regression model which included the remaining variables in the data set as independent variables. None of the variables were significant predictors of the presence/absence of missing data on any of the dependent (dummy) variables, thus supporting the less stringent assumption that the data are missing at random (MAR; Rubin, 1976).

Models were evaluated according to suggested critical values for commonly used fit indices (i.e., Comparative Fit Index, CFI > .95, Tucker-Lewis Index, TLI > .95, Root Mean Square Error of Approximation, RMSEA < .06; Hu & Bentler, 1999). Both the CFI and TLI are comparative fit indices which assess the absolute fit of the specified model compared to the absolute fit of the independence model (i.e., a model containing completely unrelated variables). The greater the discrepancy between the overall fit of the two models, the larger the values of these descriptive statistics (with 0 indicating an equivalent fit to the independence model, and 1 indicating perfect fit to the specified model). In contrast, the RMSEA is a residual-based fit index which estimates the lack of fit in a model compared to a “perfect” model (i.e., a fully saturated model with zero degrees of freedom). When categorical variables are used, another important indicator of model adequacy is the weighted root mean square residual (WRMR), which measures the weighted average differences between the sample and estimated population variances and covariances, and for which values < .90 are recommended (Yu and Muthén, 2002). Lastly, in accordance with Bentler’s (1988) suggestion that a minimum ratio of five subjects to each estimated parameter be present, all models contained fewer than 35 estimated parameters ($N = 179$).

With respect to the sequence of analysis, direct effects of affect dysregulation and deficient affect were first modeled for each of the endogenous variables (i.e., reactive and instrumental aggression, violent and non-violent offending) which were measured at two separate time points. Next, moderation effects were tested to investigate whether the constructs of affect dysregulation and deficient affect interact to create a heightened (or lowered) risk for aggression. Moderation analyses were performed on the Time 1 data via

multiple-group modeling, which allows for a direct and empirical comparison of model parameters across different groups (e.g., groups high versus low on deficient affect). Nested chi-square difference tests were conducted to examine loss of fit when moving from a “free” model (where parameters are allowed to vary across groups) to a constrained model requiring parameters of interest to be equal across groups. If a significant increase in χ^2 units per degrees of freedom is obtained upon constraining the given parameter(s), this suggests that the constrained model is not equivalent to its unconstrained counterpart and supports the presence of moderation.⁴

⁴ The WLSMV estimator in Mplus adjusts both the chi-square and degrees of freedom to obtain accurate *p*-values when using categorical variables. Consequently, these values cannot be used in the traditional way to conduct chi-square difference tests. Instead, the difference in model fit for nested models is based on the derivatives difference test and does not correspond directly with the differences in estimated chi-square and degrees of freedom between the constrained and unconstrained models. Additionally, because degrees of freedom are mean- and variance- adjusted, they do not correspond in a straightforward way with the numbers of measured variables and estimated parameters.

RESULTS

Concurrent Relations of Affect Dysregulation and Deficient Affect to Aggressive and Antisocial Behaviors

Figure 1 illustrates the general structural model that was fit to the data. The joint (independent) effects of affect dysregulation and deficient affect on outcome were tested by regressing each of the endogenous variables (i.e., reactive and instrumental overt aggression, violent and non-violent offending) onto the dysregulation and deficit constructs. Equivalent models were created to assess the two forms of affect regulation included in the study (dyscontrol and suppression) for each of the four endogenous variables (reactive and instrumental overt aggression, violent and non-violent offending), so that a total of eight models were evaluated. In addition, models for the dependent variables gathered at Time 2 (i.e., reactive and instrumental overt aggression, self-reported violent and non-violent offenses) were explored and compared to the models investigating the concurrent relationships between affect dysregulation, deficient affect, and outcome. In all cases the two exogenous latent variables (i.e., affect dysregulation and deficient affect) were allowed to covary, and all latent variables were standardized by constraining one factor loading per latent variable equal to one. The exogenous and endogenous variable reliabilities and intercorrelations are shown in Table 2. All models which are not included as figures in the text below are represented in the Appendix.

Figure 1. General structural model.

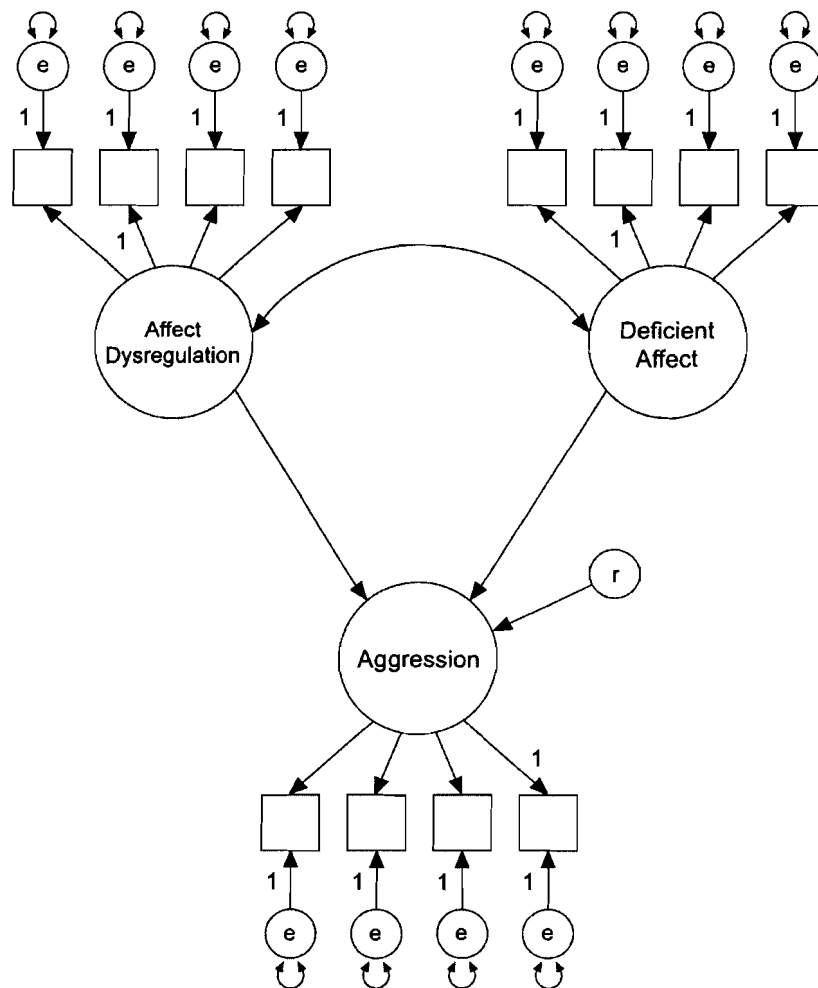


Table 2

Reliabilities and Intercorrelations among Exogenous and Endogenous Variables

Variable (Time 1)	1	2	3	4	5	6	7	8	9	10	11	α
1. Dyscontrol	--											.81
2. Suppression	.11	--										.65
3. Deficient Affect	.05	-.10	--									.65
4. Reactive Aggression	.35**	-.02	.42**	--								.85
5. Instrumental Aggression	.31**	-.02	.38**	.59**	--							.91
6. Violent Offenses	.08	.09	.37**	.48**	.48**	--						.78
7. Non-violent Offenses	.07	.04	.29*	.48**	.45**	.63**	--					.83
8. Reactive Aggression (Time 2)	-.02	.10	.38**	.56**	.35*	.49**	.35*	--				.84
9. Instrumental Aggression (Time 2)	.12	.17	.36*	.40**	.40**	.32*	.14	.54**	--			.87
10. Violent Offenses (Time 2)	-.02	.03	.32*	.37**	.31*	.54**	.32*	.62**	.37**	--		.85
11. Non-violent Offenses (Time 2)	-.12	-.03	.30*	.19	.29	.47**	.46**	.50**	.32*	.66**	--	.77

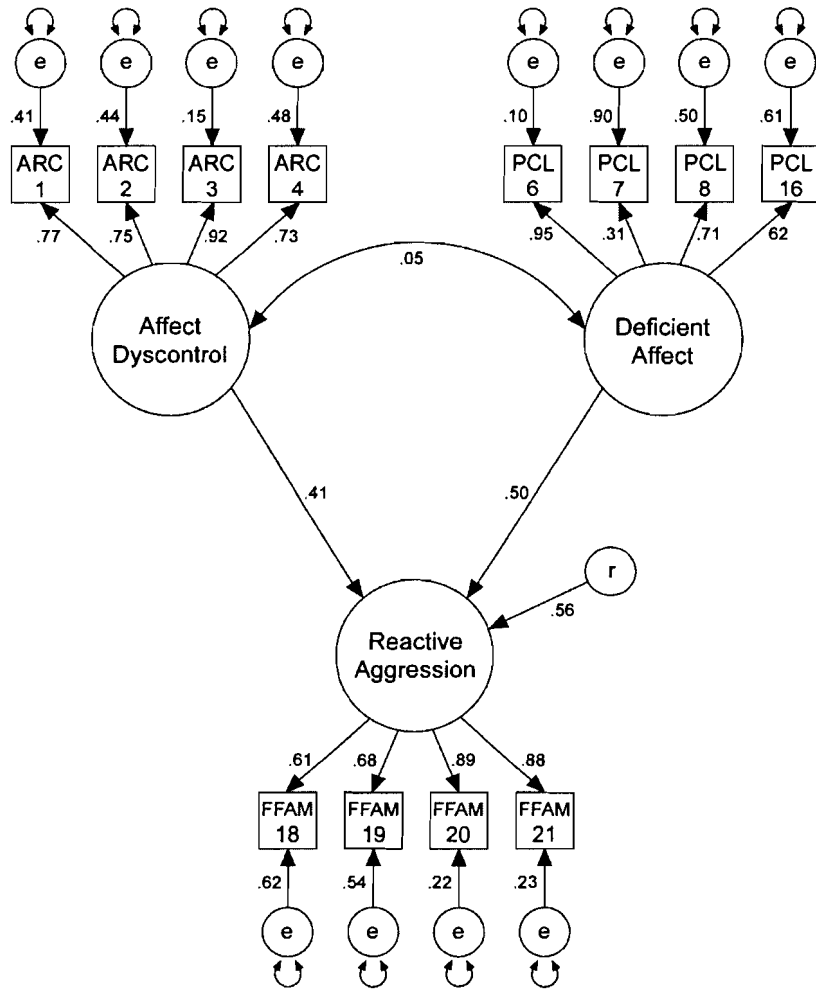
* $p < .01$ ** $p < .001$.

Affect dyscontrol. Using the dyscontrol factor of the ARC, the SEM for both types of aggression resulted in an excellent model fit, $\chi^2 (21, N = 179) = 20.10, p = .52$ (CFI = 1.0, TLI = 1.0, RMSEA = .00, WRMR = .55)⁵ for reactive aggression and $\chi^2 (20, N = 179) = 23.13, p = .28$ (CFI = .99, TLI = .99, RMSEA = .03, WRMR = .60) for instrumental aggression. Furthermore, the models accounted for 44 and 34% of the variance for reactive and instrumental aggression, respectively. Results were similar when using the number of violent and non-violent offenses a youth had engaged in as the dependent variables, $\chi^2 (37, N = 179) = 49.36, p = .08$ (CFI = .98, TLI = .98, RMSEA = .04, WRMR = .80) for violent offenses and $\chi^2 (28, N = 179) = 43.63, p = .03$ (CFI = .98, TLI = .98, RMSEA = .06, WRMR = .80) for non-violent offenses. These models accounted for 37 and 32% of the variance for violent and non-violent offenses, respectively. With respect to the measurement portion of the models, each of the indicator variables demonstrated significant loadings onto their respective latent constructs as evaluated by a z test (i.e., the value of each parameter estimate divided by its estimated standard error was > 1.96). The only exception to this was seen for PCL:YV item 7 (Shallow Affect); the standardized parameter estimate (lambda coefficient) for this item was non-significant in the models predicting violence ($\lambda = .14, p = .22$) and delinquency ($\lambda = .10, p = .36$), suggesting that this item is a less reliable indicator of the deficient affect construct. In terms of the structural component of the models, both the dyscontrol and deficient affect constructs evidenced significant associations with reactive

⁵ CFI and TLI values of 1.0 do not indicate a “perfect fit”. Rather, they indicate that there was no suggestion of misspecification of the hypothesized model relative to a baseline model (CFI) and that the hypothesized model provided a significant improvement in fit compared to the independence model (TLI). Similarly, the RMSEA value of .00 is a matter of small residuals (substantiated by the low WRMR value) rather than indicating a perfect fit.

($\beta = .41$ and $.50$, $p < .01$ for dyscontrol and deficit, respectively) and instrumental ($\beta = .34$ and $.46$, $p < .01$ for dyscontrol and deficit, respectively) aggression, while only the deficient affect factor showed a significant relationship to violence ($\beta = .60$, $p < .01$) and delinquency ($\beta = .56$, $p < .01$). In all models, the bivariate association between the dyscontrol and deficit factors was minimal and non-significant ($r < .05$). The model utilizing reactive aggression as the dependent variable is graphically displayed in Figure 2.

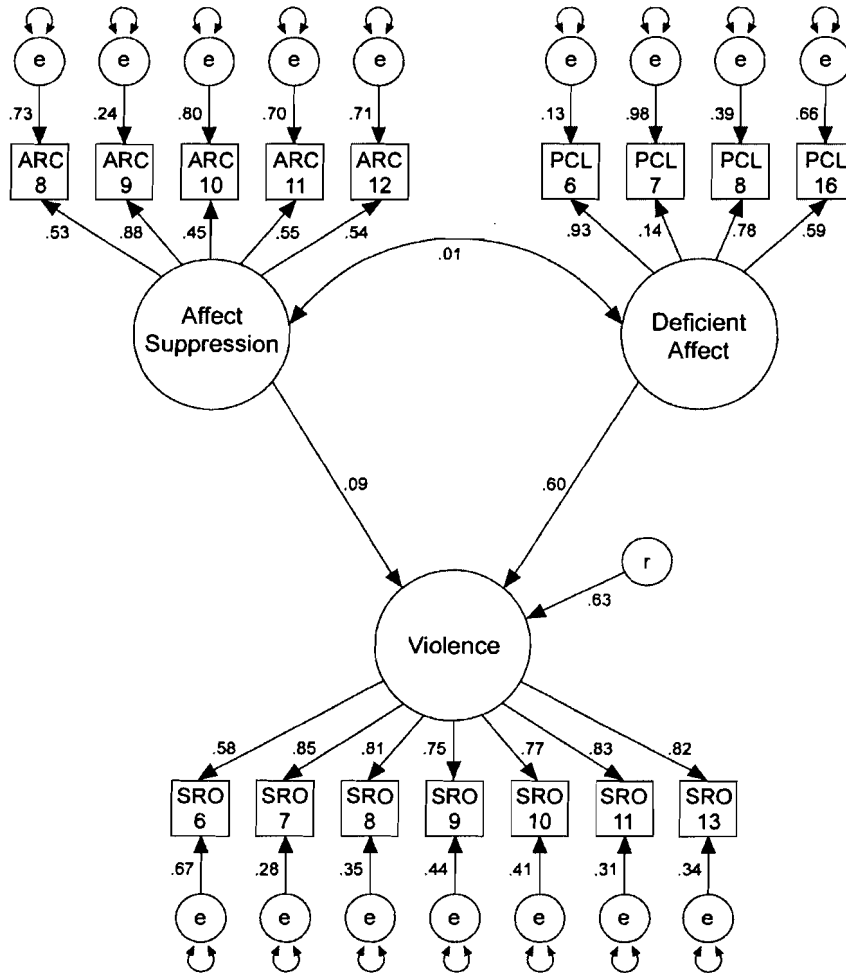
Figure 2. Joint effects of affect dyscontrol and deficient affect on concurrent reactive aggression.



Note. CFI = 1.0, TLI = 1.0, RMSEA = .00, WRMR = .55

Affect suppression. Moving to affect suppression, the SEM for both types of aggression resulted in a slightly less than acceptable model fit, $\chi^2(26, N = 179) = 40.94, p = .03$ (CFI = .93, TLI = .93, RMSEA = .06, WRMR = .80) for reactive aggression and $\chi^2(23, N = 179) = 44.51, p = .01$ (CFI = .88, TLI = .91, RMSEA = .07, WRMR = .87) for instrumental aggression. The models accounted for 28 and 24% of the variance for reactive and instrumental aggression, respectively. Results were improved when using the number of violent and non-violent offenses a youth had engaged in as the dependent variables, $\chi^2(42, N = 179) = 55.11, p = .09$ (CFI = .97, TLI = .98, RMSEA = .04, WRMR = .83) for violent offenses and $\chi^2(33, N = 179) = 60.94, p = .01$ (CFI = .95, TLI = .95, RMSEA = .07, WRMR = .92) for non-violent offenses. These models accounted for 37 and 36% of the variance for violent and non-violent offenses, respectively. The measurement portion of these models revealed that all of the indicator variables demonstrated significant loadings onto their respective latent constructs, with PCL:YV item 7 again demonstrating non-significant loadings in the models predicting violence ($\lambda = .14, p = .23$) and delinquency ($\lambda = .10, p = .37$). Looking to the structural component of the models, only the deficient affect variable evidenced significant associations with reactive ($\beta = .53, p < .01$) and instrumental ($\beta = .48, p < .01$) aggression, as well as with violence ($\beta = .60, p < .01$) and delinquency ($\beta = .56, p < .01$). Affect suppression as measured by the ARC was not significantly related to any dependent variable. Similar to the results reported for the dyscontrol factor, the bivariate association between the suppression and deficit factors was minimal and non-significant ($r < .02$). The model utilizing violence as the dependent variable is graphically displayed in Figure 3.

Figure 3. Joint effects of affect suppression and deficient affect on concurrent self-reported violence.



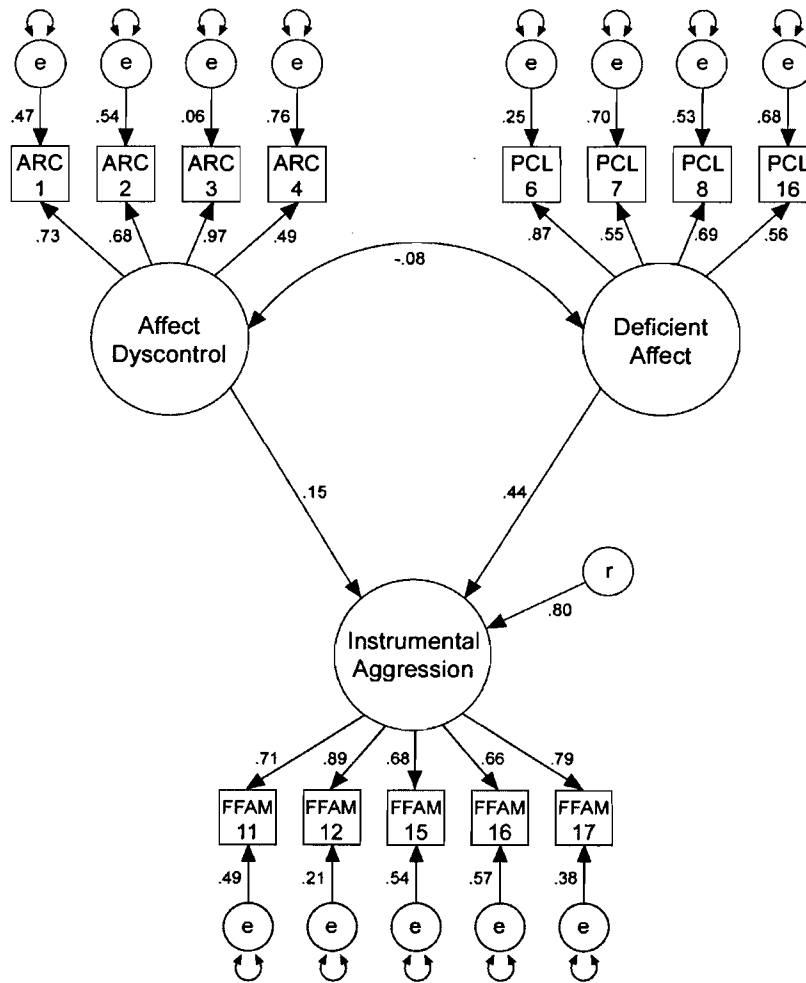
Note. CFI = .97, TLI = .98, RMSEA = .04, WRMR = .83

Prospective Relations of Affect Dysregulation and Deficient Affect to Aggressive and Antisocial Behaviors

Affect dyscontrol. The models employing prospective measures of aggression and offending (i.e., the FFAM and SRO-R administered at Time 2) revealed a similar pattern of results compared to Time 1 with one exception: affect dyscontrol was no longer a significant predictor of reactive or instrumental forms of aggression. Thus, across each of the endogenous variables tested (reactive and instrumental aggression, violent and non-violent offending), only deficient affect emerged as a significant predictor of these variables ($\beta = .51, .44, .62, .47, p < .01$ for reactive and instrumental aggression, violent and non-violent offending, respectively). The overall fit of the model for reactive aggression was excellent, $\chi^2 (18, N = 82) = 16.86, p = .53$ (CFI = 1.0, TLI = 1.0, RMSEA = .00, WRMR = .58), while the model for instrumental aggression fell just below an optimal fit, $\chi^2 (18, N = 82) = 30.10, p = .04$ (CFI = .91, TLI = .92, RMSEA = .09, WRMR = .83). Of note, these models accounted for a significant, albeit smaller proportion of variance compared to the Time 1 models (26 and 21% for reactive and instrumental aggression, respectively). The models utilizing the SRO-R evidenced satisfactory levels of fit, $\chi^2 (21, N = 82) = 22.13, p = .39$ (CFI = 1.0, TLI = .99, RMSEA = .03, WRMR = .72) for violent offenses and $\chi^2 (24, N = 82) = 26.06, p = .35$ (CFI = .99, TLI = .99, RMSEA = .03, WRMR = .69) for non-violent offenses. These models accounted for a similar amount of variance as compared to their Time 1 counterparts (38 and 26% for violent and non-violent offenses, respectively). Each of the indicator variables again demonstrated significant loadings onto their respective latent constructs (with the exception of PCL:YV item 7 for the SRO-R models only), suggesting that the

measurement portion of the models are reliable. Analogous to the results obtained at Time 1, the bivariate association between the dyscontrol and deficit factors was minimal and non-significant ($r = -.06$ to $-.08$). The model utilizing instrumental aggression as the dependent variable is graphically displayed in Figure 4.

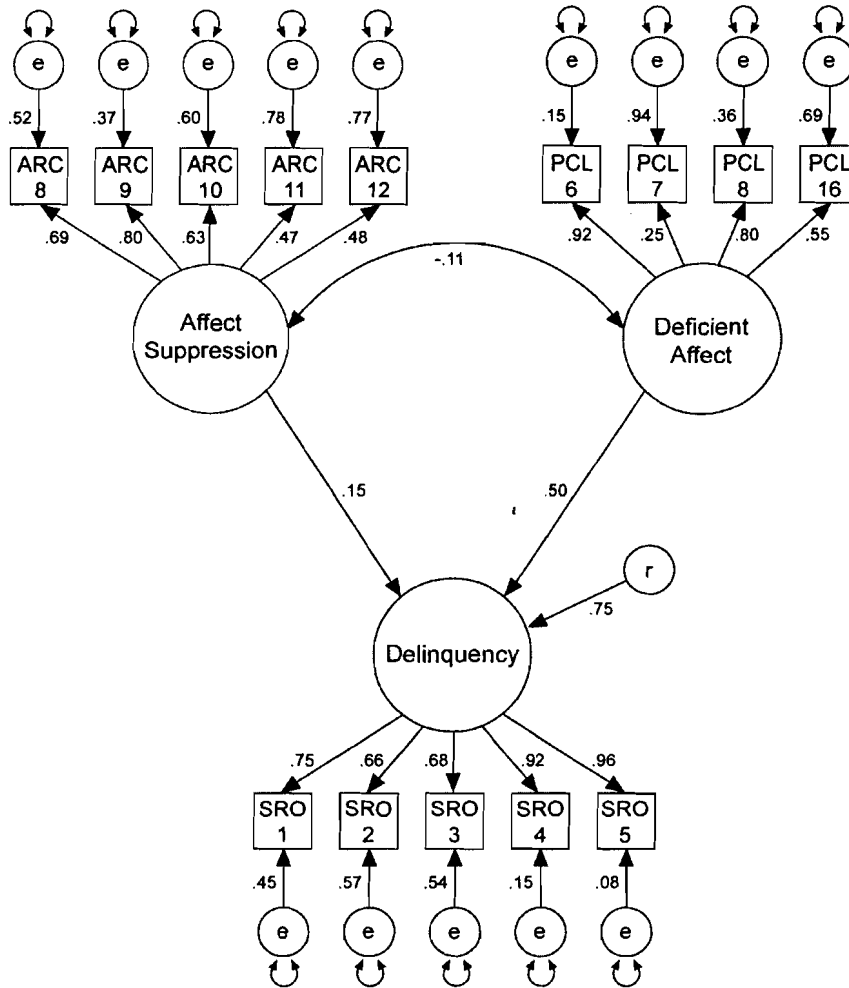
Figure 4. Joint effects of affect dyscontrol and deficient affect on prospective instrumental aggression.



Note. CFI = .91, TLI = .92, RMSEA = .09, WRMR = .83

Affect suppression. The prospective models employing the suppression factor were entirely similar to the results gathered at Time 1: in all cases only deficient affect was a significant predictor of outcome ($\beta = .49, .43, .60, .50, p < .01$ for reactive and instrumental aggression, violent and non-violent offending, respectively), while affect suppression showed minimal associations to each of these variables. The overall fit of the model for reactive aggression was excellent, $\chi^2 (22, N = 82) = 21.23, p = .51$ (CFI = 1.0, TLI = 1.0, RMSEA = .00, WRMR = .61), while the corresponding model for instrumental aggression fell slightly below an optimal fit, $\chi^2 (18, N = 82) = 26.33, p = .09$ (CFI = .92, TLI = .92, RMSEA = .08, WRMR = .81). Accordingly, the model for reactive aggression accounted for a higher proportion of variance (29%) as compared to the model for instrumental aggression (18%). The models utilizing the SRO-R evidenced satisfactory levels of fit, $\chi^2 (26, N = 82) = 25.82, p = .47$ (CFI = 1.0, TLI = 1.0, RMSEA = .01, WRMR = .70) for violent offenses and $\chi^2 (27, N = 82) = 28.87, p = .37$ (CFI = .99, TLI = .99, RMSEA = .03, WRMR = .72) for non-violent offenses. Each of these models accounted for a comparable amount of variance as compared to their Time 1 counterparts (40 and 25% for violent and non-violent offenses, respectively). All indicator variables demonstrated significant loadings onto their respective latent constructs (with the exception of PCL:YV item 7 for the SRO-R models only), suggesting that the measurement portion of the models are consistent. In each case the bivariate association between the dyscontrol and deficit factors was again minimal and non-significant ($r = -.11$ to $-.12$). The model utilizing non-violent delinquency as the dependent variable is graphically displayed in Figure 5.

Figure 5. Joint effects of affect suppression and deficient affect on prospective self-reported non-violent delinquency.



Note. CFI = .99, TLI = .99, RMSEA = .03, WRMR = .72

Thus far, results suggest that the deficient affect factor of the PCL:YV is a significant predictor of both aggression and delinquency variables. However, in light of the fact that the factors comprising the PCL:YV are themselves intercorrelated (i.e., Interpersonal, Affective, Behavioral, and Antisocial), additional regression analyses were conducted to estimate the unique variance attributable to the deficient affect factor. As represented in Table 3, each of the four PCL:YV factors were simultaneously entered into a regression with Time 1 reactive and instrumental aggression, violent and non-violent offenses as the dependent variables. Results from these analyses indicated that Factors 1 (Interpersonal), 2 (Affective), and 4 (Antisocial) each uniquely accounted for a significant proportion of variance in predicting reactive aggression, whereas only Factors 2 and 4 emerged as significant predictors of instrumental aggression. With respect to the SRO-R, only the Antisocial factor showed a unique association with violent offenses, whereas both the Behavioral (Factor 3) and Antisocial factors predicted non-violent offending. At follow-up, only the Antisocial factor demonstrated unique variance in predicting reactive aggression, as well as non-violent offenses. In contrast, only the Affective factor predicted instrumental aggression, while only the Interpersonal factor predicted violent offending.

Table 3

Stepwise Regression with PCL:YV Factor Scores Predicting Aggression and Antisocial Behavior

Variable	<i>B</i>	<i>SE B</i>	β
Reactive Aggression			
Interpersonal	-.37	.17	-.18*
Affective	.57	.18	.31**
Behavioral	.17	.17	.10
Antisocial	.33	.11	.28**
Instrumental Aggression			
Interpersonal	-.06	.20	-.03
Affective	.55	.21	.26**
Behavioral	-.04	.20	-.02
Antisocial	.37	.13	.28**
Violent Offenses			
Interpersonal	.03	.10	.03
Affective	.06	.10	.05
Behavioral	.18	.10	.17
Antisocial	.30	.06	.43**
Non-violent Offenses			
Interpersonal	-.07	.08	-.06
Affective	-.14	.08	-.13
Behavioral	.20	.08	.20*
Antisocial	.46	.05	.86**

Note. $R^2 = .28, p < .01$ (Reactive aggression); $R^2 = .20, p < .01$ (Instrumental aggression); $R^2 = .34, p < .01$ (Violent offenses); $R^2 = .54, p < .01$ (Non-violent offenses).

* $p < .05$. ** $p < .01$.

Moderation Models: Are the Effects of Affect Dysregulation Comparable Across Different Levels of Deficient Affect?

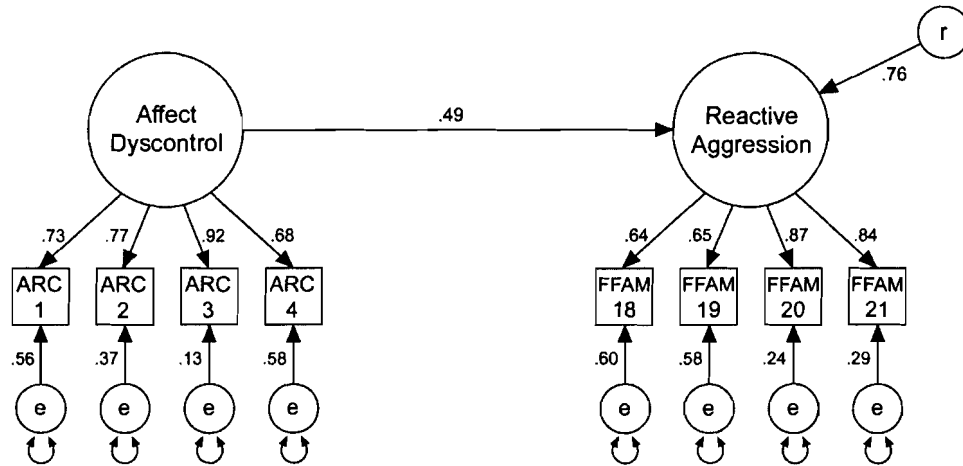
Thus far, the analyses presented have examined the independent effects of affect dysregulation and deficient affect; however, it is informative to assess if and how these constructs interact. Although no significant bivariate relationships were observed between affect dysregulation and deficient affect in the previous models, interaction effects may still exist and were therefore systematically explored via moderation analyses. For each of the eight models evaluated (two dysregulation variables, four dependent variables), two groups were created by separating youth who fell above or below the median on Factor 2 of the PCL:YV ($n = 59$ for the high group; $n = 85$ for the low group). In order to test for the presence of moderation, nested models were created which either estimated or constrained the relationship from affect dysregulation to outcome to be equal across groups high and low on deficient affect. If a significant *loss* of fit is found between nested models, this suggests that the constrained model is not equivalent to its unconstrained counterpart and supports the presence of moderation. In other words, a significant finding would suggest that the relationship between affect dysregulation and outcome changes across groups manifesting varying levels of deficient affect.

Results from these analyses did not reveal any evidence of moderation: in all cases the fit of the free and constrained models were statistically equivalent, suggesting that the relationship between affect dysregulation (both the dyscontrol and suppression factors) and outcome is comparable across youth with high and low levels of deficient

affect (see Figure 6 for an example). The χ^2 values and fit statistics for the free and constrained versions of each model are presented in Table 4.

Figure 6. Relationship between affect dyscontrol and reactive aggression at high and low levels of deficient affect.

Group HI - Deficient Affect



Group LO - Deficient Affect

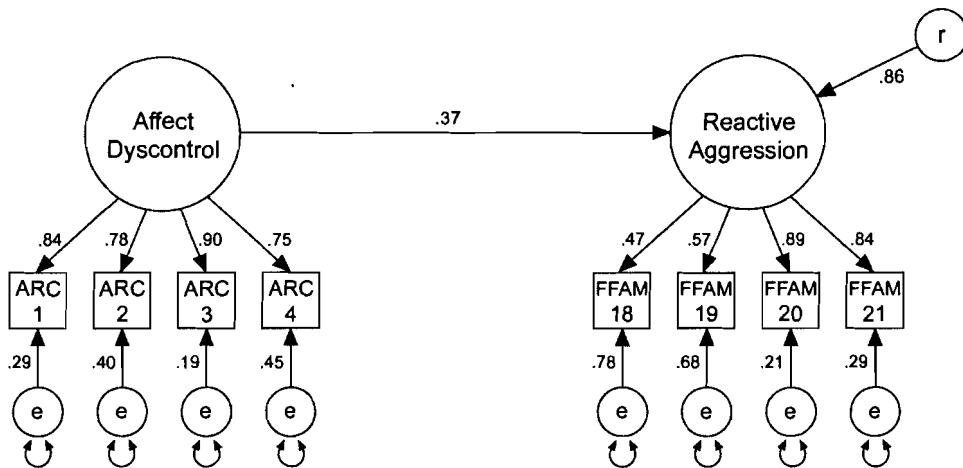


Table 4

Chi-square Difference Tests for Moderation Models

Variables	Model	χ^2	df	CFI	TLI	RMSEA	$\Delta \chi^2$	Δdf	
Deficient Affect, Dyscontrol & Reactive Aggression	Constrained parameter	14.17	17	1.0	1.0	.00	0.78 (p = .38)	1	
	Free parameter	17.75	21	1.0	1.0	.00			
Deficient Affect, Dyscontrol & Instrumental Aggression	Constrained parameter	11.18	17	1.0	1.0	.00	0.15 (p = .70)	1	
	Free parameter	14.01	19	1.0	1.0	.00			
Deficient Affect, Dyscontrol & Violent Offenses	Constrained parameter	32.73	34	1.0	1.0	.00	1.68 (p = .20)	1	
	Free parameter	33.70	37	1.0	1.0	.00			
Deficient Affect, Dyscontrol & Non-violent Offenses	Constrained parameter	NO CONVERGENCE						n/a	n/a
	Free parameter	31.02	28	.99	1.0	.04			
Deficient Affect, Suppression & Reactive Aggression	Constrained parameter	33.05	24	.90	.90	.07	0.54 (p = .46)	1	
	Free parameter	39.73	27	.86	.88	.08			

Variables	Model	χ^2	df	CFI	TLI	RMSEA	$\Delta \chi^2$	Δdf
Deficient Affect, Suppression & Instrumental Aggression	Constrained parameter	31.37	23	.92	.93	.07	0.17 (p = .68)	1
	Free parameter	35.62	24	.89	.91	.08		
Deficient Affect, Suppression & Violent Offenses	Constrained parameter	50.75	44	.97	.97	.05	2.46 (p = .12)	1
	Free parameter	49.96	45	.98	.98	.04		
Deficient Affect, Suppression & Non-violent Offenses	Constrained parameter	46.85	33	.97	.97	.08	2.25 (p = .13)	1
	Free parameter	46.39	33	.97	.97	.08		

DISCUSSION

The goal of the current study was to investigate the roles of affect dysregulation and deficient affect in youth aggression and antisocial behavior. Both constructs have received consistent support in their respective associations with aggression, violence, and even non-violent delinquency; however, few researchers have attempted to reconcile the notion that both elevated and depressed levels of emotional reactivity appear to be integrally involved in the aggressive and antisocial behaviors of at-risk youth. One team of investigators proposed that affect dysregulation can encourage the development of deficiencies in affect by causing the experience of emotion to be aversive and overwhelming (Eisenberg et al., 1996, 1998). Others have conceptualized affect dysregulation and deficient affect as distinct constructs, and have proposed developmental frameworks which assume etiological and phenotypic heterogeneity among aggressive and antisocial youth (Burke et al., 2002; Frick & Morris, 2004; Loeber & Stouthamer-Loeber, 1998; Moffitt, 2006). Nevertheless, a common theme across this literature is the view that there are diverse affective experiences that give rise to problem behaviors among adolescents. However, despite that affect dysregulation and deficient affect may represent separate risk factors that contribute to the development of aggression and antisocial behavior, this idea has not received much empirical scrutiny. The present investigation begins to address this gap in knowledge by examining the joint (independent) and interaction effects of affect dysregulation and deficient affect on aggression, violence, and non-violent delinquency.

Independent Effects of Affect Dysregulation and Deficient Affect

When the effects of affect dysregulation and deficient affect were jointly modeled to predict concurrent and future indices of aggressive and antisocial behavior, results indicated that both affect dysregulation – specifically affect dyscontrol – and deficient affect were significantly associated with aggression. In contrast, the second form of affect dysregulation investigated – affect suppression – showed no associations with aggression. Furthermore, when outcomes such as violent and non-violent offending were investigated, only deficient affect emerged as a significant correlate, while both forms of affect dysregulation were unrelated to these variables. Similarly, only deficient affect was a significant predictor of *future* indices of aggression and antisocial behavior. As expected, affect dysregulation was consistently unrelated to deficient affect across all the models tested despite that each of these variables showed independent associations with aggression.

At both time points, therefore, features of deficient affect appeared to be more robust predictors of aggression, violence and delinquency. Importantly, however, when evaluated alongside the remaining PCL:YV factors, the effects of deficient affect were limited to the prediction of reactive and instrumental forms of aggression (instrumental aggression only at Time 2). Although the extant literature on psychopathy suggests that affective psychopathic traits may offer incremental value in the prediction of violent and non-violent criminality (i.e., over and above the effects of other PCL factors; Hare, 1998; Hemphill & Hare, 1995; Salekin, Rogers, & Sewell, 1996), more recent studies have questioned the overall utility of the affective dimension, particularly when compared to the behavioral component of psychopathy (Skeem & Mulvey, 2001; Walters, 2003). In

order to address this issue empirically, it is important to regularly assess the unique effects that can be attributed to the affective dimension of psychopathy, and compare them in magnitude to the effects attributable to the behavioral and antisocial dimensions. Otherwise, there is a risk of over attributing the significance of deficient affect as a risk factor for violence and delinquency when this step is neglected. In this study, once the effects of deficient affect were evaluated in this manner, they were largely comparable to the effects of affect dyscontrol.

Consistent with expectations, affect suppression was unrelated to each of the dependent variables in the current study. It is also relevant to note that several of the models employing the affect suppression factor showed less than optimal levels of overall fit. Prior studies that have included a measure of affect suppression as one form of affect dysregulation have found that it is typically associated with outcomes that are suggestive of depressive or internalizing problems more so than overt aggression. Recall that Gross and John (2003) found their measure of expressive suppression to be associated with avoidant behavior, decreased levels of positive emotions, rumination, and low self-esteem. In a younger sample, Zeman and colleagues (2001) found that their measure of sadness inhibition (including items such as “I hold my sadness in” and “I get sad inside but don’t show it”) was related to increased mood lability, impoverished emotion awareness, and symptoms of depression and anxiety. In light of this research, it is likely that the suppression factor of the ARC is associated with different types of outcomes (e.g., depression) that were not investigated in the current study. This would be an important avenue to explore in future research, particularly to substantiate the divergent validity of different affect regulation strategies by demonstrating that distinct forms of

affect dysregulation (e.g., dyscontrol versus suppression) are associated with diverse outcomes.

With respect to the dependent variables investigated in this study, research on the typology of aggressive behavior has consistently indicated that there are at least two main classes of aggression that differ primarily in terms of the underlying motivations and patterns of affective reactivity contributing to the aggressive act (Dodge & Coie, 1987; Shields & Cicchetti, 1998). Instrumental aggression is generally viewed as a more offensive and methodical type of aggression that is largely goal-directed and driven by self-serving outcomes and external reinforcement (Dodge & Coie, 1987; Little et al., 2003). In contrast, reactive aggression is most often characterized by a high degree of sympathetic arousal and angry reactivity, and is typically seen in response to a threat or provocation that then elicits a hostile response (Berkowitz, 1993; Dodge & Coie, 1987). Based on this distinction, youth who show high levels of reactive aggression should also have the most prominent difficulties with affect regulation, assuming that poor affect regulation most often results in elevated levels of negative affect and emotional reactivity. In contrast, the emergence of other forms of aggression (e.g., instrumental) appears to be less contingent on dysregulated emotions (de Castro et al., 2005; Dodge, 1991), and may even be negatively correlated with affect dysregulation to the extent that heightened emotions impair the methodical, strategic and “nonemotional” pursuit of one’s goals.

Findings from the current study, however, did not support the idea that reactive forms of aggression are uniquely associated with higher degrees of emotional dyscontrol, or that features of deficient affect are more strongly associated with instrumental

aggression: at Time 1, both affect dyscontrol and deficient affect were significantly and positively associated with both subtypes of aggression, whereas at Time 2 only deficient affect was a significant predictor of both reactive and instrumental aggression. One reason we may not see divergent relationships among the different subtypes of aggression concerns the measure of aggression used in the current study. As suggested by a recent paper on the psychometric properties of the FFAM (Lee et al., 2007), it may be the case that the test items on the FFAM are not maximally sensitive with respect to differentiating between different subtypes of aggression among high-risk youth. Thus, if the FFAM is not optimally effective in measuring distinct subtypes of aggressive behavior, it will be difficult to see clear divergent relationships between aggression subtypes and variables such as affect dysregulation and deficient affect. At the construct level, it is also important to recognize the generally high degree of association between reactive and instrumental forms of aggression. In light of the fact that most aggressive youth engage in a mixture of both reactive and instrumental forms of aggression, it has been debated whether “pure” subtypes of aggression exist (and thus can be measured), and whether these subtypes carry with them unique correlates and psychosocial outcomes (Dodge, 2007; Vitaro, 2007).

Moderation Effects

Moderation analyses revealed little evidence in the way of significant interaction effects between affect dysregulation and deficient affect; in each case the relationship between affect dysregulation and outcome remained comparable across groups manifesting varying levels of deficient affect. However, it is important to note that a limited sample size (total sample was split into high/low groups) may have precluded

finding significant interaction effects. Nevertheless, results from these analyses are noteworthy in that they help ensure the relationship between affect dysregulation (both the dyscontrol and suppression factors) and dependent variables was not confounded with level of deficient affect. For example, across those models that revealed no significant effects of affect dyscontrol in predicting violence and delinquency, it was important to confirm that this result did not mask intergroup differences between youth who were high versus low on deficient affect. Similarly, the consistently null relationship between affect suppression and outcomes may have concealed systematic differences among youth scoring high versus low on deficient affect.

In interpreting these results, it is also useful to consider the type of information available from variable- (e.g., SEM) versus person-based (e.g., cluster or latent class analyses) methods. Although results from the current variable-based analyses suggest that affect dysregulation and deficient affect are separate and non-interacting constructs, at the individual level adolescents can fall into one of several theoretically distinct groups representing varying levels of affect deficiency and dysregulation (e.g., high-high, high-low, low-high, low-low) while still maintaining an overall negligible relationship between these two variables. For instance, as would be expected within a traditional psychopathy framework, youth who manifest significant features of deficient affect may exhibit few difficulties with affect regulation due to their generally low levels of emotional arousal and reactivity (i.e., high-low group). When inquiring about difficulties with controlling or suppressing emotions, these questions may simply not “ring true” for youth with elevated levels of deficient affect. At the same time, it is conceivable that certain youth may exhibit features of deficient affect alongside affect dyscontrol (i.e.,

high-high). Dysregulated anger, for example, is an integral part of the larger psychopathy construct, such that many individuals with elevated scores on the PCL instruments demonstrate features of deficient affect alongside poor anger control and angry reactivity (e.g., explosive anger outbursts in which people are injured and/or property is damaged). Regarding affect suppression, the very act of inhibiting one's emotions may, on the surface, appear similar to a flat or "deficient" affective style, thereby creating a "high-high" group for these youth as well. Additionally, youth manifesting minimal features of deficient affect may conceivably exhibit high or low levels of dysregulation and emotional reactivity (i.e., low-high, low-low).

Hypothetical groups such as these underscore the heterogeneity of affective experiences that may give rise to aggression and antisocial behavior, and highlight the necessity of attending to these factors when conceptualizing and intervening in the antisocial behaviors of high-risk youth. With respect to intervention, identifying the developmental processes behind problematic behaviors and personality features among children and adolescents continues to be a crucial component in tailoring appropriate treatment efforts to specific youth. Treatment efforts will continue to be hampered to the extent that there exists significant etiological, developmental, and even phenotypic heterogeneity within samples of aggressive and antisocial youth. For instance, interventions aimed at distinguishing aggressive youth whose behavioral problems appear to originate from difficulties with affect regulation, versus those youth who evidence consistently low levels of emotional reactivity, would be crucial in tailoring treatment approaches. Interventions focused on strengthening effective affect regulation skills, tolerating affective arousal, and modulating empathic arousal (e.g., Izard, 2002) may be

valuable in the former group but less so in the latter group; indeed, for these youth affect regulation may not play a critical role in their behaviors simply because they do not experience significant levels of affective arousal to begin with.

Limitations and Directions for Future Research

There are several limitations of the current study that deserve mention. One shortcoming concerns the construct of affect regulation itself. In particular, several authors have voiced concerns regarding the diffuse and overinclusive nature of affect regulation, and have pointed to the lack of consistency across studies in how individual investigators have defined and operationalized the construct. Moreover, because affect regulation is often invoked as an explanation, cause, or even outcome for a variety of developmental phenomena and disorders, its utility and viability as a construct has been questioned (Cicchetti, Ackerman, & Izard, 1995; Cole et al., 2004; Gross, 1998; Underwood, 1997). To address the often non-specific nature of affect regulation, investigators have called for rigorous measurement and specific definitions of the phenomena encompassed by the larger construct. For instance, researchers have stressed the importance of distinguishing, both on a conceptual and operational level, the emotion itself from its regulatory properties (Cole et al., 2004).

The measure of affect regulation used in the current study, while careful to inquire about regulatory abilities that are not tied to any specific emotion, is a new measure that has not yet been examined in other independent empirical investigations. Although the psychometric properties (i.e., internal consistency, factor structure) and convergent/divergent validity of the ARC appear promising, it is not yet known whether the ARC shows satisfactory levels of validity and reliability across different samples, nor

whether it is optimal in separating the processes of affect regulation from outcome (e.g., heightened negative affect). Thus, results from the current study are unable to provide a definitive answer to the question of whether the processes of regulation, or some outcome associated with it (e.g., heightened negative affect), are ultimately responsible for the relationship between affect dysregulation and aggression.

A second limitation of the current study concerns the reliance on the PCL:YV as the sole indicator of deficient affect. Employing multiple measures across different methods (e.g., self-report, psychophysiological measures) would constitute a more rigorous means of capturing the construct of deficient affect, and would substantiate findings if similar results could be replicated across different types of measures. Multiple measures of deficient affect would also reduce the amount of contamination with other constructs as much as possible. For example, one concern with the PCL:YV is that overt behaviors (e.g., victim treatment, past violence) are often used in the coding of non-behavioral items such as those appearing on the affective factor. Therefore, to the extent that the affective factor includes construct-irrelevant variance associated with behavioral features, the relationship between the affective factor and outcomes such as aggression will be inflated.

An additional limitation of the current study was that gender was not systematically investigated across the models tested. Gender disparities have been consistently demonstrated with respect to males and females' level of engagement in aggressive and antisocial behaviors (Chesney-Lind & Sheldon, 1998; Crick & Grotpeter, 1995; Ostrov & Keating, 2004; Rys & Bear, 1997), although it appears that the gap between girls and boys' rate of engagement in violent behaviors is diminishing (U.S.

Department of Health and Human Services, 2001). An important question therefore is whether different risk factors are associated with males versus females' engagement in aggression and antisocial behavior. Regrettably, however, the vast majority of large-scale studies investigating risk for violence have utilized all male samples (e.g., Cambridge Boys Study, Pittsburgh Youth Study), thereby greatly limiting our knowledge of relevant and unique domains of risk for females (see Odgers, Moretti & Reppucci, 2005 for a discussion). Unfortunately, the sample size of the current study ($N = 179$), split among males and females, would have fallen below what is typically recommended for multiple-group modeling approaches in SEM (Bentler, 1988). With a sample size large enough to accommodate between-gender analyses, it would have been informative to assess whether the models investigated in the current study were invariant across males and females. Importantly, invariance can be tested simultaneously for both the measurement and structural portions of the models, thereby answering two key questions: (1) whether individual test items are tapping the same underlying construct (e.g., affect dyscontrol, deficient affect, reactive aggression) across gender, and (2) whether the predictive relationships among the constructs are equivalent for males and females.

In a similar vein, a larger sample would have allowed for a more explicit investigation of age. Currently, it is unknown whether age affects the manifestation of psychopathic traits, nor whether the relationship between psychopathic features and outcomes varies across different developmental stages. Including age as a covariate has been a significant oversight in prior studies investigating the relationship between psychopathic features and behavioral problems; nevertheless, it is an important consideration particularly if personality development is hypothesized to be relatively in

flux during childhood and early adolescence. Similarly, given that comorbidity of internalizing and externalizing disorders is highly prevalent in high-risk samples of children and adolescents (Kazdin, 2000), an important goal for future research will be to assess how comorbid disorders impact both the manifestation of psychopathic features as well as the relationships between psychopathic features and outcomes.

Although not a limitation per se, the design of the present study did not allow for a direct evaluation of Eisenberg and colleagues' (1996, 1998) developmental framework regarding the emergence of deficiencies in affect. The theory put forth by Eisenberg and colleagues (1996, 1998) is, by nature, a developmental theory about events that are posited to unfold over time; therefore, a test of this theory would necessitate that a measure of affect regulation be administered prior to a measure of deficient affect. Since these measures were administered concurrently in the present investigation, it remains possible that some youth manifesting features of deficient affect did, in fact, experience significant difficulties with affect regulation at some earlier point in time. An important avenue for future research, therefore, is to systematically investigate the sequential effects of affect dysregulation alongside other pertinent risk factors as they impact the development of personality (Farrington, 2006). For example, longitudinal models incorporating moderating variables could assess, for example, whether the prospective effects of affect dysregulation vary depending on the strategies used to deal with overwhelming affect (e.g., rumination versus suppression). Importantly, this type of research can begin to shed light on questions surrounding the etiology of various emotional and behavioral disorders in children and adolescents.

Relatedly, an important goal for future research is to refine our understanding of the etiological mechanisms behind the development of psychopathic traits, particularly the affective dimension which is viewed as central to the syndrome (Blackburn, 1998; Hare, 1998) and which shows consistent relationships with aggression and violence across age (Frick, Cornell, Barry et al., 2003; Vincent et al., 2003) and gender (Odgers, Repucci, et al., 2005; Penney & Moretti, 2007). Person-, rather than variable-centered, approaches will be integral in parsing the heterogeneous population of at-risk youth into more homogeneous groups based on variables of interest (e.g., affect dysregulation, psychopathy). An even finer-grained level of analysis could involve examining potential subgroups among youth with similar affective presentations. Latent class analysis and cluster based approaches may be used, for example, to assess whether there are subgroups of psychopathic youth that differ along proposed etiological dimensions such as affect dysregulation or the presence of other risk factors that have been associated with emotional problems in children (e.g., coercive parenting, insecure attachment style, and a history of abuse or neglect; Levy & Orlans, 2000; McCord & McCord, 1964; Patterson, Reid, & Dishion, 1998; Porter, 1996). A key question to ask at this point would be whether all subgroups are equally consistent with a “true” psychopathy model, the answer to which requires researchers to be clear about the theoretical boundaries surrounding the psychopathy construct as well as the assumptions behind its development.

Despite the limitations of the current study, it is one of the first to compare the relative utility of two diverse risk factors (i.e., affect dysregulation and deficient affect) that have been associated with aggression, violence, and delinquency among adolescents,

but which also encompass opposing views regarding the role of emotion in aggressive and antisocial behavior. A significant challenge for future researchers will be to construct developmentally sensitive models which can account for the manifestation of pertinent risk factors over time, and which can explain the temporal progression and transformation of certain risk factors across development (e.g., what variables are involved in the progression from affect dysregulation to deficient affect). Also needed are explicit attempts to address issues of etiology in the conceptualization and measurement of significant risk factors for violence such as psychopathy. Only then can meaningful contributions continue to be made towards constructing causal models explaining aggressive, violent, and antisocial behavior among children and adolescents.

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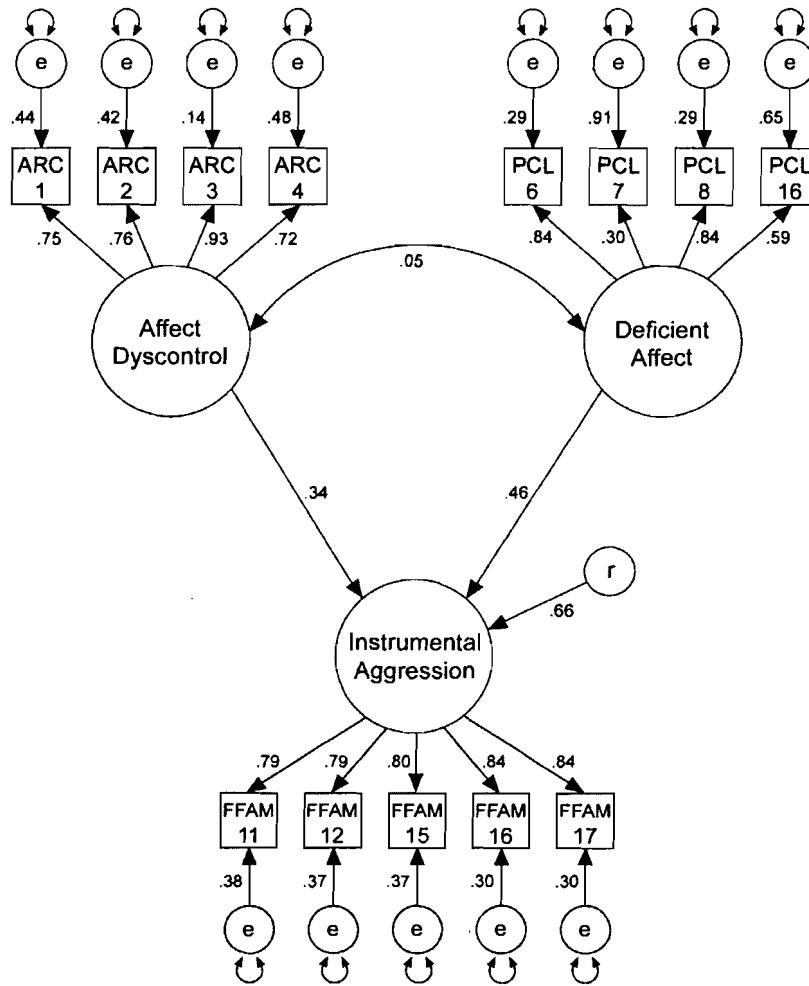
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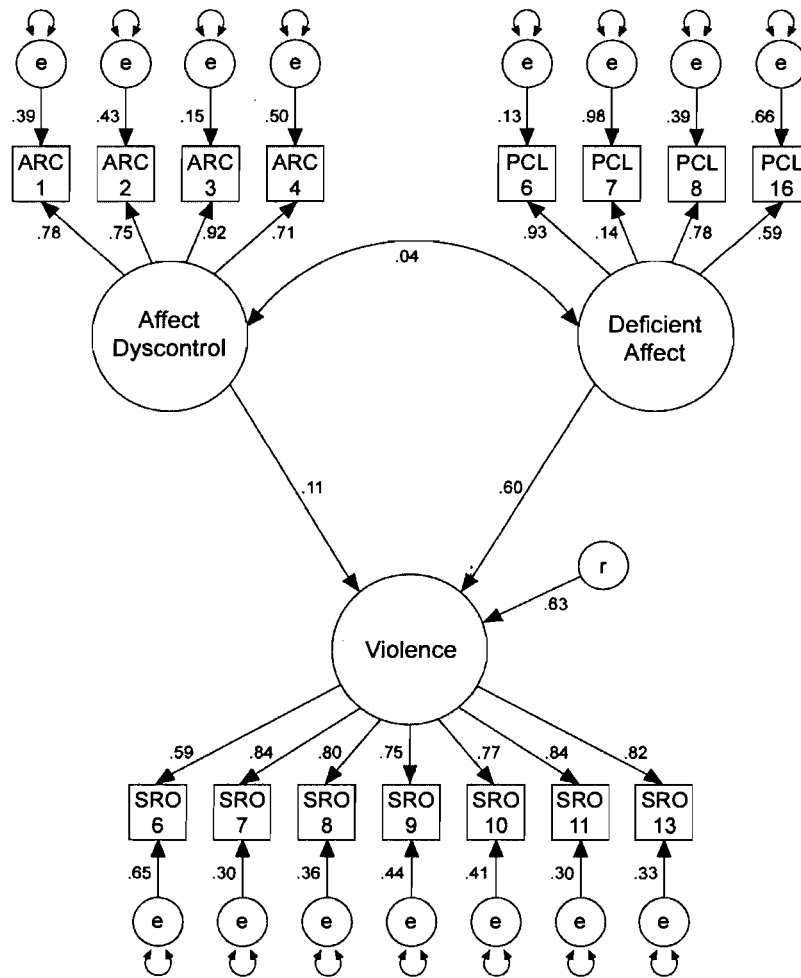
APPENDIX

Joint effects of affect dyscontrol and deficient affect on concurrent instrumental aggression.



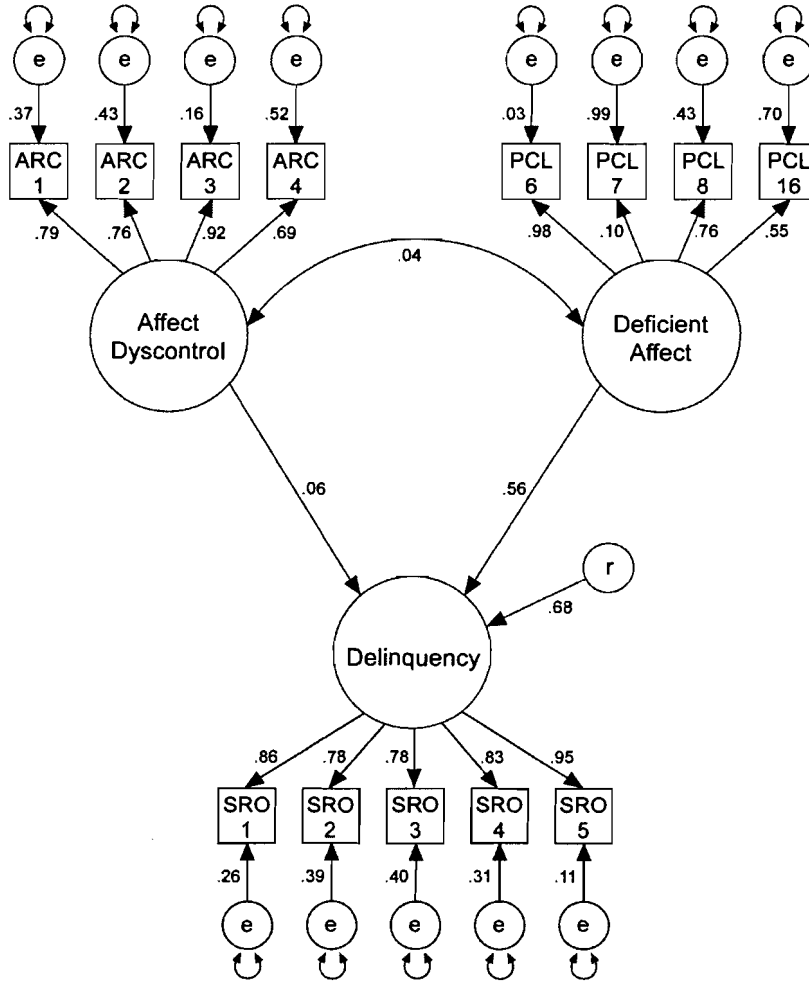
Note. CFI = .99, TLI = .99, RMSEA = .03, WRMR = .60

Joint effects of affect dyscontrol and deficient affect on concurrent self-reported violence.



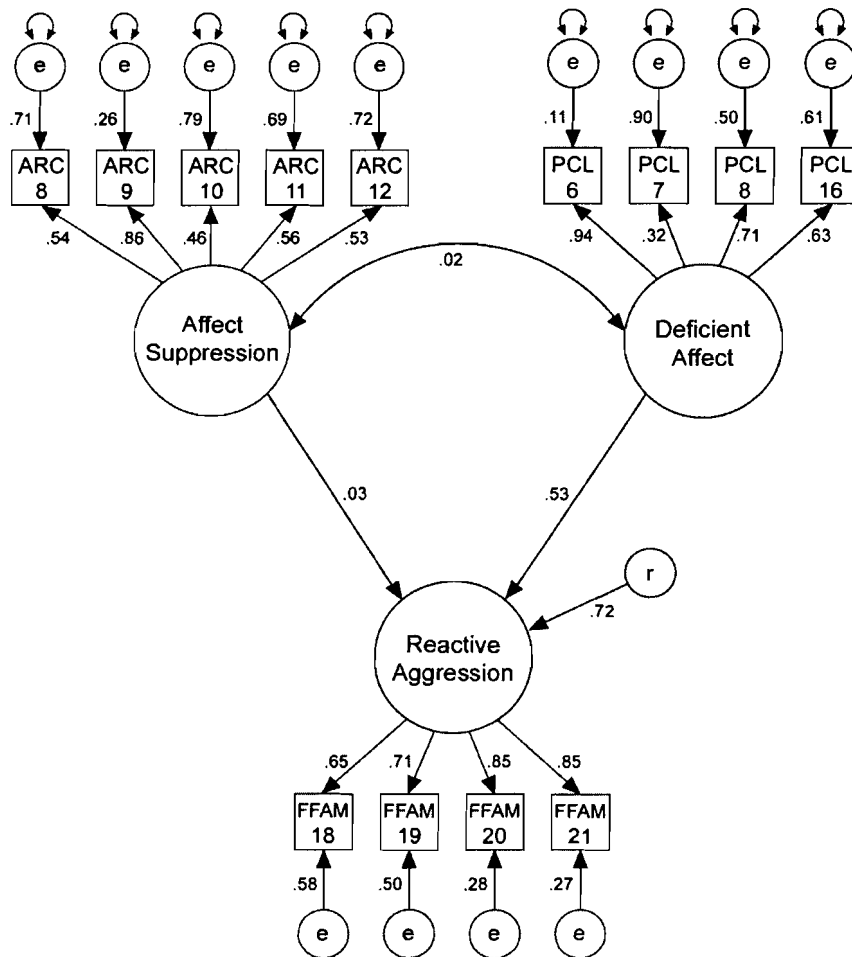
Note. CFI = .98, TLI = .98, RMSEA = .04, WRMR = .80

Joint effects of affect dyscontrol and deficient affect on concurrent self-reported non-violent delinquency.



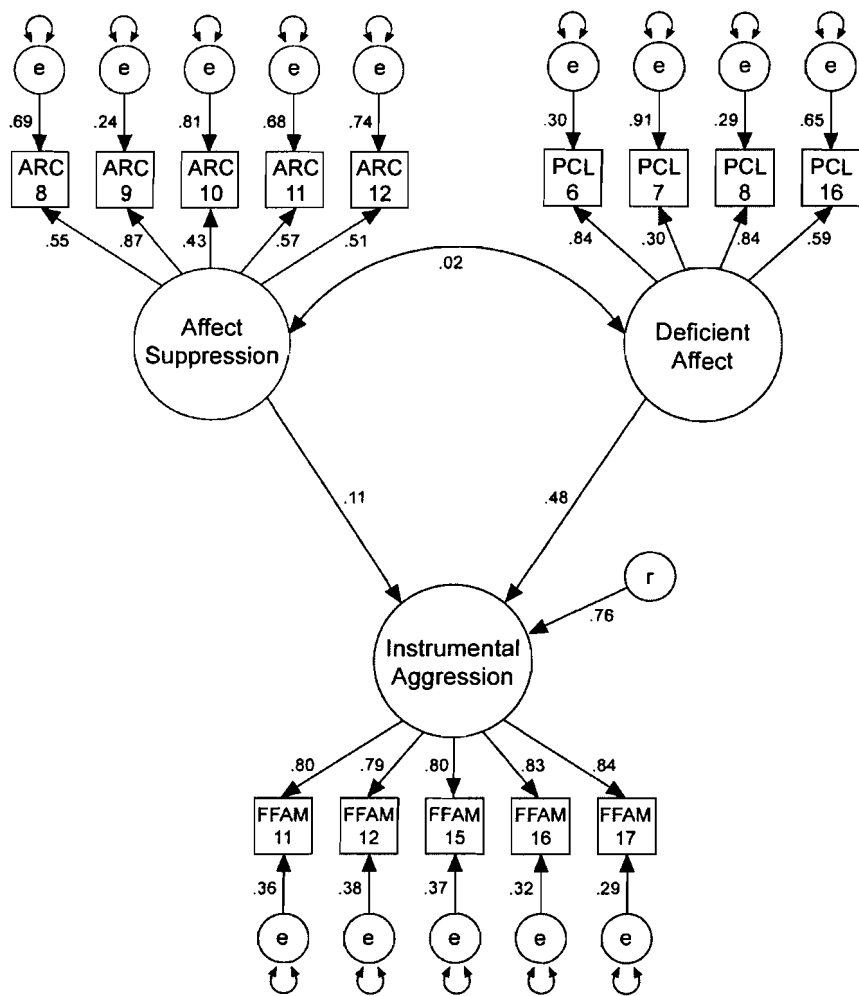
Note. CFI = .98, TLI = .98, RMSEA = .06, WRMR = .80

Joint effects of affect suppression and deficient affect on concurrent reactive aggression.



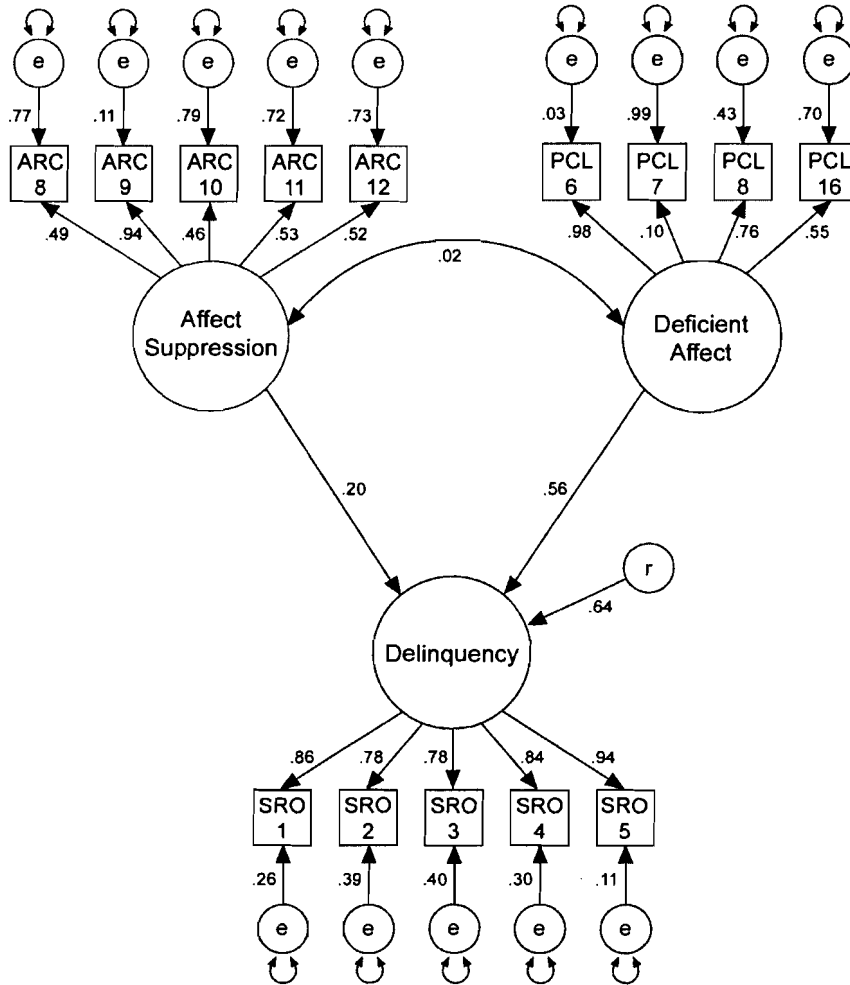
Note. CFI = .93, TLI = .93, RMSEA = .06, WRMR = .80

Joint effects of affect suppression and deficient affect on concurrent instrumental aggression.



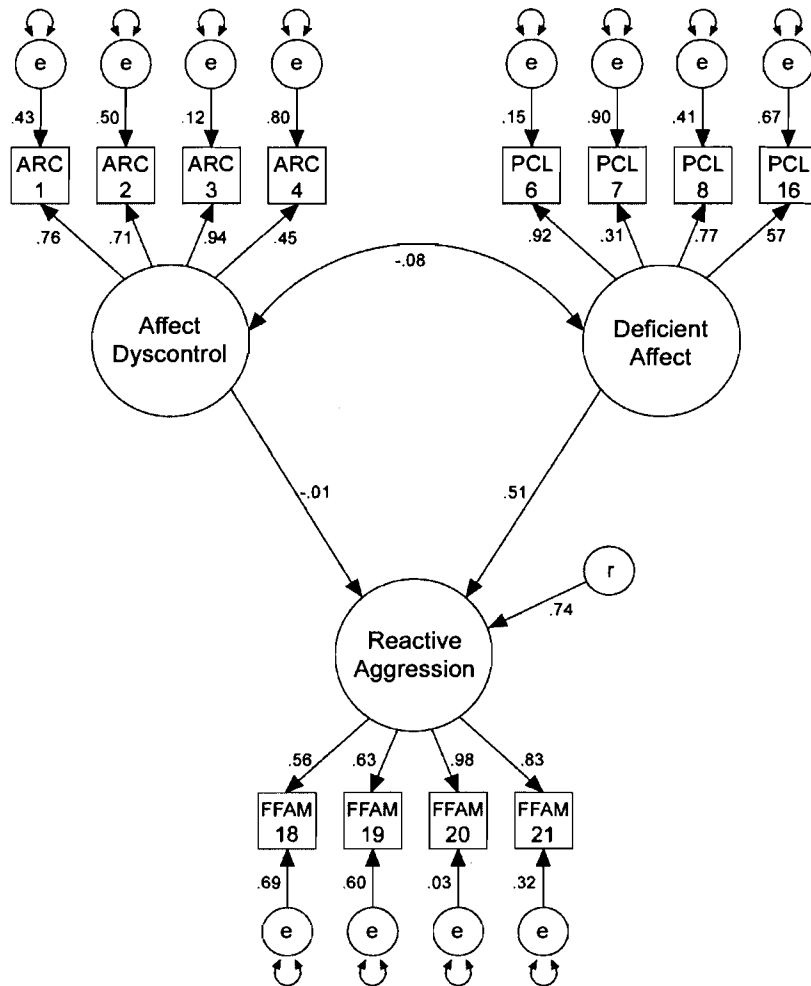
Note. CFI = .88, TLI = .91, RMSEA = .07, WRMR = .87

Joint effects of affect suppression and deficient affect on concurrent self-reported non-violent delinquency.



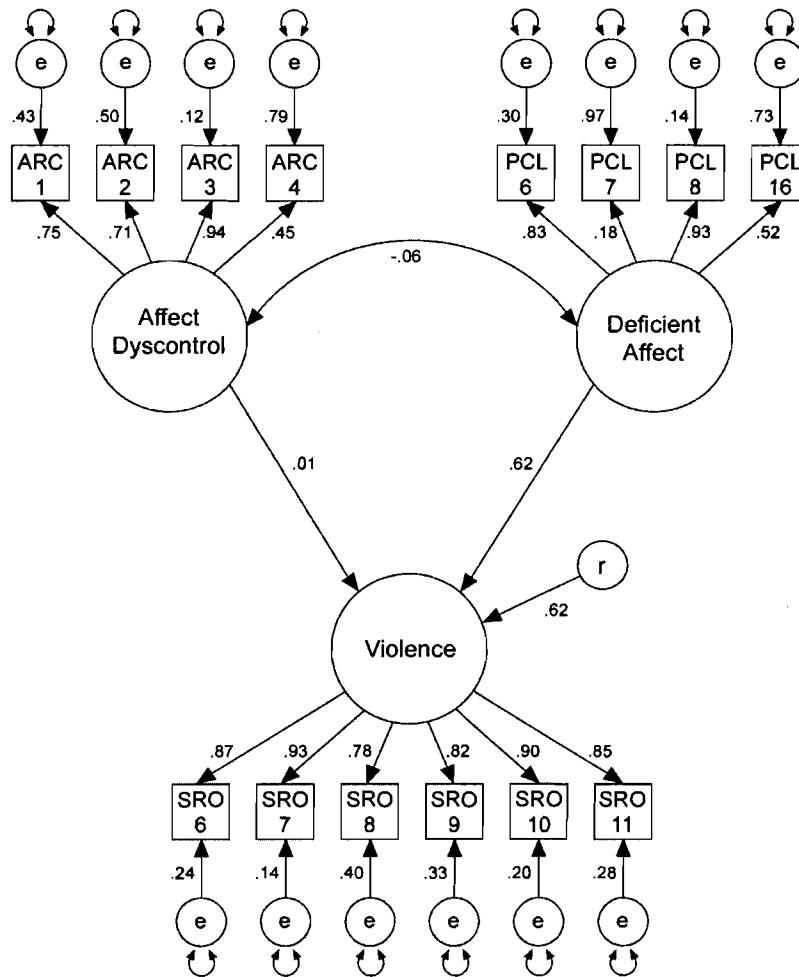
Note. CFI = .95, TLI = .95, RMSEA = .07, WRMR = .92

Joint effects of affect dyscontrol and deficient affect on prospective reactive aggression.



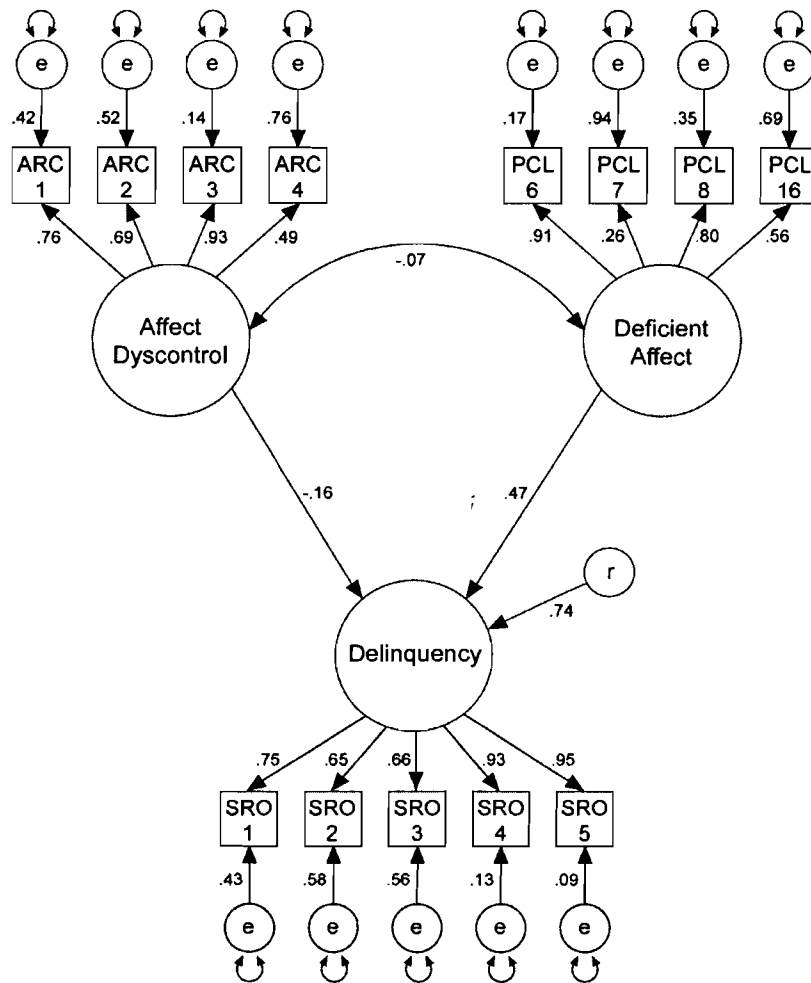
Note. CFI = 1.0, TLI = 1.0, RMSEA = .00, WRMR = .58

Joint effects of affect dyscontrol and deficient affect on prospective self-reported violence.



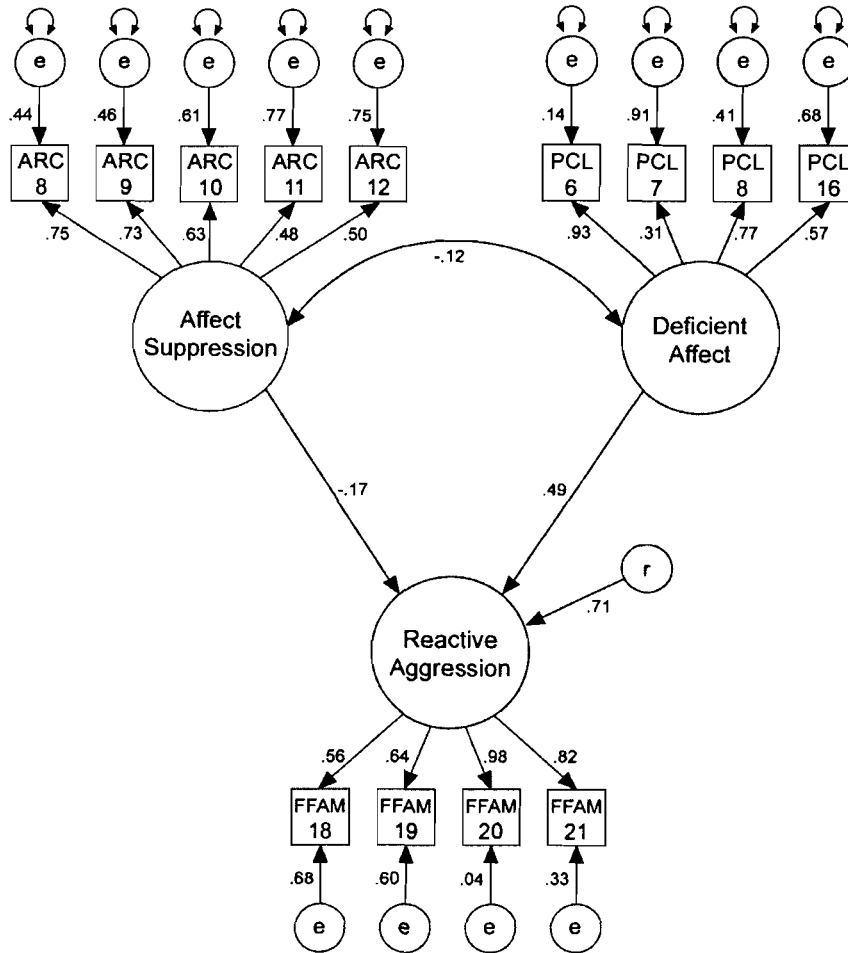
Note. CFI = 1.0, TLI = .99, RMSEA = .03, WRMR = .72

Joint effects of affect dyscontrol and deficient affect on prospective self-reported non-violent delinquency.



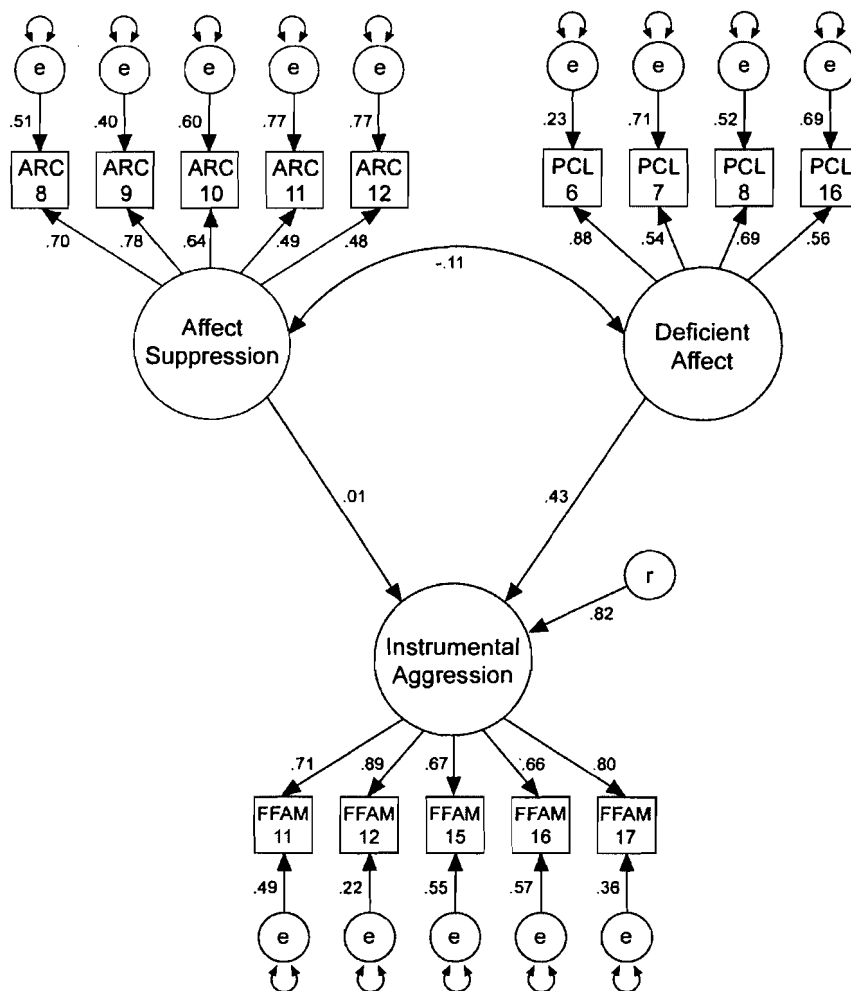
Note. CFI = .99, TLI = .99, RMSEA = .03, WRMR = .69

Joint effects of affect suppression and deficient affect on prospective reactive aggression.



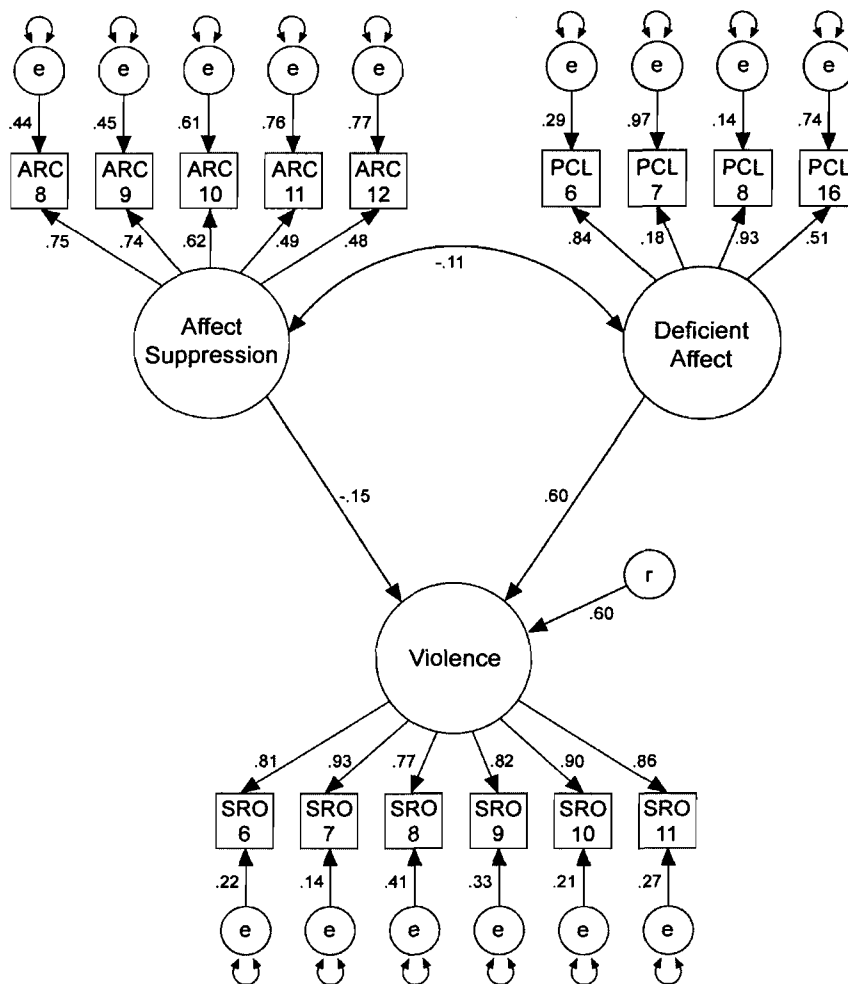
Note. CFI = 1.0, TLI = 1.0, RMSEA = .00, WRMR = .61

Joint effects of affect suppression and deficient affect on prospective instrumental aggression.



Note. CFI = .92, TLI = .92, RMSEA = .08, WRMR = .81

Joint effects of affect suppression and deficient affect on prospective self-reported violence.



Note. CFI = 1.0, TLI = 1.0, RMSEA = .01, WRMR = .70