EXECUTIVE CONTROL FUNCTION AND EMOTION REGULATION PROCESSES IN THE DEVELOPMENTAL PATHWAY FROM CHILDHOOD MALTREATMENT TO ALCOHOL USE PROBLEMS

A Dissertation
Submitted to
The Temple University Graduate Board

In Partial Fulfillment of the
Requirements for the Degree
DOCTOR OF PHILOSOPHY

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August 2015

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ABSTRACT

Executive Control Function and Emotion Regulation Processes in the Developmental Pathway from Childhood Maltreatment to Alcohol Use Problems

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Doctor of Philosophy

Temple University, 2015

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Experiencing maltreatment during childhood has been implicated in numerous and diverse developmental impairments, including problematic alcohol use and alcohol use disorders. However, little research examines the processes by which childhood maltreatment confers risk for alcohol use problems, or potential risk or protective factors in the emergence of problematic alcohol use among individuals experiencing childhood maltreatment. To address this gap, the current study investigated executive cognitive functions and emotion regulation as probable risk or protective factors linking childhood maltreatment and subsequent problematic alcohol use, given that deficits in both executive cognitive functions and emotion regulation are associated with maltreatment and problematic alcohol use. Participants were drawn from a longitudinal sample of children at both high and low risk for substance dependence, based on their paternal history of psychiatric or substance use disorder (N = 475; 70% male; 74% Caucasian, 23% African American, 3% multiracial; M = 11.38±.93 years at Time 1). Analyses involved both person- and variable-centered approaches. The person-centered approach identified groups of individuals based on maltreatment experiences, executive cognitive

functions, and emotion regulation, and then examined whether and to what extent these classes differed on concurrent and longitudinal problematic alcohol use. Findings suggested that there are distinct risk groups consisting of abuse/neglect, neglect only, executive control function deficits, and emotion regulation deficits. These groups did not differ on levels of alcohol use, counter to prediction. Variable-centered approaches involved a longitudinal examination of pathways from childhood maltreatment to alcohol use frequency and symptoms of alcohol use disorder that included executive cognitive functioning and emotion regulation. Results of these variable-centered structural equation modeling analyses indicated that maltreatment, executive control function, and emotion regulation concurrently predicted problematic alcohol use. Investigating the current models allows for a better understanding of pathways to alcohol use in both adolescence and adulthood, which has implications for prevention and intervention, particularly in identifying groups at highest risk for problematic alcohol use outcomes and in treatment selection or modification.

DEDICATION

This dissertation is dedicated to my parents, John and Ellen Hampton, who have inspired me to work hard and dream big throughout my life, and made countless sacrifices for my sake. Without their encouragement and belief in me, I would not have considered pursuing a doctoral degree. This dissertation also is dedicated to my dear husband, Brian Shields, who consistently provides me with unconditional love and support. It has been a joy and delight to go through graduate school with him as my classmate and to grow as clinicians and researchers side by side.

ACKNOWLEDGEMENTS

First and foremost, I would like to thank and acknowledge my advisor, Deb Drabick, for her valuable feedback and insight on this dissertation. She has provided unwavering support and dedication to my professional and personal development during her time as my mentor. Her passion for her research inspires me to remember the greater meaning, purpose, and implications behind the work that we do. I look forward to future opportunities to collaborate with her.

I also would like to thank and acknowledge the faculty, staff, and participating families at the Center for Education and Drug Abuse Research (CEDAR) at the University of Pittsburgh. Without the generosity of the CEDAR faculty, this project would not have come to fruition. It was their rich dataset that allowed me to pursue my research questions. I am extremely appreciative of the study coordinators and research assistants who collected the data, and to the families who participated. This parent project was sponsored by the National Institute for Drug Abuse.

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CHAPTER 1
INTRODUCTION

In the US, approximately 22.2 million individuals are substance dependent or abuse illicit drugs or alcohol (US Department of Health and Human Services, 2009), with an estimated $365 billion ($185 billion for alcohol and $181 billion for illicit drugs) spent yearly on substance abuse and addiction-related costs (Harwood, 2000; Office of National Drug Control Policy, 2004). After substance use (SU) develops into abuse or dependence, it is typically considered to be a chronic illness, with relapse rates of approximately 40% to 60% within a year following treatment (McLellan, Lewis, O'Brien, & Kleber, 2000). Perhaps more alarmingly, the majority of individuals who need treatment for an illicit drug or alcohol use problem do not actually receive it, with only 11.2% of those who needed treatment actually receiving it in 2009 (Substance Abuse and Mental Health Services Administration, 2010). The consequences of SU are life-long and often intractable, clearly illustrating the importance of reducing both initial engagement in and the impact of SU once it has been initiated. It is, therefore, critical to understand how earlier processes and individual characteristics confer risk for later SU to inform the development of both etiological models of SU and more focused prevention and intervention efforts that could potentially mitigate the negative sequelae of SU.

Researchers increasingly have suggested that substance use disorders (SUD; e.g., alcohol abuse or dependence; opiate abuse or dependence) are best conceptualized as developmental phenomena (e.g., Brown et al., 2009; Dodge et al., 2010; Haller, Handley, Chassin, & Bountress, 2010; Masten, Faden, Zucker, & Spear, 2009; Rogosch, Oshri, & Cicchetti, 2010; Schulenberg & Maslowsky, 2009; Staff et al., 2010; Tarter, 2002). There

is a strong association between developmental influences in childhood and adolescence and development of SUDs or problematic SU (as reviewed in Zucker, Donovan, Masten, Mattson, & Moss, 2008), highlighting the need for further investigation of the contribution of potential risk or protective processes (Rogosch et al., 2010). One such risk factor for later SUD is childhood maltreatment. Child maltreatment is a prevalent problem in the US; 10.2% of US children experienced some form of maltreatment during 2008, according to a nationally representative study of children (Finkelhor, Turner, Ormrod, & Hamby, 2009). Thus, it is of paramount importance to consider the impact of childhood maltreatment on later SU.

Experiencing maltreatment during childhood has been implicated as a risk factor for developing SUDs or problematic SU (e.g., Dube et al., 2003; Rogosch et al., 2010; Strine et al., 2012; Thornberry, Henry, Ireland, & Smith, 2010), with the potent association between childhood maltreatment and SUD becoming particularly evident during adolescence (Oshri, Rogosch, Burnette, & Cicchetti, 2011; Rogosch et al., 2010). Child maltreatment generally encompasses four categories, including (a) physical abuse, (b) sexual abuse, (c) emotional (or psychological) abuse, and (d) physical and emotional neglect, that may lead to potential or actual harm to a child's health, development, or dignity (Barnett, Manly, & Cicchetti, 1993; Krug, Dahlberg, Mercy, Zwi, & Lozano, 2002; Norman, Byambaa, De, Butchart, & Scott, 2012). Experiencing each of these forms of maltreatment during childhood may then influence later development and functioning.

Childhood Maltreatment as a Risk Factor for Impaired Development

Development among individuals with childhood maltreatment has been conceptualized as a transactional-ecological model (e.g., Cicchetti & Lynch, 1993;

Cicchetti & Toth, 2005; Cicchetti & Valentino, 2006), whereby risk and protective factors determine both the likelihood of abuse and subsequent development following abuse, with poor developmental outcomes occurring when there are more risk than protective factors. The numerous risk factors associated with and following maltreatment signify a deviation from the circumstances that enable typical or "normal" development (Cicchetti & Toth, 2005). As a consequence of maltreatment, children's abilities to meet developmental milestones and competencies are compromised because of a severe and fundamental failure of the home environment to provide the experience-expectant opportunities required for normal development, thus creating a pathogenic relational environment (Cicchetti & Lynch, 1995; Cicchetti & Toth, 2005). Processes occurring both at successive stages and within different domains influence other concurrent developmental processes and subsequent development within this dynamic cascade conceptualization (Masten et al., 2005; Obradović, Burt, & Masten, 2010; Sameroff, 2000), with each new stage building on and incorporating previous developmental experiences and processes (Cicchetti & Lynch, 1993). Consequently, failing to meet early competencies as a result of maltreatment and its resulting repeated developmental disruptions may then lead to other risk factors that function as more proximal influences on adolescent or adult outcomes, with potential maladaptation across diverse domains of development (e.g., Cicchetti, 2002; Cicchetti & Toth, 2005; Dodge et al., 2009; Martell et al., 2009). Over time, these risk factors may "cascade" as they compound or amplify one another to increase the likelihood of unfavorable adult outcomes (Obradović et al., 2010; Schulenberg & Maslowsky, 2009).

Among children who have experienced maltreatment, deficits have been observed in nearly all important developmental milestones and across biological and psychological domains of developmental functioning (as reviewed by Cicchetti & Lynch, 1995). These children are at an increased risk for manifesting unfavorable developmental competencies in multiple realms (Cicchetti & Toth, 1995), including emotional regulation, expression, and recognition (as reviewed by Camras, Sachs-Alter, & Ribordy, 1996); social information processing (Dodge, Pettit, Bates, & Valente, 1995); secure attachment relationships (Cicchetti, Toth, & Rogosch, 2006); autonomous self-development and self-identity (Calverley et al., 1994; Cicchetti et al., 2003); peer relations (Dodge, Pettit, & Bates, 1994; Shields & Cicchetti, 2001); and adapting to the school environment and academic progress (Eckenrode, Laird, & Doris, 1993; Shonk & Cicchetti, 2001). This maladaptive functioning potentially experienced by children who have been maltreated may then contribute to the concurrent and/or longitudinal development of psychopathology typically linked to child maltreatment.

The link between childhood trauma or maltreatment and a broad range of adverse psychological symptoms and diagnoses is robustly established (Andrews, Corry, Slade, Issakidis, & Swanston, 2004; Cicchetti & Toth, 2005; Norman et al., 2012). For example, individuals experiencing childhood maltreatment are at enhanced risk for depression, anxiety disorders, posttraumatic stress disorder, personality dysfunction and disorders (e.g., antisocial personality disorder), suicidal and self-injurious behavior, somatization, dissociation, conduct disorder and delinquency, and oppositional defiant disorder, compared to individuals who did not experience childhood maltreatment (e.g., Famularo, Kinscherff, & Fenton, 1992; Jaffee et al., 2005; Jewkes, Dunkle, Nduna, Jama, & Puren,

2010; Kaplow & Widom, 2007; Runyan, Wattam, Ikeda, Hassan, & Ramiro, 2002; Widom, Du-Mont, & Czaja, 2007). Along with this wide range of psychiatric diagnoses, the literature suggests that there is a clear association between childhood maltreatment and higher rates of SU and SUD, the evidence for which is reviewed below.

Childhood Maltreatment and Substance Use. Childhood maltreatment has been established as a robust predictor of SU (pooled OR range: 1.09 to 2) in meta-analytic work (Norman et al., 2012). In two epidemiological studies (Fergusson, Boden, & Horwood, 2008; Kendler et al., 2000), maltreatment during childhood, particularly sexual abuse, was associated with a higher prevalence of SU and SUD. Further epidemiological support for this linkage (adjusted OR range: 1.3 to 4.7) has been found in the National Epidemiologic Survey of Alcohol and Related Conditions, a nationally representative sample of adults in the US aged 20 years and older (N = 34,653), which examined physical, sexual, and emotional abuse, and physical and emotional neglect (Afifi, Henriksen, Asmundson, & Sareen, 2012). A similarly strong association between maltreatment and SU problems (OR range: 0.87 to 3.78) has been found in longitudinal work, including in a sample identified through Child Protective Services records followed through middle adulthood (Widom, Marmorstein, & White, 2006; Wilson & Widom, 2009), and in a cohort study of 1,000 urban youth followed from age 13 into adulthood (OR = 1.76; Smith, Ireland, & Thornberry, 2005). In a health maintenance organization sample, a history of adverse childhood experiences (including physical, emotional, or sexual abuse) also placed adults at enhanced risk for SU and SUD (adjusted OR range: 1-10.3), compared to those without such a history (Felitti et al., 1998).

Along with a heightened lifetime vulnerability for SU, experiencing childhood maltreatment places individuals at particularly high risk of an earlier initiation of SU during adolescence compared to individuals who do not experience maltreatment (Nomura et al., 2012; Tonmyr, Thornton, Draca, & Wekerle, 2010; Wekerle, Leung, Goldstein, Thornton, & Tonmyr, 2009). Among adolescents, findings of a survey of more than 100,000 6^{th} through 12^{th} grade public school students suggest that experiencing child physical or sexual abuse places adolescents at 2 to 4 times increased risk for alcohol, marijuana, and other drug use (Harrison, Fulkerson, & Beebe, 1997), with similarly increased risk for SU during adolescence demonstrated in work by Rodgers and colleagues (2004) and Moran, Vuchinich, and Hall (2004). Adolescent girls with a history of maltreatment also have qualitatively different patterns of SU, including being more likely to engage in polysubstance use, compared to male adolescents (Shin, Hong, & Hazen, 2010). Thus, the link between maltreatment and SU has been robustly established among both adolescent and adult samples.

Childhood Maltreatment and Alcohol Use. The research discussed thus far has included abuse or dependence of multiple illicit drugs (e.g., marijuana, opiates) and alcohol as an outcome. However, considering all substances as a composite outcome masks the heterogeneity observed in various illicit drugs and alcohol regarding timing of onset, escalation, and decline during adolescence and adulthood (Johnston, O'Malley, Bachman, & Schulenberg, 2009; Schulenberg & Maslowsky, 2009), and may result in obfuscating important developmental associations regarding onset (Dodge et al., 2009). Additionally, prior to illicit drug use, tobacco or alcohol use (AU) typically occurs (Bailey, 1992; Kandel, Yamaguchi, & Chen, 1992), although not all individuals who use

tobacco or alcohol progress to drug use (as reviewed in Kandel & Yamaguchi, 1999; Schulenberg & Maslowsky, 2009). Thus, when examining patterns of SU, it is important to consider alcohol separately from illicit drugs, rather than combining alcohol, tobacco, and illicit drugs into a composite variable.

Childhood traumatic experiences create heightened vulnerability for the development of alcohol use disorder (AUD) (Hughes, Johnson, Wilsnack, & Szalacha, 2007; Lown, Nayak, Korcha, & Greenfield, 2011; Mullings, Hartley, & Marquart, 2004; Widom, Ireland, Glynn, 1995; also reviewed in Stewart, 1996), and maltreatment during adolescence particularly places individuals at a higher risk for problematic AU (Thornberry et al., 2010). In a longitudinal study covering more than 30 years, individuals with a history of maltreatment reported a higher frequency of AU problems across their lifetime than individuals without a history of maltreatment (Herrenkohl, Hong, Klika, Herrenkohl, & Russo, 2013). A similar association between alcohol dependence with childhood physical, sexual, and emotional abuse, and physical neglect was found in a national survey of 27,712 adults, with ORs ranging from 1.45 (emotional neglect) to 2.29 (emotional abuse) (Fenton et al., 2012). An additional national survey of women found that women with a history of childhood sexual abuse reported more recent AU, intoxication, drinking-related problems, and symptoms of alcohol dependence, compared to women without such a history (Wilsnack, Vogeltanz, Klassen, & Harris, 1997). In the Adverse Childhood Experiences study, which involves an adult health maintenance organization sample, individuals experiencing adversities during childhood (including childhood abuse and neglect) had a higher risk of alcohol abuse as adults and earlier initiation of AU in early adolescence, along with a heightened likelihood of other

risk factors relating to AU, including heavy drinking, self-reported alcoholism, and marrying an alcoholic, regardless of parental alcoholism (Dube, Anda, Felitti, Edwards, & Croft, 2002; Dube et al., 2006). Adolescents also are at risk for AU, particularly use at younger ages, if they have a history of abuse (Benseley, Spieker, Van Eenwyk, & Schoder, 1999).

In spite of this preponderance of evidence supporting the relation between maltreatment and problematic AU, there have been some inconsistent findings regarding this relation among males. A study that matched controls with middle-aged adults with documented cases of child maltreatment found that women with a history of maltreatment reported higher typical quantity of drinks and more days drinking eight or more drinks, compared to their matched controls, whereas men with such a history did not differ from controls (Widom, White, Czaja, & Marmorstein, 2007). In contrast, another study demonstrated that males with a history of child maltreatment were 1.74 times more likely to meet criteria for AUD, compared to males without such a history (Young-Wolff, Kendler, Ericson, & Prescott, 2011). The findings of Young-Wolff and colleagues (2011), along with the other evidence among both males and females reviewed above, suggests that, overall, childhood maltreatment leads to a heightened susceptibility for problematic AU and AUD, though not all maltreated individuals develop AU problems.

Taking a Developmental Psychopathological Approach

More recent research has examined the pathway to adolescent SU from child maltreatment by taking a developmental psychopathological approach. Within this perspective, there are several themes that are relevant when studying child maltreatment,

including (1) typical and atypical development, (2) risk and resilience, and (3) continuity and discontinuity across development (Drabick & Steinberg, 2011).

When examining the heightened risk for impaired or atypical development among children experiencing maltreatment, it is first important to consider typical pathways of development. Understanding the challenges of experiencing child maltreatment requires being cognizant of the processes associated with typical development, to determine whether and how maltreatment results in a deviation from those processes (Cicchetti, 1993). Comparing the severe disturbances within the environment of a child who has experienced maltreatment to an average, expectable environment enables researchers to understand the processes and mechanisms that are affected by maltreatment (Cicchetti & Lynch, 1995) and then identify pathways from this atypical environment to a more adaptive course (Cicchetti, 1993; Zigler & Glick, 1986).

Central to a developmental psychopathology perspective is the importance of studying both risk and resilience, considered to be positive adaptation despite adversity (Luthar, Cicchetti, & Becker, 2000). Protective factors may offset or neutralize negative influences or risk factors, and experiencing stress may lead to enhanced coping skills, which could, in turn, result in further positive outcomes (Drabick & Steinberg, 2011), illustrating the necessity of considering risk and resilience to allow for individual differences in coping following stress. These concepts of risk and resilience are generally examined in combination, and across multiple aspects of development, such as the biological, psychological, and social-contextual domains, to enable a more complete view of how risk or protective factors lead to adverse or resilient outcomes (Cicchetti & Dawson, 2002; Drabick & Steinberg, 2011).

Finally, a fundamental part of a developmental psychopathology perspective is the consideration of individual pathways or trajectories. Specifically, multifinality and equifinality (Cicchetti & Rogosch, 1996, 2002) are important for understanding different developmental pathways from childhood experiences. Multifinality stipulates that identical initial pathways or the same starting point likely leads to varied outcomes (e.g., a variety of sequelae may be associated with childhood maltreatment beyond problematic SU). These diverse outcomes unfold because of the dynamic influence of risk and protective processes that individuals experience (e.g., different child-specific or contextual processes that confer risk or resilience; Cicchetti & Rogosch, 2002). Conversely, equifinality specifies that varied pathways or different starting points may result in a common outcome developing over time, rather than all individuals following a single progression to an outcome (Cicchetti & Rogosch, 1996, 2002). For example, individuals with problematic SU may have evidenced a variety of risk processes that contributed to this outcome, and childhood maltreatment is only one potential process that might confer risk for SU.

The developmental psychopathology approach is based on a transactional-ecological model where maltreatment generates risk factors that function more proximally to increase the likelihood of SU. Rogosch and colleagues (2010) used a developmental cascade model to examine prospective associations among child maltreatment, other risk factors, and cannabis abuse and dependence symptoms. They found both independent direct pathways from maltreatment to cannabis abuse or dependence, as well as cascading and indirect influences across development through social competence and externalizing and internalizing symptoms, suggesting that these

symptoms are sequelae of child maltreatment. Further support for a transactional-ecological model has been garnered by research linking childhood maltreatment severity and less adaptive personality functioning in childhood, to externalizing problems in preadolescence, and finally, to cannabis abuse and dependence symptoms in adolescence (Oshri et al., 2011). This recent advance in the literature with the more frequent use of a developmental cascade framework is an important addition to conceptual approaches for modeling the etiology of SU. It allows inclusion of the progressive developmental failures found among children experiencing maltreatment that precede the emergence of SU (Cicchetti & Lynch, 1993; Cicchetti & Valentino, 2006; Rogosch et al., 2010).

In spite of the recent evidence supporting the use of a developmental cascade framework to examine linkages between maltreatment and SU, little research has utilized this framework to examine AU and AUD specifically or considered the processes by which childhood maltreatment confers risk or interacts with other factors in the development of AU. Investigating these processes and the pathway from maltreatment to AU or AUD necessitates the consideration of multifinality and equifinality (Cicchetti & Rogosch, 1996, 2002). In the case of childhood maltreatment, multifinality is demonstrated by evidence that the influence and effect of trauma varies among individuals, with some maltreated individuals exhibiting resilience (Cicchetti & Rogosch, 1997; Cicchetti & Toth, 1995; Luthar, Cicchetti, & Becker 2000). Thus, though there is extensive evidence of the increased risk for AU or AUD conferred by maltreatment, not all individuals experiencing maltreatment develop problematic AU. Similarly, there are multiple distinct pathways to AU or AUD (e.g., maltreatment, difficulties with emotion regulation and executive control functioning; Fishbein, Hyde, Coe, & Paschall, 2004),

suggesting that research is needed to examine the pathway from childhood maltreatment to AU and AUD, and to determine which developmentally relevant factors affect this relation and thereby contribute to equifinality and multifinality.

A Transactional–Ecological Model of Alcohol Use

The lack of research focusing on the processes that link maltreatment and AU highlights the need for using a developmental psychopathology perspective to examine these processes, as prior research investigating illicit drug use may not be applicable to AU due to the heterogeneity observed between SU and AU patterns and development (Schulenberg & Maslowsky, 2009). Thus, the current study specifically considers maltreatment, potential factors related to risk and resilience, emotion regulation and executive cognitive functioning, and AU and AUD as developmental outcomes.

Childhood maltreatment places individuals at risk for dysregulation across major biological stress response systems (De Bellis, 2002). Deficits in regulation in emotional and cognitive functions also have been implicated in the emergence of SU (e.g., Kirisci, Vanyukov, & Tarter, 2005; Tarter, Kirisci, Feske, & Vanyukov, 2007; Tarter, Kirisci, Habeych, Reynolds, & Vanyukov, 2004). Thus, emotion and cognitive regulatory processes could function as risk or protective factors linking childhood maltreatment to adolescent and adult AU or AUD. Although there are likely multiple individual and contextual factors that may link maltreatment and AU or AUD, I focus on these self-regulatory processes given the empirical support for their relation with maltreatment and AU or AUD, their malleability throughout childhood and adolescence (Steinberg, 2008), and their potential amenability to intervention efforts that consequently could attenuate or preclude problematic SU among individuals who have experienced childhood

maltreatment. The current study thus addresses a critical gap in the literature, as little prior research has investigated putative cognitive and emotional features of child maltreatment (Pechtel & Pizzagalli, 2011), and has potentially important implications for preventive interventions.

To discuss the theoretical underpinnings of the current models, first, a conceptualization of executive control function (ECF) and emotion regulation (ER) are presented. Next, relations between these factors of risk and resilience and both child maltreatment and AU are presented, along with initial theoretical support for a model incorporating these factors, maltreatment, and AU. Finally, ECF and ER are incorporated into integrative, conceptual models of the relation between child maltreatment and AU or AUD.

Executive Control Function. Generally, ECF is conceptualized as a multidimensional set of abilities required to control complex cognition and effortfully direct behavior towards a goal, particularly in non-routine, unstructured, or novel circumstances (Banich, 2009). These abilities include control functions that are related to inhibiting and delaying responses, creating and shifting mental sets, monitoring and regulating performance, ignoring non-relevant information and utilizing relevant information for decision making, updating task demands, maintaining goals, and planning, as well as working memory and cognitive flexibility (Banich, 2009; McCabe, Roediger, McDaniel, Balota, & Hambrick, 2010). Broadly, the higher order capacities integrated in ECF are considered to exert a “supervisory” or regulating influence on behavior and goal attainment (Mezzich, Tarter, Giancola, & Kirisci, 2001).

Emotion Regulation. ER is a set of processes that influence when and how individuals experience emotions, how individuals may alter their emotions, and how emotions are ultimately expressed (Gross, 1998). Gross, Sheppes, and Urry (2011) have conceptualized ER as requiring the activation of a goal to increase or decrease either the strength or duration of an emotional response. Individuals typically regulate their emotions by trying to increase positive emotions and decrease negative ones (Gross, 2013). Within the framework of a process model of ER, an individual is able to intervene and regulate at each step in the emotion generation process, including situation selection, situation modification, attentional deployment, cognitive change, and response modulation (Gross, 1998). As ER is the process by which emotions initiate and assist in other psychological processes, or bring about difficulties and detrimental effects (Cole et al., 2004), it plays an important role in the field's understanding of typical and atypical development (Cole, Michel, & Teti, 1994).

Associations with Childhood Maltreatment

Executive Control Function. Global deficits in both cognitive function and ECF frequently are evinced among individuals who have experienced maltreatment (van der Kolk, 2003). Although early adversity such as abuse or neglect is associated with cognitive deficits generally, more complex functions, like ECF, are particularly likely to be negatively affected by maltreatment (Pechtel & Pizzagalli, 2011). This ECF impairment may be explained by alterations in prefrontal functions caused by psychosocial stress or trauma (Bremner et al., 1999, 2000). Children with a history of abuse demonstrate less improvement in response inhibition compared to non-abused children in both therapeutic and public schools, with larger differences in effect sizes

among older children (Mezzacappa, Kindlon, & Earls, 2001). Similarly, significant deficits on measures of attention and abstract reasoning/executive function were found among children with abuse-related post-traumatic stress disorder (PTSD), compared to healthy controls (Teicher, Glod, Surrey, & Swett, 1993).

Further research among children with maltreatment-related PTSD illustrates comparable findings, as children with PTSD evidence significantly poorer performance on a variety of ECF tasks, compared to sociodemographically similar children without a history of maltreatment (Beers & De Bellis, 2002). Children who experienced neglect had significantly lower IQs than controls and, even when IQ was controlled, demonstrated deficits in attention and executive functioning compared to controls (De Bellis, Hooper, Spratt, & Woolley, 2009). Furthermore, De Bellis et al. (2009) reported that greater severity of abuse was associated with decreased IQ, which confirmed similar findings by Perez and Wilson (1994). Finally, among a community sample of children, experiencing familial trauma (such as physical or sexual abuse, or witnessing domestic violence) was associated with lowered performance on a composite variable of ECF (including working memory, inhibition, auditory attention, and processing speed tasks; DePrince, Weinzierl, & Combs, 2009).

Emotion Regulation. Children who have been maltreated demonstrate significant difficulties in emotional regulation, expression, and recognition (as reviewed by Camras et al., 1996; Lyons-Ruth, 2008; Tottenham et al., 2010), which suggests that ER difficulties may link childhood maltreatment and impaired developmental outcomes. Cicchetti and Toth (2005) propose that child maltreatment is a “significant threat” to optimally developing affective processing abilities. Research on ER among individuals

exposed to child maltreatment exemplifies this conceptualization. Longitudinal research among 421 children, half of whom had been indicated as maltreated by Child Protective Services records indicates that children who had experienced more neglect, physical or sexual abuse, and maltreatment earlier in life evidenced higher emotion dysregulation (Kim & Cicchetti, 2010). Moreover, a similar tendency for emotion dysregulation was found by Maughan and Cicchetti (2002), who reported that maltreated 4- to 6-year-olds exhibited difficulties with ER after witnessing an angry situation between adults.

Associations with Alcohol Use

Along with a preponderance of evidence suggesting that lowered ECF and ER are associated with childhood maltreatment, numerous studies have demonstrated a negative association between SU with ECF and ER. In particular, impaired levels of self-regulation have been implicated as major risk factors in AU and alcohol abuse during late adolescence (as reviewed in Brown et al., 2009).

Executive Control Function. ECF difficulties, including impulsivity, poor decision-making, disinhibition, and inability to assess consequences, may contribute to risk for SU (Fishbein, 2000; Kirisci, Vanyukov, & Tarter, 2005). Giancola (2005) also posits that having lowered cognitive control over one's behavior as a result of ECF deficits may allow for maladaptive behaviors (e.g., SU and AU) to dominate. Longitudinal work among adolescents has robustly supported this relation, demonstrating that individuals with ECF deficits have higher rates of SU, total number of drugs ever tried, severity of drug involvement, and earlier onset of SUD (Aytaclar, Tarter, Kirisci, & Lu, 1999; Giancola & Parker, 2001; Tapert, Granholm, Leedy, & Brown, 2002; Tarter et al., 2003).

Moreover, ECF is considered an important factor in the development of AUD (Giancola & Moss, 1998), with a negative association found between ECF and AU (Deckel & Hesselbrock, 1996). ECF predicts age at first drink, and adolescents with disorders relating to attention (consistent with ECF deficits) are at particular risk for AUD and other SUD (as reviewed by Brown et al., 2009). Boys at risk for AUD (e.g., children of parents with AUD) also exhibit ECF deficits (Harden & Pihl, 1995). Among adults, individuals with AUD and those at high risk for AUD demonstrate impaired ECF, specifically in abstract reasoning, set shifting, set persistence, information processing, cognitive flexibility, verbal fluency, and problem solving (Beatty, Katzung, Nixon, & Moreland, 1993; Cynn, 1992; Hewett, Nixon, Glenn, & Parsons, 1991; Smith & Oscar-Berman, 1992; Tarter, 1973; Tarter & Parsons, 1971). Along with the role of ECF as a risk factor for AUD, chronic excessive consumption of alcohol has been implicated in the development and exacerbation of ECF deficits (Sullivan, Rosenbloom, & Pfefferbaum, 2000). More recent work similarly suggests that compared to controls, individuals with AUD exhibit worse performance on tasks of ECF, including planning, inhibition, rule detection, verbal fluency, problem solving, and working memory, even after a period of abstinence of up to one year from AU (Noël et al., 2001; Stavro, Pelletier, & Potvin, 2013). In summary, robust evidence suggests that ECF deficits place individuals at risk for AU and AUD, and continued AU may lead to additional risk for impaired ECF.

Emotion Regulation. Difficulties with ER also are associated with the development of SU, with some (e.g., Sher & Grekin, 2007) proposing that ER is an etiologic pathway to SU. Individuals with ER deficits who are also high in reward sensitivity may use substances as a method of reducing the distress they experience

because of their dysregulated emotions, which may increase their risk of developing SUD (Carver, Johnson, & Joormann, 2008). Research has supported this conceptualization, as affect lability and ER deficits have been implicated in AU and SU (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996; Simons, Carey, & Gaher, 2004) in both longitudinal studies (Caspi et al., 1997; Chassin, Curran, Hussong, & Colder, 1996) and among adolescent girls (Mezzich et al., 1997). ER also may maintain abuse and dependence, as affective disturbance is an aspect of withdrawal symptoms (e.g., Sellers et al., 1991), which may lead to a harmful cycle where SU results in affective disturbance, which then motivates further SU as a strategy for managing and regulating emotions (Sher & Grekin, 2007). Thus, evidence overall supports the role of ER deficits as risk factors for SUD, including AUD.

One possible model linking maltreatment, self-regulation, and AU was proposed by Klanecky and McChargue (2013), who reviewed the literature regarding associations among sexual abuse during childhood or adolescence and regulatory deficits, AU, and AUD. They propose that difficulties with effortful control, which is a construct similar to ECF and part of self-regulation (Rothbart & Bates, 2006), may increase individual's vulnerability to AU or AUD following sexual abuse in childhood or adolescence. They conceptualize these relations using a diathesis-stress model, whereby, among individuals with more deficits in effortful control, experiencing less severe sexual abuse during childhood or adolescence still increases their vulnerability to develop AUD or problematic AU. Consistent with the developmental psychopathology framework and, more specifically, the influence of risk factors, Klanecky and McChargue (2013) posit that experiencing sexual abuse during these developmental periods may interfere with the

development of effortful control, which could increase risk for later AUD or problematic AU. Although their review focuses on sexual abuse, rather than all forms of childhood maltreatment, and on effortful control, rather than including other important aspects of self-regulation (e.g., ER, ECF), the conceptualization provided by the authors further supports the role of self-regulation factors in heightening vulnerability to problematic AU or AUD among individuals with a history of maltreatment.

Gaps in the Literature

As noted, although a large literature suggests that experiencing childhood maltreatment enhances risk for later problematic AU and AUD, processes through which this association develops remain unclear. Despite more recent research utilizing a transactional-ecological framework to examine pathways from maltreatment to SU and SUD, little research has investigated AU or AUD as outcomes. Areas of research that warrant further investigation include the roles of ECF and ER, as both constructs are potential sequelae of abuse and are associated with increased risk for both development and maintenance of problematic AU. In spite of the extant work independently linking childhood maltreatment, ECF, and ER to AU and AUD, there is a gap in the literature examining concurrent associations among childhood maltreatment, AUD, and psychological regulation, such as ECF and ER. In addition, there is a particular need for these relations to be examined prospectively to determine how AU develops from adolescence into adulthood.

The current study additionally addressed a gap in the literature by examining ECF in late childhood and ER across multiple developmental time periods. ECF and ER have a very protracted course of development, continuing on through childhood, adolescence,

and adulthood (Anderson, 2002; Banich, 2009; Calkins & Hill, 2007; Gross & Thompson, 2007; Raffaelli, Crockett, & Shen, 2005; Steinberg, 2007, 2008; Thompson & Meyer, 2007; Zelazo, Frye, & Rapus, 1996). Because typical ER development features a shift towards increasingly healthier patterns of ER during adulthood (John & Gross, 2004), considering ER at multiple time points can inform models of the role of ER in the development of AU and AUD.

The Current Study

The current study aimed to investigate the relations among childhood maltreatment, ECF, and ER with AU frequency and AUD symptoms at various time points from late childhood through young adulthood. I first determined whether childhood maltreatment, ECF, and ER independently predict AU frequency and AUD symptoms both cross-sectionally and prospectively. I then used person-centered analyses to identify groups of youth based on (a) self-reported childhood maltreatment prior to age 18, (b) ECF measured at ages 10-12 (Time 1), and (c) ER at ages 10-12 (Time 1). To further examine these groups, I explored relations of the groups with AU and AUD, both cross-sectionally at ages 10-12 (Time 1), and prospectively at ages 12-14 (Time 2), 16 (Time 3), 19 (Time 4), and 22 (Time 5) to examine equifinality in AU. Finally, I utilized a variable-centered approach, structural equation modeling, to evaluate a transactional-ecological model linking childhood maltreatment to AU frequency and AUD symptoms through ECF and ER, which permitted a test of multifinality regarding AU and AUD. Specific goals and hypotheses for the current study are outlined below. Given the lack of research in some areas, hypotheses are generated using the available (albeit limited) literature and knowledge of typical developmental changes in the constructs of interest.

The current study used both person- and variable-centered approaches to examine relations between child maltreatment and later AU and AUD. Using a variable-centered approach (e.g., regression, analysis of variance, structural equation modeling) allows for models that best describe the behavior and patterns of a sample on average, which enables researchers to better understand general tendencies in the population. However, solely utilizing a variable-centered approach does have limitations, as this approach will produce findings that may not be applicable to individuals who greatly deviate from the mean on variables of interest (Labouvie, Pandina, & Johnson, 1991; Nylund, Bellmore, Nishina, & Graham, 2007). Person-centered approaches, such as latent class analysis (LCA), allow for examining observed patterns of responses within individuals, and then grouping heterogeneous groups of individuals into classes based on quantitative and/or qualitative similarities on dimensions of interest (Muthén & Muthén, 2000; Nylund et al., 2007). LCA assumes that there is an underlying categorical latent variable that determines class membership (Nylund et al., 2007), which enables generating reliable classes based on the use of predictor and outcome variables taken from multiple methods of measurement and assessment, with diverse variances.

Aim 1. To examine self-reported childhood maltreatment (indexed retrospectively at Time 6, age 25), and ECF and ER at ages 10-12 (Time 1) as predictors of AU frequency and AUD symptoms both cross-sectionally (Time 1) and prospectively (Times 2, 3, 4, and 5) to determine if findings regarding these relations in the existing literature are replicated in the present sample, given that the current study involves different indices and time points for the constructs of interest. This aim was preliminary and occurred prior to the main analyses of interest.

Hypothesis 1. Consistent with prior research, I hypothesized that higher levels of AU frequency and AUD symptoms would be associated with higher rates of all types of childhood maltreatment and deficits in ECF and ER across all time points.

Aim 2. To identify groups of youth defined by type and levels of (a) self-reported childhood maltreatment prior to age 18, (b) ECF at ages 10-12 (Time 1), and (c) ER at ages 10-12 (Time 1) self-reported by index youth and by mothers reporting on index youth.

Hypothesis 2. I hypothesized that I would identify four groups of youth based on their history of childhood maltreatment, ECF, and ER. Specifically, I expected groups of youth exhibiting (a) high levels of childhood maltreatment, and low levels of both ECF and ER (High Mal + Low ER/ECF); (b) high levels of childhood maltreatment and moderate or relatively high levels of both ECF and ER (High Mal + High ER/ECF); (c) low levels of childhood maltreatment and low levels of both ECF and ER (Low Mal + Low ER/ECF); and (d) low levels of childhood maltreatment and relatively high levels of both ECF and ER (Low Mal + High ER/ECF). This hypothesis was based on prior evidence suggesting that children enduring maltreatment may be at particular risk for impaired ER and ECF, though some children with this history may have intact psychological regulation. Additionally, it was expected that some children will report lower rates of childhood maltreatment, with these children demonstrating variability regarding ECF and ER.

Aim 3. To explore relations among the maltreatment, ECF, and ER classes with AU and AUD, both cross-sectionally at ages 10-12 (Time 1), and prospectively at ages 12-14 (Time 2), 16 (Time 3), 19 (Time 4), and 22 (Time 5).

Hypothesis 3a. Given low rates of alcohol use at ages 10-12, I hypothesized that the multiple risk group (High Mal + Low ER/ECF) would have higher levels of AU and AUD cross-sectionally relative to the other subgroups identified. I hypothesized that levels of AU and AUD in the three other groups would be minimal or zero at ages 10-12 (Time 1).

Hypothesis 3b. It was hypothesized that classes with higher levels of maltreatment and/or lower levels of ER/ECF would exhibit elevated levels of AU and AUD relative to classes with lower levels of youth maltreatment and higher levels of ECF/ER, given prior evidence that maltreatment and lower ECF and ER increase vulnerability to AU during adolescence and adulthood. Specifically, the following prospective relations were hypothesized: (1) High Mal + Low ER/ECF group membership will have the highest levels of AU and AUD compared to all other groups, (2) Low Mal + Low ER/ECF group membership will have the second highest levels of AU and AUD, (3) High Mal + High ER/ECF group membership will have the second lowest levels of AU or AUD, and (4) Low Mal + High ER/ECF group membership will have the lowest levels of AU and AUD compared to all other groups.

Aim 4. To utilize structural equation modeling to evaluate a transactional-ecological model linking childhood maltreatment to longitudinal AU frequency and AUD symptoms through ECF and ER.

Hypothesis 4a. I hypothesized that there will be longitudinal continuity in ER, which would be demonstrated by ER at one time point predicting ER at the following time point. I expected to observe similar longitudinal continuity for both AU and AUD.

Hypothesis 4b. Consistent with previous work, I expected that maltreatment will predict both ECF and ER difficulties.

Hypothesis 4c. Childhood maltreatment was expected to predict AU frequency and AUD symptoms at Times 1, 2, 3, 4, and 6. I also hypothesized that deficits in ECF and ER will be associated with higher AU frequency and more AUD symptoms over time.

Hypothesis 4d. I hypothesized that there would be pathways from maltreatment to AU and AUD through ECF or ER, as difficulties with ECF and ER were expected to be associated with problematic AU among individuals experiencing maltreatment.

CHAPTER 2
METHOD

Participants

Participants were enrolled in a National Institute on Drug Abuse (NIDA)-funded prospective study conducted at the Center for Education and Drug Abuse Research (CEDAR) at the University of Pittsburgh that aimed to delineate the etiological pathways to SUD. Fathers with a child aged 10-12 years (index child) were recruited through substance treatment programs and other mediums. They were then further screened based on exclusion and inclusion criteria. Index children were classified into one of three groups at both high and low risk for substance dependence (SUD): (a) offspring of biological fathers with a history of SUD, (b) offspring of biological fathers with a history of a psychiatric disorder not including SUD, and (c) offspring of biological fathers with no lifetime history of either SUD or psychiatric disorder. Determination of the father's SUD history was based on meeting lifetime *DSM-III-R* criteria (APA, 1987) for any substance abuse or dependence disorder.

Participant recruitment and baseline assessments began in 1990 when the index children were 10-12 years old (Time 1, N=775). Follow-up assessments occurred when the index children were 12-14 years old (Time 2, n=653); 16 years old (Time 3, n=625); 19 years old (Time 4, n=584); 22 years old (Time 5, n= 481); and 25 years old (Time 6, n=475). Index children were included in the present study if they completed the assessment of childhood trauma (described below; n=475) at Time 6, along with the assessments at Time 1. Of the participants included in the present study, there were more males than females (70% male) because only males were initially recruited; recruitment of females began following changes in NIH regulations about gender equity four years

after the CEDAR project began. Participants in the present study were predominantly Caucasian (74%; 23% African American; 3% multiracial), and their average age at Time 1 was 11.38±.93 years. For a more detailed description of the study methodology, recruitment sources, and procedures, along with inclusion and exclusion criteria, see Clark et al. (1997).

Procedure

The University of Pittsburgh Institutional Review Board (IRB) has continuously approved the CEDAR project (grant number 2 P50 DA05605), along with its informed consent procedures, and consent and assent forms. The present project was determined to be exempt by the Temple University Institutional Review Board because the analyses involved the study of existing data and the information was recorded by the investigators in such a manner that participants cannot be identified. The purpose and nature of the research procedures and risks/benefits were explained to all participants prior to obtaining written documentation of informed consent from adult participants. All participants younger than 18 assented to participate and provided written documentation of this assent. Eighty-seven percent of fathers who met criteria to participate consented. Participant confidentiality was assured through a Certificate of Confidentiality provided by NIDA. Childhood maltreatment was measured via self-report at Time 6. Self-report of AU frequency was measured via self-report at Times 1, 2, 3, 4, and 5, and AUD symptoms were measured via structured clinical interview at those same time points. Youth neuropsychological functioning was measured at Time 1 using laboratory measures. Youth emotion regulation was measured at Times 1, 3, and 5 using both mother- and self-report. Each of these measures is presented in greater detail below.

Measures

Childhood maltreatment. Childhood maltreatment was self-reported by participants at Time 6 using the brief screening version of the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003), which is a 28-item retrospective assessment of abuse and neglect during childhood and adolescence. This brief screening version was developed based on a 70-item self-administered inventory (Bernstein et al., 1994) to provide more rapid screening for maltreatment histories in both clinical and nonreferred populations and to reduce respondent burden. Participants rated items on a scale from 1 (*never true*) to 5 (*very often true*).

The CTQ consists of five clinical, empirically derived subscales: physical abuse, defined as, "bodily assaults on a child by an adult or older person that posed a risk of or resulted in injury;" sexual abuse, defined as, "sexual contact or conduct between a child younger than 18 years of age and an adult or older person;" emotional abuse, defined as, "verbal assaults on a child's sense of worth or well-being or any humiliating or demeaning behavior directed toward a child by an adult or older person;" physical neglect, defined as, "the failure of caretakers to provide for a child's basic physical needs, including food, shelter, clothing, safety, and health care," and poor parental supervision, if it placed the child's safety in question; and emotional neglect, defined as, "the failure of caretakers to meet children's basic emotional and psychological needs, including love, belonging, nurturance, and support" (Bernstein et al., 1994; Bernstein, Ahluvalia, Pogge, & Handelsman, 1997). The brief screening version of the CTQ includes five items for each type of maltreatment, along with an additional three-item minimization/denial validity subscale that was included to detect the underreporting of abuse and neglect

(Bernstein & Fink, 1998). Relevant items are summed to create a total score for each subscale.

The brief screening version of the CTQ has demonstrated criterion-related validity, which was assessed by comparing adolescents' scores on the CTQ subscales to therapist ratings of the adolescents' experiences of each type of abuse and neglect (Bernstein et al., 2003). CTQ factors predict therapist ratings of their adolescent clients' experiences of abuse and neglect, and scores on the CTQ were significantly correlated with therapist ratings. Additionally, the brief screening version of the CTQ has measurement invariance, as the factor structure, pattern of factor loadings, and covariances among the latent dimensions are equivalent across clinical and normal populations, including substance-abusing adults in clinical settings and the community, adolescent psychiatric inpatients, and a normative community sample of adults (Bernstein et al., 2003), which supports its appropriateness for usage with the CEDAR sample of participants with varying backgrounds and risk for SU. Finally, the CTQ has excellent test-retest reliability, along with convergent and discriminant validity with a structured trauma interview (Bernstein et al., 1994).

Executive control functioning. Participants completed a battery of ECF tests that were administered by trained master's-level clinical associates at Time 1. This administered battery includes the following tasks: Stroop Color Word Test (Stroop, 1935), Porteus Mazes Test (Porteus, 1965), Vigilance Test (Schneider & Detweiler, 1987), Motor Restraint Test (Parsons, Tarter, & Edelberg, 1972), Forbidden Toys Test (Cole, Usher, & Cargo, 1993; Silverman & Ragusa, 1992), and Block Design Test of the WISC-III-R (Wechsler, 1972). Previous work has suggested that scores on these tests

(described below) constitute a single first-order factor that can discriminate between youth at high and low risk for SUD and predict SU between childhood and adolescence (Aytaclar et al., 1999). Thus, a composite of the combined scores on these tests was used as an index of ECF in the present study.

For the Stroop Color Word Test, the ability to suppress perceptual interference, which indicates attentional control, was tested as participants were asked to label the colors of word stimuli while ignoring the word name (e.g., to say "red" when the word "blue" is printed in red ink; Stroop, 1935) using a computer-interactive format. For the Porteus Mazes Test (Porteus, 1965), participants were required to trace the paths of increasingly difficult mazes as a measure of planning ability. The Vigilance Test (Schneider & Detweiler, 1987) involved participants' viewing rapidly changing computer displays of four single letters in a 2×2 matrix while required to hit the space bar when 1 of 2 target letters appear, with errors of commission to non-target stimuli (e.g., responding when the child should instead inhibit) used to index impulse errors. For the Motor Restraint task (Parsons et al., 1972), participants were asked to trace the outline of a 180° arc as slowly as possible on a computer monitor using a light-pen, with performance on this task indicated by the total time taken to complete five trials. In the Forbidden Toy task (Cole et al., 1993; Silverman & Ragusa, 1992), participants performed a chip-sorting task in the presence of toys that they were instructed not to play with, while observed through a mirror by two experimenters, who rated the frequency of off-task distraction. The two raters' mean scores (interrater reliability correlation coefficient = .98) were used to indicate off-task behavior. The Block Design task

(Wechsler, 1972) required youth to organize blocks into patterns that matched a model design presented by the examiner in a series of timed trials.

Emotion regulation. Temperament was self-reported by participants at Times 1, 3, and 5, and reported by mothers at Time 1 using the Revised Dimensions of Temperament Survey (DOT-S; Windle, 1992). The Mood Quality subscale (7 items, α = .87) from the DOT-S was used as an index of emotion regulation. Participants and mothers rated items on a four-point scale from 1 (*usually false*) to 4 (*usually true*), with higher scores indicating higher levels of temperamental positive mood and lower scores indicating higher levels of negative mood (Tarter et al., 2003). Based on previous research in the current sample (Kirisci & Blackson, 1996; Mezzich et al., 2007), ER was indexed by the sum of endorsed responses on this subscale. Previous evidence in this sample also suggests that higher scores on the DOTS-R differentiate youth at high risk for substance abuse from those at from low risk (Blackson, Tarter, Martin, & Moss, 1996; Windle, 1991), along with distinguishing adolescent substance abusers from controls (Tarter & Mezzich, 1991).

To further index ER, the Dysregulation Inventory (DI; Mezzich et al., 2001), which is a 92-item measure of self-regulation, also was used. Participants completed the DI at Times 1, 3, and 5, and their mothers completed the DI at Times 1 and 3. Participants rated items on a four-point scale from 0 (*never true*) to 3 (*always true*). The DI assesses three different aspects of dysregulation: emotional/affective subscale, which involves arousability, emotional control, and irritability; behavioral subscale, which involves aggression, impulsivity, inattention, and hyperactivity; and cognitive, which involves ability to devise a plan, ability to execute a plan, ability to learn from

experience, and cognitive flexibility. The DI, and its abbreviated version, have demonstrated significant relations with other established measures of emotional and behavioral distress (Mezzich et al., 2001; Pardini, Lochman, & Frick, 2003), including among the current sample (Mezzich et al., 2007). Previous evidence also has demonstrated that the DI significantly discriminates between abstinent drug abusers and controls (Fishbein et al., 2005).

Alcohol use frequency. At Times 1, 2, 3, 4, and 5, participants reported frequency of AU during phases in their lifetime (for Time 1) or since the prior visit (for all other time points), with the length of the phase self-defined by participants as a period of time in which their behaviors changed from one level of drinking to another. These self-defined phases could be a few weeks or many years, depending on how they were assigned and depending on participant attendance at prior visit times. Participants rated their frequency of drinking based on a scale of 1 (*every day*) to 11 (*once*). The phase with the highest frequency of drinking beer, liquor, or wine was used at each time point as the participants' alcohol use frequency score.

Alcohol use disorder symptoms. At Times 1, 2, 3, 4, and 5, symptoms of AUD (e.g., alcohol abuse, alcohol dependence) were assessed using an expanded version of the Structured Clinical Interview for *DSM-III-R* (SCID; Spitzer, Williams, & Gibbon, 1987). Although the *DSM-III-R* was the most recent version of the SCID when the study began and was used to determine parental psychiatric diagnoses, *DSM-IV* (APA, 1994) criteria were applied subsequent to its publication to identify symptoms of AUD for the index child. The SCID included extensive assessment of AU behavior for the phase at which the participant reported the highest frequency of use since their previous visit. A

committee of a board-certified psychiatrist (chair), another psychiatrist or clinical psychologist, and the research associate who conducted the interview used the best-estimate procedure to derive symptom counts. If participants endorsed a symptom in one or more of the self-defined phases of alcohol use since the prior visit, the participant was considered to endorse that symptom for that time point.

Socioeconomic status. The Hollingshead ratings (Hollingshead, 1990) were used to index household socioeconomic status (SES; $M = 41.40$, $SD = 13.62$). Although investigating the relations between the variables of interest and SES was not an aim of the current project, SES was examined in Aims 1 and 3 because it is related to recruitment group status in the current sample (Moss et al., 2003) and previous research has documented higher rates of substance abuse among individuals of lower SES (Dohrenwend et al., 1992).

Paternal Axis I psychiatric diagnoses. Lifetime paternal psychiatric and SUD diagnoses were made based on *DSM-III-R* criteria using the Structured Clinical Interview for *DSM-III-R* (SCID; Spitzer et al., 1987), which was the most recent version of the *DSM* at the time of recruitment and initial data collection. As previous research in the current sample has demonstrated that SU, SUD, neuropsychological functioning, and ER are individually related to paternal psychiatric history (Aytaclar et al., 1999; Moss et al., 2003), paternal risk status was examined in analyses for Aims 1 and 3. Paternal risk status was recoded into a two-level categorical variable. Participants with fathers with a history of psychiatric or SU diagnosis were considered High Risk (51.8%) and coded 1, and participants with no paternal psychiatric history were considered Low Risk (48.2%) and coded 0.

Analysis Plan

SPSS/PASW were used for preliminary analyses, and Mplus version 7.11 (Muthén & Muthén, 1998-2014) was used for the primary analyses. As only participants for whom the assessment of childhood trauma using the CTQ was completed were included in the present study, independent samples *t*-tests were conducted between participants with data for the CTQ and those who were missing data for that measure to determine if there were differences between included and excluded individuals on the variables of interest in the present study, and to address concerns about generalizability. Other preliminary analyses included computing descriptive statistics and correlation matrices to determine if there were any problems with distributions (e.g., outliers, skewness, kurtosis) or multicollinearity among the data.

Only participants who completed the Childhood Trauma Questionnaire at Time 6 were included in the current study. However, given the longitudinal nature of the present data, participants could have missing data at other time points on the variables of interest. Thus, Full-Information Maximum Likelihood (FIML) estimation, which conducts parameter estimation and estimates standard errors all in one step using all available data (Graham, 2009), was used to address missing data among the sample. Other strategies for managing missing data (e.g., listwise or pairwise deletion, mean imputation) may result in biased analyses (Bodner, 2008; Graham, 2009; Little & Rubin, 2002). Rather than estimating values for missing data, as would occur for mean- or regression-based imputation techniques, FIML fits the covariance structure model directly to the available, observed raw data for each participant (Enders, 2001). In FIML, the missing data are assumed to be either missing completely at random (MCAR) or missing at random

(MAR), the latter of which was expected to be more likely to characterize the present data.

Aim 1

The first research aim was to determine if self-reported childhood maltreatment, ECF, and ER were associated with AU and AUD. Separate hierarchical ordinary least squares regressions were conducted to determine if (a) childhood maltreatment measured at Time 6, (b) ECF at Time 1, and (c) ER at Time 1 predict AU frequency and AUD symptoms at Times 1, 2, 3, 4, and 5, controlling for SES, sex, and paternal Axis I psychiatric diagnostic status.

Aim 2

To identify groups of youth with varying risk profiles for AU based on (a) self-reported childhood maltreatment prior to age 18 (indexed at Time 6), (b) ECF measured at ages 10-12 (Time 1), and (c) ER measured at ages 10-12 (Time 1), LCA was used (see Figure 1). LCA (Muthén & Muthén, 2000) is a person-centered data reduction technique that empirically derives groups of individuals, referred to as latent categorical classes, based on the aggregation of observed categorical and/or continuous variables. Each latent class (e.g., a risk group categorized by high levels of maltreatment, and low levels of ECF and ER) describes the relations among the observed items (i.e., severity of ECF and ER deficits and childhood maltreatment). Empirically deriving latent classes of observed items is considered to be more effective than using a traditional classification with cut-off scores (Nylund et al., 2007), as using arbitrary cut-off scores could lead to false positives or negatives because of classification errors, reduced ability to predict differences in outcomes, challenges identifying and generalizing rates of behaviors, and reduced

validity in identifying groups of youth who may benefit from intervention efforts. Thus, LCA was an appropriate approach for identifying varying risk profiles based on childhood maltreatment, ECF, and ER.

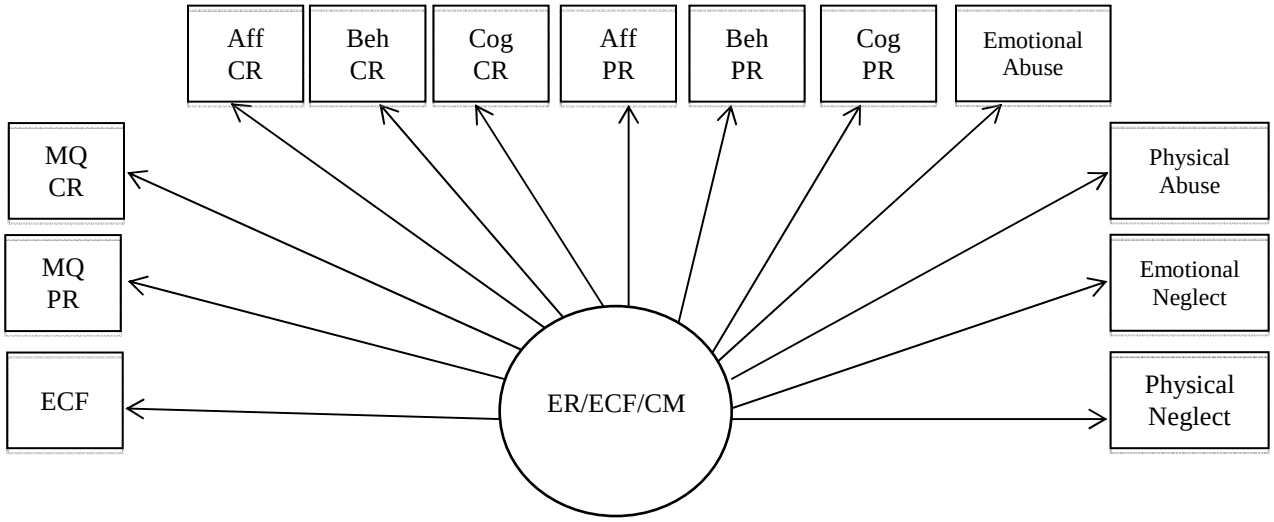

Figure 1. Latent class model of index youth ECF and ER at Time 1 (ages 10-12). PR = Parent Report, CR = Child Report, ECF = Observed executive control function composite variable, MQ = Observed DOT-S mood quality items, Aff = Observed Dysregulation Inventory Affective subscale items, Beh = Observed Dysregulation Inventory Behavioral subscale items, Cog = Observed Dysregulation Inventory Cognitive Subscale items, ECF/ER/CM = Latent variable describing type and severity of index youth executive control functioning, emotion regulation abilities, and childhood maltreatment.

The purpose of LCA is to identify the best fitting model for the observed data based on statistical fit indices and conceptual/practical implications (Nylund et al., 2007), with the goal of finding the smallest number of classes that could adequately describe associations among the observed variables. First, an unconditional 1-class model is specified, which consists of the mean levels of the maltreatment, ECF, and ER variables. An increasing number of classes is then specified and compared to the model with one fewer class in an iterative process until the models specified no longer converge and/or

no longer are conceptually sound. The fit of each model is examined using item probability parameters and class probability parameters. Item probabilities refer to the likelihood of an individual in a given latent class endorsing an item, whereas class probabilities represent the prevalence of each latent class in the population. Next, posterior class probabilities are examined to determine if the majority of participants in the sample are placed in a latent class that reflects their respective levels of severity of childhood maltreatment, ECF, and ER. Posterior class probabilities are based on both item and class probabilities, and reflect a participant's probabilities of being in each latent class, based on their response pattern for the observed items. Each participant is assigned to membership in a latent class based on their highest posterior probability score (i.e., modal class assignment).

Determining Model Fit. As there is no single "gold standard" of statistical fit indices for LCA, a combination of indices was used to assess model fit and determine the optimal number of classes, based on recommendations by Nylund and colleagues (2007). For the current study, the Bayesian Information Criterion (BIC; Schwartz, 1978); sample-size adjusted BIC (ABIC; Sclove, 1987); Akaike Information Criterion (AIC; Akaike, 1987); and Bootstrap Likelihood Ratio Test (BLRT; McLachlan & Peel, 2000; Nylund et al., 2007) were used to assess model fit. For the BIC, ABIC, and AIC, lower values correspond to better fitting models. As the BIC penalizes a large number of parameters or small class sizes, this index generally identifies the best fitting model as one with larger class sizes and fewer parameters. The BLRT estimates log likelihood differences, and compares the fit of a model with *k* classes to a model with *k-1* classes to determine if model fit significantly improves with an additional class. The BLRT and BIC indices are

considered the most reliable indicators of model fit in LCA (Nylund et al., 2007). Others tests for model comparison include entropy scores, which indicate the distinguishability between latent classes and the precision with which participants are placed into classes (Masyn, Henderson, & Greenbaum, 2010). Entropy values closer to one indicate clearer delineation of classes (Celeux & Soromenho, 1996), suggesting that classes are distinct and distinguishable from one another. Class size also should be taken into consideration when the smallest class is not conceptually meaningful and/or does not represent a significant proportion of the sample (e.g., <5%), as very small classes suggest over-fitting of the data and reduced likelihood of model replication in future studies. Finally, model selection should be based on the conceptual model and theories underpinning the analyses, the interpretability of the number of classes, and practical considerations, including how well the identified classes correspond with hypothesized classes.

Aim 3

Levels of AU and AUD were examined among classes with different risk profiles to test the predictive validity of the identified classes. Tests of equality of means across the identified classes were conducted to determine whether and to what extent classes differ regarding (a) AU frequency at Times 1, 2, 3, 4, and 5; and (b) symptoms of AUD at Times 1, 2, 3, 4, and 5. SES, sex, and paternal Axis I psychiatric diagnoses also were examined in tests of equality of means across the identified latent classes. For the test of equality of means, class membership is held constant and χ^2 statistics for omnibus tests, along with pairwise comparisons across latent classes, are provided. These pairwise comparisons are interpreted only if the omnibus test is significant. All analyses were determined to be significant if $p < .05$.

Aim 4

Structural equation modeling was utilized to evaluate a transactional-ecological model linking childhood maltreatment to AU frequency and AUD symptoms through ECF and ER. First, a measurement model using confirmatory factor analysis was examined to assess goodness of fit for the following latent variables: childhood maltreatment, consisting of the 5 subscales of the CTQ; ER at Time 1, consisting of mother and child reports of ER using the DOT-S mood quality subscale and mother and child reports of ER using the DI; ER at Time 3, consisting of child reports of ER using the DOT-S mood quality subscale and mother and child reports of ER using the DI; and ER at Time 5, consisting of child reports of ER using the DOT-S mood quality subscale and using the DI. Loadings of the observed indicators on these latent variables were examined using standardized effect estimates to determine the ability of the measurement model (latent variable) to explain the observed variables that load on them. If evaluation of these latent variables suggested that model modification was required, modifications were made to the latent variables based on suggested modification indices if theoretically tenable. Modifications (e.g., omitting or correlating variables) were made until adequate model fit was achieved for each latent variable.

Next, using the final measurement models for each of the predictors, pathways between variables were added. Although AU frequency and AUD symptoms were examined separately, identical measurement models were used. In the model examined, the maltreatment latent variable predicted ER at all three time points, Time 1 ECF, and the AU (Figure 2) or AUD (Figure 3) outcome variables at Times 1, 2, 3, 4, and 5. To evaluate goodness of fit, the following indices were used: Pearson χ^2 statistic, the Root

Mean Squared Error of Approximation (RMSEA), the Tucker-Lewis Index (TLI), the Comparative Fit Index (CFI), and the Standardized Root Mean Residual (SRMR). A significant χ^2 indicates possible poor model fit; however, this index is sensitive to sample size and may falsely indicate poor fit in larger samples (Ullman & Bentler, 2009). The RMSEA compares the model and the actual data to measure lack of fit. RMSEA values of less than 0.05 indicate close fit (Brown & Cudeck, 1993), though some have suggested that RMSEA values of 0.08 and below are sufficient (Byrne, 2001). The TLI is an incremental fit index that penalizes models for adding parameters, with better fit indicated by having a lower χ^2 to degrees of freedom ratio, and the CFI assesses discrepancies between the current model and the null model. Both TLI and CFI scores range from 0 to 1, and scores of 0.95 are considered to demonstrate excellent fit. The SRMR is the square root of the difference between the residuals of the sample covariance matrix and the hypothesized covariance model, and SRMR scores range from 0 to 1 (Hooper, Coughlan, & Mullen, 2008). SRMR values of less than .08 are associated with acceptable fit (Hu & Bentler, 1999). After examining these indices of model fit, it was determined if model modification was required. Modifications suggested by the modification index that were theoretically tenable were made until good model fit is achieved, with a balance between improving model fit and maintaining the conceptual model of interest without overfitting the model to the current data. Once the model fit was adequate, the paths were interpreted. This process was repeated for both AU/AUD outcome variables.

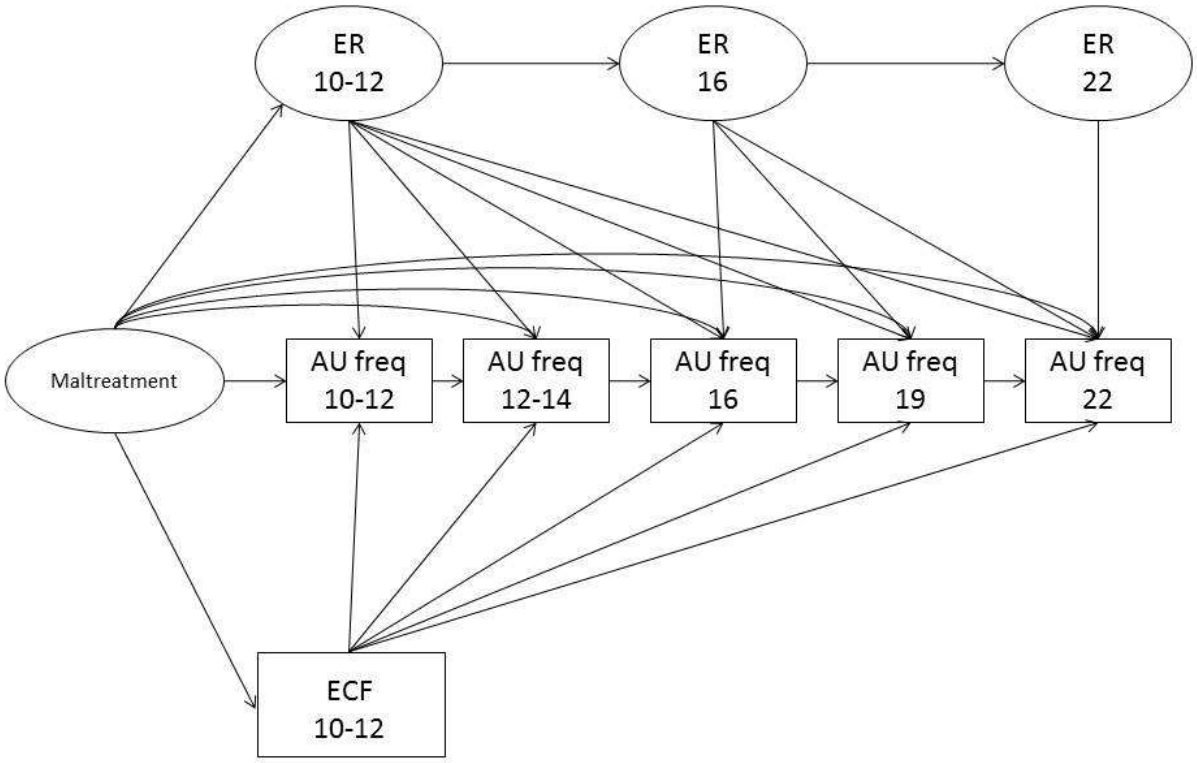

Figure 2. Predictive model indicating pathways from childhood maltreatment, executive cognitive functioning (ECF), and emotion regulation (ER) to alcohol use (AU) frequency.

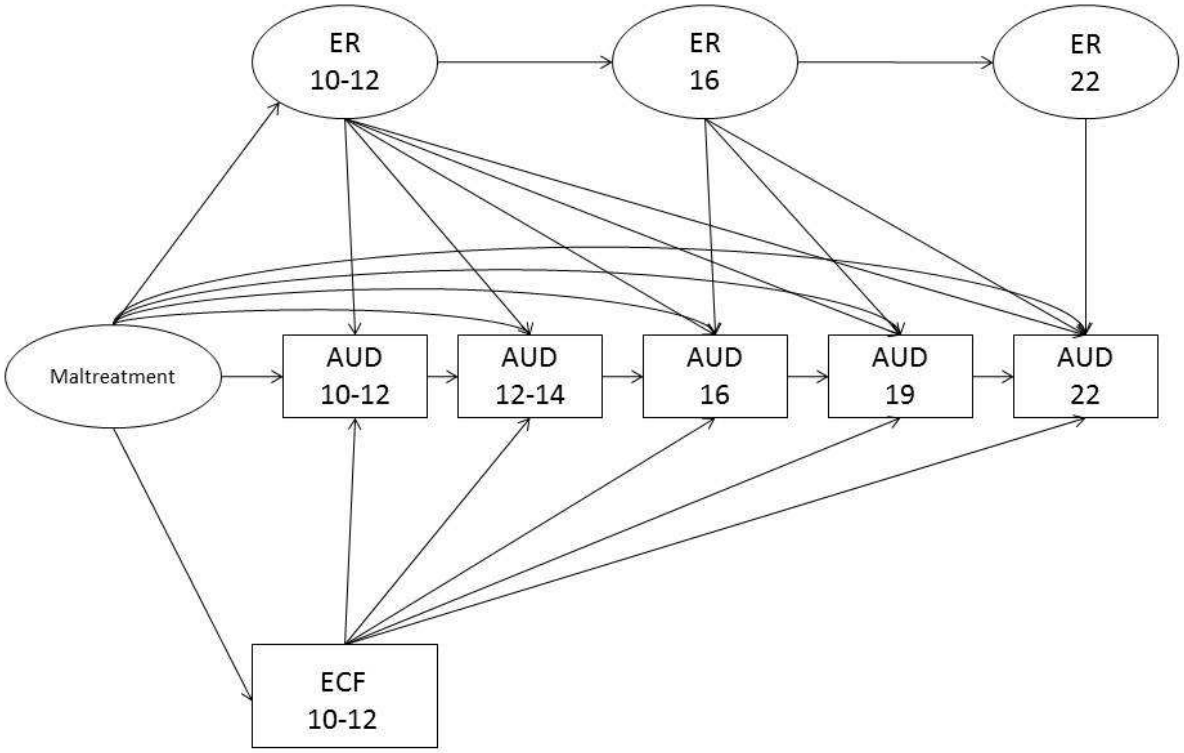

Figure 3. Predictive model indicating pathways from childhood maltreatment, executive cognitive functioning (ECF), and emotion regulation (ER) to alcohol use disorder (AUD) symptoms.

CHAPTER 3
RESULTS

Descriptive Statistics

Means, standard deviations, and *n*'s for the study variables for participants included and excluded from the current study because of missing Childhood Trauma Questionnaire data are presented in Table 1. Independent samples *t*-tests were conducted to compared study participants and excluded participants on the independent and dependent variables. No significant differences were found between the included and excluded groups (all *t*s < 1.68, all *p*s > .10).

Table 1.
Descriptive Statistics for the Independent and Dependent Variables for Included Study Participants and Participants Excluded because of Incomplete Childhood Trauma Questionnaire Data

	Study Participants		Excluded Participants	
	n	Mean (*SD*)	*n*	Mean (*SD*)
Childhood Trauma Questionnaire				
Emotional Abuse	475	6.90 (2.89)	0	N/A
Physical Abuse	475	6.21 (1.95)	0	N/A
Sexual Abuse	475	5.25 (1.29)	0	N/A
Emotional Neglect	475	8.03 (3.72)	0	N/A
Physical Neglect	475	6.22 (2.11)	0	N/A
Dysregulation Inventory – Child Report				
Time 1				
Affective	215	25.80 (12.99)	151	25.09 (14.33)
Behavioral	216	27.99 (14.37)	151	28.23 (16.60)
Cognitive	215	30.83 (9.17)	151	30.92 (9.03)
Time 3				
Affective	183	21.03 (12.07)	110	22.74 (13.70)
Behavioral	183	26.31 (13.12)	110	27.97 (14.84)
Cognitive	183	28.37 (9.55)	110	30.35 (10.21)
Time 5				
Affective	366	17.07 (12.49)	107	17.62 (12.61)
Behavioral	367	21.86 (13.83)	107	23.00 (14.41)
Cognitive	366	25.62 (9.86)	107	26.63 (10.22)
Dysregulation Inventory – Parent Report				
Time 1				
Affective	218	21.03 (11.24)	153	19.52 (11.12)
Behavioral	218	26.67 (13.55)	153	26.13 (14.68)
Cognitive	218	34.67 (10.20)	153	34.35 (9.35)
Time 3				
Affective	172	16.72 (11.81)	105	16.66 (12.04)
Behavioral	172	21.31 (12.35)	105	21.01 (14.50)
Cognitive	172	30.48 (10.94)	105	29.73 (12.80)

Revised Dimensions of Temperament Survey Mood Quality Subscale – Child Report				
Time 1	446	23.54 (3.62)	277	23.59 (3.94)
Time 3	419	23.82 (3.97)	206	24.19 (3.82)
Time 5	368	23.90 (4.26)	99	24.49 (4.05)
Revised Dimensions of Temperament Survey Mood Quality Subscale – Parent Report				
Time 1	472	25.30 (3.15)	292	25.27 (3.14)
Executive Control Function Composite (Time 1)	475	.02 (.99)	277	-.02 (1.05)
Alcohol Use Disorder Symptoms				
Time 2	423	.02 (.25)	230	.04 (.39)
Time 3	419	.16 (.73)	206	.12 (.41)
Time 4	409	.60 (1.29)	175	.49 (1.13)
Time 5	377	1.15 (1.74)	105	.84 (1.62)
Alcohol Use Frequency				
Time 1	475	.17 (2.40)	300	.03 (.18)
Time 2	423	1.56 (7.95)	230	1.06 (4.83)
Time 3	419	13.61 (45.46)	206	13.14 (45.37)
Time 4	409	46.62 (77.97)	175	45.24 (72.26)
Time 5	377	93.46 (103.88)	105	95.47 (105.25

Bivariate correlations among childhood maltreatment, ER, and ECF variables are presented in Table 2. As expected, all maltreatment variables were correlated with one another. Table 2 also illustrates the numerous positive correlations among maltreatment variables and subscales of the DI, particularly at Time 5, suggesting that higher levels of maltreatment are associated with more dysregulation. Additionally, there are multiple negative correlations between the maltreatment variables, particularly emotional and physical neglect, and the DOT-S mood quality subscale variables, suggesting that more maltreatment is associated with more negative mood quality.

Table 2.
Bivariate Correlations among Child Maltreatment, Emotion Regulation, and Executive Control Function Variables

	(1)	(2)	(3)	(4)	(5)
(1) Emotional Abuse	-	.47***	.15**	.56***	.40***
(2) Physical Abuse		-	.17***	.337***	.27***
(3) Sexual Abuse			-	.10*	.17***
(4) Emotional Neglect				-	.57***
(5) Physical Neglect					-
(6) T1 CR DI Affective	.05	-.02	.04	.06	.01
(7) T1 CR DI Behavioral	.01	.02	.02	.04	-.01
(8) T1 CR DI Cognitive	-.06	-.02	-.03	-.01	-.09
(9) T3 CR DI Affective	.19*	.05	.14	.13	.06
(10) T3 CR DI Behavioral	.18*	.14	.12	.14	.07
(11) T3 CR DI Cognitive	.21**	.13	.14	.25**	.12
(12) T5 CR DI Affective	.30***	.16**	.12*	.26***	.34***
(13) T5 CR DI Behavioral	.29***	.21***	.05	.23***	.25***
(14) T5 CR DI Cognitive	.23***	.15**	.08	.30***	.04***
(15) T1 PR DI Affective	.02	.07	.02	.11	.03
(16) T1 PR DI Behavioral	.03	.13	.03	.10	.03
(17) T1 PR DI Cognitive	.05	.14*	-.01	.17*	.13
(18) T3 PR DI Affective	.07	.15	.21**	.08	.14
(19) T3 PR DI Behavioral	.05	.15	.09	.03	.07
(20) T3 PR DI Cognitive	.06	.15*	.10	.09	.15*
(21) T1 CR Mood Quality	-.10*	-.11*	.07	-.21***	-.08
(22) T3 CR Mood Quality	-.12*	-.10	-.01	-.28***	-.18***
(23) T5 CR Mood Quality	-.11*	-.13*	-.06	-.25***	-.31***
(24) T1 PR Mood Quality	-.04	-.05	.02	-.14**	-.09*
(25) ECF	.13**	.23***	.07	.15**	.20***

Note: T1 = Time 1, T3 = Time 3, T5 = Time 5, CR = Child Report, PR = Parent Report, DI = Dysregulation Inventory, ECF = Executive Control Function Composite
* $p < .05$, ** $p < .01$, *** $p <. 001$.

Bivariate correlations among childhood maltreatment and AU variables are presented in Table 3. As expected, most AU variables were correlated with one another. Several maltreatment variables were significantly correlated with AU variables. In particular, AU frequency at Time 3 was positively correlated with physical and sexual abuse, and emotional neglect, and Time 4 AU was positively correlated with physical and sexual abuse, and emotional and physical neglect.

Table 3.
Bivariate Correlations among Child Maltreatment and Alcohol Use Variables

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
(1) T2 AUD	-	.23***	-.02	.015	.01	.45***	.02	.04	.21***
(2) T3 AUD		-	.09	.119*	-.01	.14**	.49***	.07	.11*
(3) T4 AUD			-	.355***	-.01	.12*	.17**	.54***	.21***
(4) T5 AUD				-	.15**	.06	.18**	.36***	.59***
(5) T1 AU Freq					-	.07	.02	.01	.06
(6) T2 AU Freq						-	.10	.20***	.18**
(7) T3 AU Freq							-	.24***	.15**
(8) T4 AU Freq								-	.39***
(9) T5 AU Freq									-
(10) Emotional Ab	.03	-.02	.07	.062	-.03	.02	.06	.09	.01
(11) Physical Ab	.04	.01	.07	.079	.02	.06	.11*	.20***	.01
(12) Sexual Ab	-.02	.11*	.06	.098	-.01	-.02	.14**	.15**	.06
(13) Emotional Neg	.08	.03	.11*	.102*	-.03	.03	.10*	.13*	.09
(14) Physical Neg	-.01	-.04	.14**	.118*	-.02	.06	-.01	.14**	.07

Note: T1 = Time 1, T2 = Time 2, T3 = Time 3, T4 = Time 4, T5 = Time 5, AUD = Alcohol Use Disorder, AU Freq = Alcohol Use Frequency, Ab = Abuse, Neg = Neglect
* $p < .05$, ** $p < .01$, *** $p <$ .001.

Bivariate correlations among ECF and ER variables are presented in Table 4. ECF was positively correlated with most subscales of the DI and negatively correlated with the DOT-S mood quality subscale variables, suggesting that higher ECF is associated with less dysregulation and more negative mood quality, which is unexpected. In general, the ER measures were correlated with one another.

Table 4.

Bivariate Correlations among Executive Control Function and Emotion Regulation Variables

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)	(20)
(1) ECF	-	.18 **	.17 **	.14 *	.12	.18 *	.19 *	.20 ***	.14 **	.16 **	.12	.27 ***	.32 ***	.09	.23 **	.34 ***	-.19 ***	-.18 ***	-.20 ***	-.12 *
(2) T1 CR DI Aff		-	.84 ***	.52 ***	.23 **	.30 ***	.17	.15	.12	.10	.21 **	.17 *	.11	.12	.12	.06	-.21 **	-.13	.02	-.06
(3) T1 CR DI Beh			-	.59 ***	.18 *	.39 ***	.19 *	.07	.11	.15	.16 *	.22 **	.14 *	.10	.18 *	.15	-.25 ***	-.16 *	-.04	-.08
(4) T1 CR DI Cog				-	.12	.19*	.25 **	.13	.07	.17 *	.11	.18 **	.23 **	.05	.07	.12	-.20 **	-.09	-.05	-.09
(5) T3 CR DI Aff					-	.71 ***	.43 ***	.52 ***	.37 ***	.29 ***	.28 ***	.18 *	.12	.46 ***	.33 ***	.27 ***	-.18 *	-.37 ***	-.30 ***	-.01
(6) T3 CR DI Beh						-	.55 ***	.42 ***	.52 ***	.39 ***	.32 ***	.30 ***	.18 *	.44 ***	.50 ***	.39 ***	-.23 **	-.32 ***	-.28 **	-.05
(7) T3 CR DI Cog							-	.33 ***	.36 ***	.51 ***	.18 *	.30 ***	.29 ***	.34 ***	.35 ***	.42 ***	-.27 ***	-.29 ***	-.36 ***	-.09
(8) T5 CR DI Aff								-	.80 ***	.63 ***	.14	.04	.10	.33 ***	.16	.23 **	-.11 *	-.15 **	-.36 ***	-.11 *
(9) T5 CR DI Beh									-	.67 ***	.13	.15	.05	.25 **	.23 **	.24 **	-.13 *	-.11 *	-.27 ***	-.08
(10) T5 CR DI Cog										-	.15	.21 **	.21 **	.27 **	.26 **	.33 ***	-.21 ***	-.20 ***	-.41 ***	-.13 *
(11) T1 PR DI Aff											-	.69 ***	.53 ***	.58 ***	.43 ***	.36 ***	-.06	-.19 *	-.08	-.23 **
(12) T1 PR DI Beh												-	.66 ***	.48 ***	.58 ***	.50 ***	-.09	-.23 **	-.10	-.12
(13) T1 PR DI Cog													-	.37 ***	.45 ***	.59 ***	-.19 **	-.18 *	-.27 ***	-.24 ***
(14) T3 PR DI Aff														-	.78 ***	.56 ***	-.11	-.23 **	-.07	-.23 **
(15) T3 PR DI Beh															-	.73 ***	-.10	-.26 **	-.13	-.20 **
(16) T3 PR DI Cog																-	-.14	-.29 **	-.13	-.20 **
(17) T1 CR MQ																	-	.28 ***	.21 ***	.13 **
(18) T3 CR MQ																		-	.45 ***	.15 **
(19) T5 CR MQ																			-	.10 *
(20) T1 PR MQ																				-

Note: ECF = Executive Control Function Composite, T1 = Time 1, T3 = Time 3, T5 = Time 5, CR = Child Report, PR = Parent Report, DI = Dysregulation Inventory, Aff = Affective, Beh = Behavioral, Cog = Cognitive, MQ = Mood Quality

* $p < .05$, ** $p < .01$, *** $p <. 001$.

Table 5 presents bivariate correlations among Time 1 ER and ECF variables, and AU variables. Unexpectedly, ECF at Time 1 was not correlated with any AU variables at any time points. Similarly unexpected, very few Time 1 ER variables were correlated with AU variables, with the exception of several ER variables with Times 2 and 3 AU frequency.

Table 5.
Bivariate Correlations among Alcohol Use Variables and Time 1 ECF and ER Variables

	(10) ECF	(11) T1 CR DI Aff	(12) T1 CR DI Beh	(13) T1 CR DI Cog	(14) T1 PR DI Aff	(15) T1 PR DI Beh	(16) T1 PR DI Cog	(17) T1 CR MQ	(18) T1 PR MQ
(1) T2 AUD	-.07	.08	.10	.10	.04	.10	.12	-.02	-.05
(2) T3 AUD	.03	.03	.08	.04	-.03	-.02	.03	.01	-.01
(3) T4 AUD	-.06	.05	.12	.10	.10	.11	.01	-.02	.00
(4) T5 AUD	.01	-.01	.03	.01	-.05	.01	.06	-.03	-.05
(5) T1 AU Freq	.07	-.04	.07	.04	.01	.06	.09	.05	-.04
(6) T2 AU Freq	-.07	.13	.20**	.17*	.08	.18*	.17*	.01	-.06
(7) T3 AU Freq	.02	.06	.15*	.07	-.05	-.03	.02	-.02	-.01
(8) T4 AU Freq	-.09	-.13	-.07	-.05	-.10	-.07	-.07	.08	-.03
(9) T5 AU Freq	-.04	-.01	-.08	.01	-.12	.01	-.04	-.03	-.09

Note: T1 = Time 1, T2 = Time 2, T3 = Time 3, T4 = Time 4, T5 = Time 5, AUD = Alcohol Use Disorder, AU Freq = Alcohol Use Frequency, ECF = Executive Control Function, ER = Emotion Regulation, CR = Child Report, PR = Parent Report, DI = Dysregulation Inventory, Aff = Affective, Beh = Behavioral, Cog = Cognitive, MQ = Mood Quality
* $p < .05$, ** $p < .01$, *** $p <$. 001.

Table 6 presents bivariate correlations among Time 3 ER variables, and AU variables. Unexpectedly, very few Time 3 ER variables were correlated with AU variables.

Table 6.
Bivariate Correlations among Alcohol Use Variables and Time 3 Emotion Regulation Variables

	(10) T3 CR DI Aff	(11) T3 CR DI Beh	(12) T3 CR DI Cog	(13) T3 PR DI Aff	(14) T3 PR DI Beh	(15) T3 PR DI Cog	(16) T3 CR MQ
(1) T2 AUD	N/A [a]	N/A [a]	N/A [a]	N/A [a]	N/A [a]	N/A [a]	.04
(2) T3 AUD	-.01	.03	.06	-.02	.06	.08	.01
(3) T4 AUD	-.01	.16*	.13	-.02	.04	.03	-.15**
(4) T5 AUD	-.05	.08	.16	-.04	-.03	.07	-.06
(5) T1 AU Freq	-.05	.01	.05	-.03	.08	.12	-.02
(6) T2 AU Freq	.18*	.21**	.08	.08	.20*	.16*	.01
(7) T3 AU Freq	.02	.11	.08	-.02	.09	.06	-.04
(8) T4 AU Freq	-.03	.11	.17*	.10	.13	.10	-.03
(9) T5 AU Freq	-.07	.12	.16	.02	.12	.17*	-.06

Note: T1 = Time 1, T2 = Time 2, T3 = Time 3, T4 = Time 4, T5 = Time 5, AUD = Alcohol Use Disorder, AU Freq = Alcohol Us Frequency, CR = Child Report, PR = Parent Report, DI = Dysregulation Inventory, Aff = Affective, Beh = Behavioral, Cog = Cognitive, MQ = Mood Quality
[a] This correlation could not be calculated, as all participants with data for T2 AUD had the same score.
* $p < .05$, ** $p < .01$, *** $p <$. 001.

Finally, Table 7 presents bivariate correlations among Time 5 ER variables and AU variables. AUD at Times 4 and 5 were significantly correlated with each Time 5 ER variable, with results demonstrating that AUD symptoms are associated with more dysregulation and more negative mood quality. AU frequency at Times 3, 4, and 5 were significantly correlated with the majority of Time 5 DI measures, with results demonstrating that greater AU frequency is associated with more dysregulation.

Table 7.
Bivariate Correlations among Alcohol Use Variables and Time 5 Emotion Regulation Variables

	(10) T5 CR DI Aff	(11) T5 CR DI Beh	(12) T5 CR DI Cog	(13) T5 CR MQ
(1) T2 AUD	.01	.08	-.03	.04
(2) T3 AUD	.07	.04	.09	-.09
(3) T4 AUD	.17**	.22***	.14**	-.21***
(4) T5 AUD	.23***	.27***	.30***	-.16**
(5) T1 AU Freq	.01	-.02	-.03	-.09
(6) T2 AU Freq	.03	.08	.01	.03
(7) T3 AU Freq	.12*	.11*	.14**	-.10
(8) T4 AU Freq	.14**	.19***	.14**	-.09
(9) T5 AU Freq	.09	.16**	.19***	-.04

Note: T1 = Time 1, T2 = Time 2, T3 = Time 3, T4 = Time 4, T5 = Time 5, AUD = Alcohol Use Disorder, AU Freq = Alcohol Use Frequency, ECF = Executive Control Function, CR = Child Report, PR = Parent Report, DI = Dysregulation Inventory, Aff = Affective, Beh = Behavioral, Cog = Cognitive, MQ = Mood Quality
* $p < .05$, ** $p < .01$, *** $p <. 001$.

Aim 1: Examining childhood maltreatment, ECF, and ER at ages 10-12 as predictors of AU frequency and AUD symptoms cross-sectionally and prospectively

Childhood maltreatment. Results of OLS regressions with AUD symptoms at Times 2-5 and AU frequency at Times 1-5 are presented in Table 8. For each analysis, Step 1 included Male (1 = male, 0 = female), High Risk paternal diagnosis (1 = yes, 0 = no), and SES. Step 2 included Emotional Abuse, Physical Abuse, Sexual Abuse, Emotional Neglect, and Physical Neglect. Being male and having a father with either an SUD or psychiatric diagnosis were positively predictive of more AUD symptoms and

higher AU frequency at Time 5. Being male also significantly predicted higher AU frequency at Time 2. Regarding Step 2 findings, Physical Abuse predicted higher AU frequency at Time 4. Sexual Abuse predicted more AUD symptoms at Times 3 and 5, and higher AU frequency at Times 3 and 4. Emotional Neglect predicted higher Time 3 AU frequency, whereas Physical Neglect predicted lower AU frequency at that time point. Experiencing Emotional Abuse was not predictive of any AU outcome.

Table 8.
OLS Regressions for Child Maltreatment Predicting AU Frequency and AUD Symptoms (N = 475)

	AUD Symptoms				AU frequency				
Predictor	Time 2	Time 3	Time 4	Time 5	Time 1	Time 2	Time 3	Time 4	Time 5
	β	β	β	β	β	β	β	β	β
Step 1									
Male	.05	-.01	.07	.15**	.04	.11*	.07	.06	.13*
High Risk	.05	.08	.09	.12*	.05	.10	.04	.06	.14**
SES	-.05	.01	.01	.01	-.02	-.01	-.09	-.03	.01
Step 2									
Male	.04	.01	.08	.16***	.04	.10*	.08	.06	.01**
High Risk	.06	.10*	.06	.10	.05	.09	.05	.04	.13*
SES	-.04	.02	.05	.05	-.02	.01	-.06	.03	.03
Emotional Abuse	-.02	-.08	-.04	-.03	-.04	-.01	-.02	-.07	-.07
Physical Abuse	.02	.02	.02	.02	.03	.04	.07	.17**	-.03
Sexual Abuse	-.01	.13**	.06	.11*	-.01	-.01	.15**	.12*	.08
Emotional Neglect	.12	.10	.05	.05	-.04	-.02	.13*	.06	.10
Physical Neglect	-.10	-.11	.11	.07	-.01	.06	-.14*	.07	.01

Note: For AUD Symptoms, Time 2: Step 1 R^2= .01 (p = .33); Step 2 R^2= .02 (p = .15); Time 3: Step 1 R^2= .01 (p = .41); Step 2 R^2= .03 (p = .06); Time 4: Step 1 R^2= .01 (p = .25); Step 2 R^2= .04 (p = .05); Time 5: Step 1 R^2= .04 (p = .06); Step 2 R^2= .06 (p = .01). For AU Frequency, Time 1: Step 1 R^2= <.01 (p = .47); Step 2 R^2= .01 (p = .33); Time 2:Step 1 R^2= .02 (p = .13); Step 2 R^2= .03 (p = .10); Time 3: Step 1 R^2= .02 (p = .20); Step 2 R^2= .05 (p = .01); Time 4: Step 1 R^2= .01 (p = .33); Step 2 R^2= .07 (p = .01); Time 5: Step 1 R^2= .03 (p = .07); Step 2 R^2= .05 (p = .03). OLS = Ordinary Least Squares, AU = Alcohol Use, AUD = Alcohol Use Disorder, SES = Socioeconomic Status.
* p < .05, ** p < .01, ***p < .001.

Executive control function. Results of OLS regressions with AUD symptoms at Times 2-5 and AU frequency at Times 1-5 are presented in Table 10. For each analysis, Step 1 included Male (1 = male, 0 = female), High Risk paternal diagnosis (1 = yes, 0 = no), and SES. Step 2 included the ECF composite. Findings suggest that higher levels of

ECF predicted fewer AUD symptoms at Time 2, and lower AU frequency at Time 4. ECF did not significantly predict AUD symptoms or AU frequency at any other time points.

Table 9.
OLS Regressions for Executive Control Function Predicting AU Frequency and AUD Symptoms (N = 475)

	AUD Symptoms				AU frequency				
Predictor	Time 2	Time 3	Time 4	Time 5	Time 1	Time 2	Time 3	Time 4	Time 5
	β	β	β	β	β	β	β	β	β
Step 1									
Male	.05	-.01	.07	.15**	.04	.11*	.07	.06	.13*
High Risk	.05	.08	.09	.12*	.05	.10	.04	.06	.14**
SES	-.05	.01	.01	.01	-.02	-.01	-.09	-.03	.01
Step 2									
Male	.05	-.01	.07	.15**	.04	.11*	.07	.06	.13
High Risk	.06	.08	.09	.12*	.04	.11*	.04	.07	.14**
SES	-.07	.02	-.01	.01	<.001	-.03	-.09	-.06	-.02
ECF	-.11*	.03	-.08	-.01	.06	-.09	-.01	-.11*	-.07

Note: For AUD Symptoms, Time 2: Step 1 R^2= .01 (p = .33); Step 2 R^2= .02 (p = .15);Time 3: Step 1 R^2= .01 (p = .41); Step 2 R^2= .01 (p = .38); Time 4: Step 1 R^2= .01 (p = .25); Step 2 R^2= .02 (p = .17); Time 5: Step 1 R^2= .04 (p = .06); Step 2 R^2= .04 (p = .06). For AU Frequency, Time 1: Step 1 R^2= <.01 (p = .47); Step 2 R^2= .01 (p = .33); Time 2: Step 1 R^2= .02 (p = .13); Step 2 R^2= .03 (p = .07); Time 3: Step 1 R^2= .02 (p = .20); Step 2 R^2= .02 (p = .20); Time 4: Step 1 R^2= .01 (p = .33); Step 2 R^2= .02 (p = .14); Time 5: Step 1 R^2= .03 (p = .07); Step 2 R^2= .04 (p = .05). OLS = Ordinary Least Squares, AU = Alcohol Use, AUD = Alcohol Use Disorder, SES = Socioeconomic Status, ECF = Executive Control Function Composite. * p < .05, ** p < .01, ***p < .001.

Emotion regulation. Results of OLS regressions with AUD symptoms at Times 2-5 and AU frequency at Times 1-5 are presented in Table 9. For each analysis, Step 1 included Male (1 = male, 0 = female), High Risk paternal diagnosis (1 = yes, 0 = no), and SES. Step 2 included self and parent reports of emotion regulation, including the Affective, Behavioral, and Cognitive subscales of the DI, and the Mood Quality subscale of the DOT-S. Unexpectedly, there were few significant findings regarding the prediction of AU variables by ER variables. AUD symptoms at all time points were not predicted by any ER variable. Higher levels of AU frequency at Time 1 were predicted by lower levels of self-reported affective dysregulation and higher levels of self-reported behavioral dysregulation. Self-reported behavioral dysregulation also positively predicted AU

frequency at Time 3. Parent-reported behavioral dysregulation positively predicted AU frequency at Time 2. Mood quality, both parent and self reports, did not significantly predict either AUD symptoms or AU frequency.

Table 10.
OLS Regressions for Emotion Regulation Predicting AU Frequency and AUD Symptoms (N = 475)

	AUD Symptoms				AU frequency				
Predictor	Time 2	Time 3	Time 4	Time 5	Time 1	Time 2	Time 3	Time 4	Time 5
	β	β	β	β	β	β	β	β	β
Step 1									
Male	.05	-.01	.07	.15**	.04	.11*	.07	.06	.13*
High Risk	.05	.08	.09	.12*	.05	.10	.04	.06	.14**
SES	-.05	.01	.01	.01	-.02	-.01	-.09	-.03	.01
Step 2									
Male	.04	-.02	.08	.12*	-.06	.03	.04	.04	.09
High Risk	.03	.08	.07	.11*	-.01	.04	.04	.09	.14**
SES	-.03	.01	.02	.02	-.01	-.02	-.09	-.06	-.01
Affective Dysregulation Self-report	.01	-.10	-.25	-.08	-.67*	-.08	-.24	-.23	-.16
Behavioral Dysregulation Self-report	.04	.14	.26	.09	.79*	.10	.38*	.13	.20
Cognitive Dysregulation Self-report	.03	-.02	.11	-.03	-.02	.04	-.03	.02	-.05
Affective Dysregulation Parent-report	-.04	-.02	.16	-.10	-.08	-.16	-.05	-.09	-.18
Behavioral Dysregulation Parent -report	.05	-.09	.08	.01	-.01	.86***	-.13	.03	.15
Cognitive Dysregulation Parent -report	.08	.07	-.12	.09	.20	-.23	.05	-.03	-.11
Mood Quality Self-report	.03	.01	.03	.01	.06	.04	.03	.08	-.01
Mood Quality Parent-report	-.02	.01	.05	-.03	.01	.01	.01	-.04	-.11

Note: For AUD Symptoms, Time 2: Step 1 R^2= .01 (p = .33); Step 2 R^2= .02 (p = .18); Time 3: Step 1 R^2= .01 (p = .41); Step 2 R^2= .02 (p = .30); Time 4: Step 1 R^2= .01 (p = .25); Step 2 R^2= .08 (p = .65); Time 5: Step 1 R^2= .04 (p = .06); Step 2 R^2= .05 (p = .06). For AU Frequency, Time 1: Step 1 R^2= < .01 (p = .47); Step 2 R^2= .82 (p < .001); Time 2: Step 1 R^2= .02 (p = .13); Step 2 R^2= .45 (p < .001); Time 3: Step 1 R^2= .02 (p = .20); Step 2 R^2= .07 (p = .08); Time 4: Step 1 R^2= .01 (p = .33); Step 2 R^2= .05 (p = .08); Time 5: Step 1 R^2= .03 (p = .07); Step 2 R^2= .08 (p < .05). OLS = Ordinary Least Squares, AU = Alcohol Use, AUD = Alcohol Use Disorder, SES = Socioeconomic Status.
* p < .05, ** p < .01, ***p < .001.

Aim 2: Identification of Risk Classes

LCA models were conducted using variables reflecting risk, including four[1] child maltreatment variables (Physical Abuse, Emotional Abuse, Physical Neglect, and Emotional Neglect); Time 1 ECF composite variable; and seven Time 1 ER variables reflecting parent- and self-reports of affective, cognitive, and behavioral regulation, and positive mood quality. The one-class model was fit first, followed by models with two, three, and four classes. Table 11 describes fit information (i.e., log likelihood ratio, AIC, BIC, ABIC, *p* value for the BLRT, entropy, smallest class size) for the LCA models with one through four classes. As previously noted, the BLRT and BIC indices provide the most reliable indicators of the number of classes that best fit the data (Nylund et al., 2007), so these indices primarily were considered in the model building process.

Examination of Table 11 indicates that the lowest BIC, AIC, and ABIC were found for the four-class model; moreover, the BLRT indicated that the four-class model provided an improvement in fit from the three-class model. The four-class model had adequate delineation of classes as indicated by the entropy (.793). Posterior class probabilities ranged from .802 to .987, indicating most youth in the sample (≈ 90%) were placed into a class that reflects their respective levels of severity of risk variables. The smallest class size (4.6%, $n = 22$) was both reasonable in size and conceptually meaningful. Because of the small size of this class in the four-class model, a five-class model was not examined. In sum, multiple fit indices and parameters indicate that the four-class model best fits the data.

[1] When all five child maltreatment variables were included in the LCA analyses, it was not possible for the model to replicate. The Sexual Abuse variable was removed from the model, as it was determined that its positive skew had led to the lack of replication.

Table 11.
Risk Class Model Comparison

Classes	Free parameters	Log likelihood	AIC	BIC	ABIC	BLRT	Entropy	Smallest Class Size *n* (%)
1	26	-12625.95	25303.90	25412.15	25329.63	N/A[a]	1	475 (100.0)
2	40	-12323.58	24727.15	24893.68	24766.73	0.000	0.96	50 (10.5)
3	54	-12170.77	24449.54	24674.36	24502.97	0.000	0.76	43 (9.1)
4	68	-12074.28	24284.57	24567.67	24351.85	0.000	0.79	22 (4.6)

Note. AIC = Akaike Information Criterion, BIC = Bayesian Information Criterion, ABIC=Adjusted BIC, BLRT = Bootstrap Likelihood Ratio Test.
[a]BLRT not available for the one-class model.

Conceptual considerations and the theoretical framework were used to characterize the four-class model. Mean raw scores for the risk variables across the four classes are included in Table 12. Class 1 (labeled the ECF deficits class; *n*=295) was characterized by marked impairment in ECF relative to the other classes and lowest rates of all types of maltreatment. Regarding ER, Class 1 was characterized by the highest levels of positive mood quality, and average levels of affective, behavioral, and cognitive dysregulation relative to the other classes. Individuals in Class 2 (labeled the ER deficits class; *n*=96) were characterized by the highest levels of self-reported affective, behavioral, and cognitive dysregulation relative to the other classes, as well as moderate levels of parent-reported dysregulation and lower levels of positive mood quality. However, Class 2 demonstrated low rates of all types of maltreatment, and higher levels of ECF relative to the other classes. Class 3 (labeled the Neglect class; *n*=62) demonstrated high rates of both physical and emotional neglect relative to Classes 1 and 2. This class also was characterized by the highest levels of parent-reported affective, behavioral, and cognitive dysregulation relative to the other classes. Class 3 demonstrated

lower levels of positive mood quality and average levels of self-reported dysregulation. Regarding ECF, Class 3 demonstrated higher levels of ECF relative to the other classes. Class 4 (labeled the Abuse/Neglect class; *n*=22) was distinct in terms of the high rates of both abuse and neglect, with the highest rates of all types of maltreatment reported by this class. This class also was characterized by lower levels of positive mood quality. However, individuals in Class 4 demonstrated the highest levels of ECF, as well as average levels of self- and parent-reported dysregulation, relative to the other classes.

Table 12.
Risk Scores Across Four-class Model

	Latent Class 1 ECF Deficits (*n*=295)	Latent Class 2 ER Deficits (*n*=96)	Latent Class 3 Neglect (*n*=62)	Latent Class 4 Abuse/Neglect (*n*=22)
	M	*M*	*M*	*M*
Maltreatment				
Emotional Abuse	5.89	6.37	8.93	16.67
Physical Abuse	5.74	6.17	7.07	9.94
Emotional Neglect	6.36	7.79	13.71	14.45
Physical Neglect	5.53	6.08	8.57	9.11
Self-reported Dysregulation				
Affect	19.12	43.14	25.34	24.47
Behavioral	20.91	46.99	26.69	25.51
Cognitive	28.37	38.77	29.24	26.31
Parent-reported Dysregulation				
Affective	19.40	23.14	27.09	14.87
Behavioral	24.76	29.64	33.48	17.65
Cognitive	32.80	36.94	40.90	30.43
Positive Mood Quality				
Self-report	24.49	22.01	22.49	21.93
Parent-report	25.80	24.56	24.47	25.08
ECF Composite	-0.25	0.43	0.31	0.44

Note. ECF = Executive Control Function; ER = Emotion Regulation.

Sex, Parental Psychiatric Status, and SES Analyses. Results for differences among classes in terms of categorical covariates (sex, parental psychiatric status) are presented in Table 13 and for dimensional covariates (SES, AU variables) are presented in Table 14. In terms of demographic differences, auxiliary tests of equality of means for categorical variables (Table 13) suggested that, compared to youth in the Abuse/Neglect

class, youth in the ECF Deficits class were less likely to have a paternal history of substance use disorder or other psychiatric disorder. The Abuse/Neglect class did not differ in terms of the proportion of boys and girls from the other three classes (Table 13). Omnibus between-group analyses revealed that classes differed significantly in SES (Table 14). Follow-up pairwise comparisons revealed that Class 1 (ECF deficits) had significantly higher SES than Class 2 [ER deficits; $\chi2(1)= 7.80$, $p=.01$], Class 3 [Neglect; $\chi2(1)=19.44$, $p<.01$], and Class 4 [Abuse/neglect; $\chi2(1)= 15.94$, $p<.01$].

Table 13.
Log Odds Coefficients for Four-Class AU Risk Model at Ages 10-12 Using the Abuse/Neglect Class as the Comparison Group

Class	Effect	Logit	*SE*
ER Deficits	Male	.48	.53
	Parental Dx	-.51	.52
ECF Deficits	Male	-.07	.48
	Parental Dx	-1.09*	.48
Neglect	Male	.07	.55
	Parental Dx	.09	.56

Note. AU = Alcohol Use, ER = Emotion Regulation, ECF = Executive Control Function, Male = sex (female = 0, male = 1), Paternal Dx = paternal history of substance use disorder or other psychiatric disorder (no history = 0, positive history =1).
* $p < .05$.

Aim 3: Examination of AU frequency and AUD symptoms among the latent classes

As with the demographic factors, auxiliary analyses also were conducted to compare latent classes on AU variables. Omnibus between-group analyses comparing AU frequency and AUD symptoms both concurrently (Time 1) and longitudinally (Times 2, 3, 4, and 5) did not show significant differences among the four classes (Table 14).

Table 14.
AU Frequency and AUD Symptoms Across Four-class Model

	Latent Class 1 ECF Deficits (*n*=292)	Latent Class 2 ER Deficits (*n*=98)	Latent Class 3 Neglect (*n*=63)	Latent Class 4 Abuse/Neglect (*n*=22)	Omnibus chi-square test		Pairwise Comparisons
	M	*M*	*M*	*M*	*χ2*	*P*	
SES	44.21	38.91	35.84	34.50	37.61	<.01	1 > (2, 3, 4)
AU Frequency							
Time 1	.19	.19	.09	.04	1.19	.76	-
Time 2	1.37	1.86	2.16	.77	1.08	.78	-
Time 3	10.85	16.77	16.27	25.92	1.78	.61	-
Time 4	45.27	39.96	55.25	77.82	2.50	.48	-
Time 5	93.74	92.06	98.47	83.38	.12	.99	-
AUD Symptoms							
Time 2	.01	.05	.04	<.001	1.90	.59	-
Time 3	.15	.22	.20	<.001	5.98	.11	-
Time 4	.53	.63	.80	.91	2.43	.49	-
Time 5	1.11	1.11	1.29	1.55	.94	.82	-

Note. AUD Symptoms at Time 1 could not be analyzed as no participants met criteria for any AUD.

Aim 4: Evaluation of transactional-ecological model linking childhood maltreatment to longitudinal AU through ECF and ER

Structural equation modeling (SEM) was used to examine pathways from childhood maltreatment to concurrent and prospective AU frequency and AUD symptoms, and to examine the roles of ECF and ER in predicting those AU variables. Separate SEM models were examined for the two AU outcome variables.

Measurement models. Separate measurement models were first examined to assess goodness of fit for the following latent variables: Childhood Maltreatment, ER at Time 1, ER at Time 3, and ER at Time 5.

Childhood Maltreatment. The standardized coefficients (βs) were examined to determine the ability of the measurement model to explain the five subscales of the CTQ. As Sexual Abuse did not significantly load on the latent variable, that variable was

removed from the measurement model. The final measurement model for Childhood Maltreatment was an adequate fit to the data, χ^2 (df = 2) = 38.28, $p < 0.001$; CFI = 0.93; RMSEA = 0.20 (90% C.I. = .14 - .25); SRMR = .05. The latent variable was significantly represented by all of its four indicators (all at $p<.01$), with standardized coefficients (βs) of .48 for Physical Abuse, .70 for Emotional Abuse, .65 for Physical Neglect, and .82 for Emotional Neglect. Higher scores on this construct indicate greater levels of childhood maltreatment.

ER at Time 1. The standardized coefficients (βs) were examined to determine the ability of the measurement model to explain the Time 1 parent- and self-reports of the Affective, Behavioral, and Cognitive subscales of the DI, and the DOT-S mood quality subscale. As a latent variable that included parent reports of these measures indicated inadequate indices of fit, the model was modified and parent-report variables were removed. The final measurement model for ER at Time 1 was a very good fit to the data, χ^2 (df = 2) = .86, $p = 0.70$; CFI = 1.00; RMSEA = 0.00 (90% C.I. = .00 - .07); SRMR = .01. The latent variable was significantly represented by each of the four indicators (all at $p<.01$), with standardized coefficients (βs) of -.86 for Affective Regulation, -.98 for Behavioral Regulation, -.60 for Cognitive Regulation, and .26 for Positive Mood Quality. Higher scores on this construct indicate greater levels of emotion regulation.

ER at Time 3. The standardized coefficients (βs) were examined to determine the ability of the measurement model to explain the Time 3 parent- and self-reports of the Affective, Behavioral, and Cognitive subscales of the DI, and self-report of the DOT-S mood quality subscale. As a latent variable that included parent reports of these measures indicated inadequate indices of fit, the model was modified and parent-reported variables

were removed. The final measurement model for ER at Time 3 was a good fit to the data, χ^2 (df = 2) = 6.78, p = 0.03; CFI = 0.98; RMSEA = 0.08 (90% C.I. = .02 - .14); SRMR = .03. The latent variable was significantly represented by each of its four indicators (all at p<.01), with standardized coefficients (βs) of -.79 for Affective Regulation, -.90 for Behavioral Regulation, -.60 for Cognitive Regulation, and .41 for Positive Mood Quality. Higher scores on this construct indicate greater levels of emotion regulation.

ER at Time 5. The standardized coefficients (βs) were examined to determine the ability of the measurement model to explain the Time 5 self report of the Affective, Behavioral, and Cognitive subscales of the DI, and the DOT-S mood quality subscale. Parent reports of these measures were not collected at this time point. The measurement model for ER at Time 5 was a good fit to the data, χ^2 (df = 2) = 33.22, p < .001; CFI = 0.95; RMSEA = 0.21 (90% C.I. = .15 - .27), SRMR = .04. The latent variable was significantly represented by each of its four indicators (all at p<.01), with standardized coefficients (βs) of -.88 for Affective Regulation, -.90 for Behavioral Regulation, -.74 for Cognitive Regulation, and .37 for Positive Mood Quality. Higher scores on this construct indicate greater levels of emotion regulation.

Structural equation model predicting AU frequency. Following the creation of latent variables, pathways were added to test the current study's questions. The Childhood Maltreatment latent variable was hypothesized to predict ER at all three time points, ECF at Time 1, and AU frequency at Times 1, 2, 3, 4, and 5. First, fit was examined and modifications were made as needed based on suggestions from the modification indices. Based on these modification indices, ER at Time 3 was not included in the final model (Figure 4).

The final AU frequency model was a moderate fit to the data, χ^2 (df = 117) = 282.71, $p < 0.001$; CFI = 0.87; RMSEA = 0.07 (90% C.I. = .05 - .07); SRMR = .13. The standardized coefficients (βs) were examined to determine the ability of the model to predict the endogenous variables. The findings indicated that childhood maltreatment positively predicted ECF at Time 1, as well as AU frequency at Time 4. Childhood maltreatment also negatively predicted ER at Time 1. ER at Time 1 positively predicted ER at Time 5, as well as negatively predicted AU frequency at Time 3. ECF at Time 1 negatively predicted AU frequency at Time 4. AU frequency at Time 3 positively predicted AU frequency at Time 4, and AU frequency at Time 4 positively predicted AU frequency at Time 5.

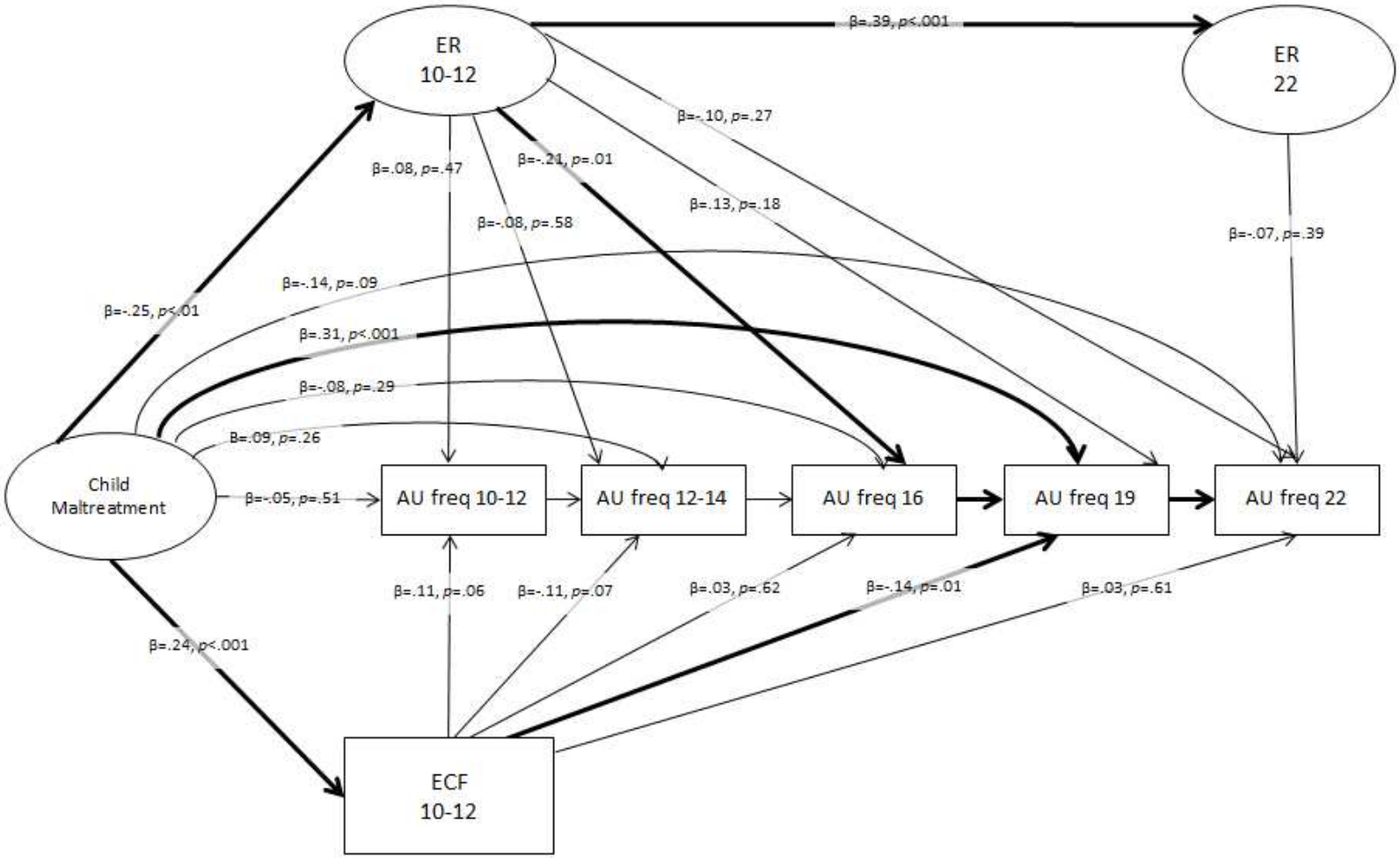

Figure 4. Structural equation model predicting alcohol use frequency at five time points. Childhood maltreatment, executive control function at ages 10-12, and emotion regulation at ages 10-12 predicted prospective alcohol use frequency.
Note: AU freq = alcohol use frequency; ER = emotion regulation; ECF = executive control function.

Structural equation model predicting AUD symptoms. Similarly, pathways among latent variables were added to evaluate AUD symptoms. As with the AU frequency analyses described above, the Childhood Maltreatment latent variable was hypothesized to predict ER at all three time points, ECF at Time 1, and the AUD symptoms outcome variable at Times 2, 3, 4, and 5. First, fit was examined and modifications were made based on suggestions from the modification indices. Based on the modification indices suggesting that model fit would be improved by omitting ER at Time 3, this latent variable was not included in the final model. The final model included an additional path from Childhood Maltreatment to ER at Time 5, as modification indices suggested that this addition would improve model fit (Figure 5).

The final AUD symptoms model was a moderate fit to the data, χ^2 (df = 103) = 202.92, $p < 0.001$; CFI = 0.92; RMSEA = 0.06 (90% C.I. = .04 - .07); SRMR = .08. The standardized coefficients (βs) were examined to determine the ability of the model to predict the endogenous variables. The findings indicated that childhood maltreatment positively predicted ECF at Time 1, as well as AUD symptoms at Time 4. Childhood maltreatment also negatively predicted ER at Time 5. ER at Time 1 and ECF at Time 1 negatively predicted AUD symptoms at Time 4. ER at Time 1 positively predicted ER at Time 5, and ER at Time 5 negatively predicted AUD symptoms at Time 5. AUD symptoms at Time 2 positively predicted AUD symptoms at Time 3, and AUD symptoms at Time 4 positively predicted AUD symptoms at Time 5.

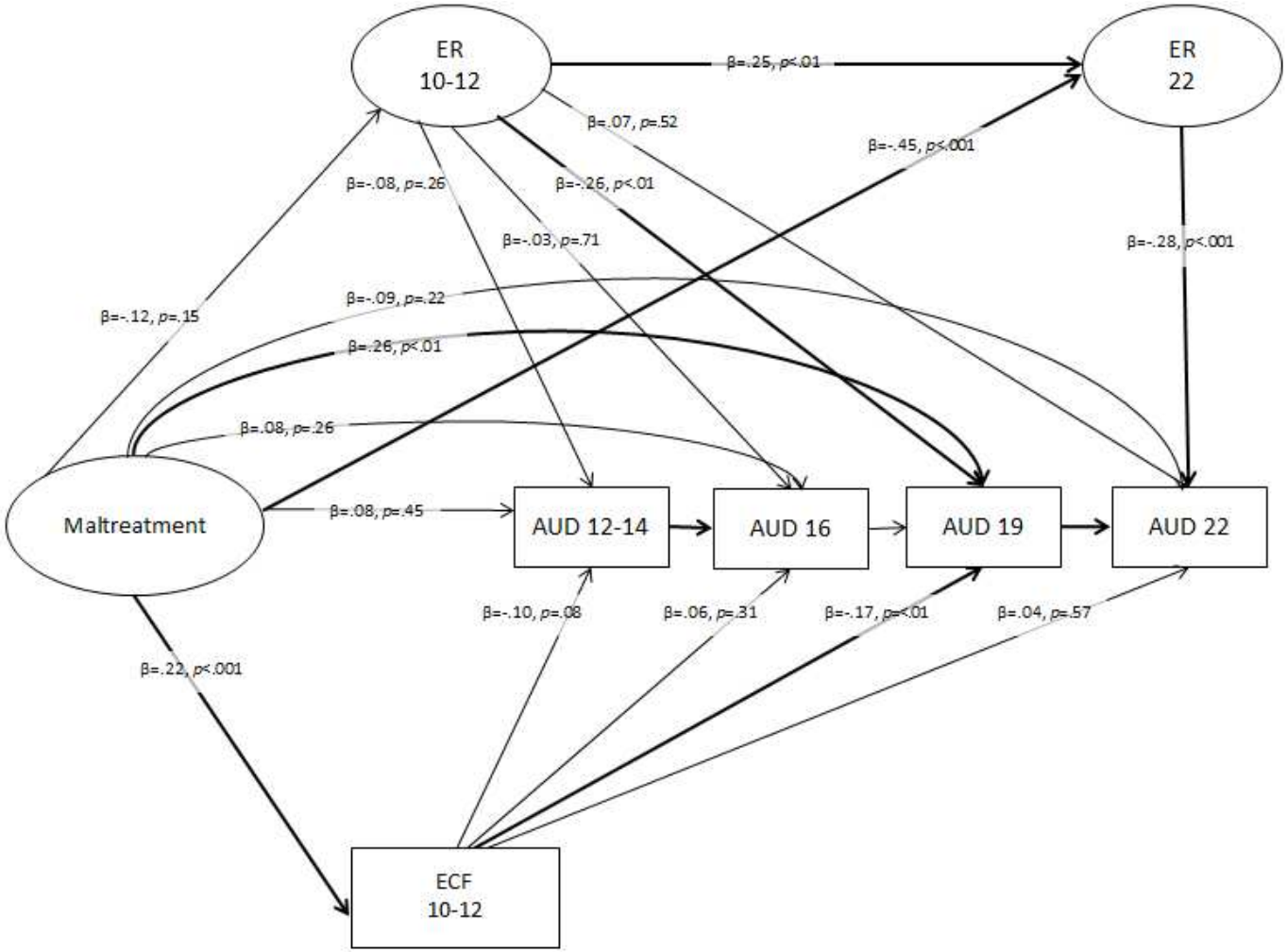

Figure 5. Structural equation model predicting alcohol use disorder symptoms at four time points. Child maltreatment, executive control function at ages 10-12, and emotion regulation at age 22 predicted prospective alcohol use disorder symptoms.

Note: AUD = alcohol use disorder symptoms; ER = emotion regulation; ECF = executive control function.

Comparing structural equation models for AU frequency and AUD symptoms. Overall, results of the SEM analyses were highly similar between AU frequency and AUD symptoms models. In both models, childhood maltreatment positively predicted ECF at Time 1 and the Time 4 AU variable. Childhood maltreatment negatively predicted ER in both models, though it predicted Time 1 ER in the AU frequency model and Time 5 ER in the AUD symptoms model. ECF at Time 1 predicted the Time 4 AU variable in both models. Time 1 ER was predictive of prospective AU in both models. However, in the AUD symptoms model, Time 5 ER was also predictive of concurrent AUD symptoms. Finally, both models provided evidence of longitudinal continuity regarding ER and AU, as Time 1 ER predicted Time 5 ER and several significant pathways from earlier AU variables to AU at later time points were found in both models.

In general, findings of these analyses highlighted the inter-relations among childhood maltreatment, ECF, and ER in predicting longitudinal AUD symptoms and AU frequency. In particular, results indicated that there are significant pathways from maltreatment to both ECF and ER. Subsequently, there are pathways from ECF and ER to problematic AU in both models. Additionally, there are prospective pathways from childhood maltreatment to problematic AU in both models. In summary, this evaluation of a transactional-ecological model confirmed the existence of a prospective pathway from childhood maltreatment to AU and indicated that ECF and ER function to increase the likelihood of problematic AU, even when considered concurrently with maltreatment.

CHAPTER 4
DISCUSSION

Although experiencing maltreatment during childhood has been implicated in the development of problematic AU and AUD, few studies have investigated processes that affect how this association develops. In addition, no prior research exists, to my knowledge, examining the roles of ECF and ER concurrently with childhood maltreatment, although both constructs are potential sequelae of maltreatment and associated with risk for problematic AU. Lastly, there is a gap in the literature examining how ECF and ER may influence (e.g., exacerbate, buffer, mediate) the association between childhood maltreatment and problematic AU and AUD. Specifically, there is a dearth of research considering whether ECF and ER serve as risk or protective factors in the relation between maltreatment and AU and AUD. The present study addressed these gaps in the literature through (a) investigation of childhood maltreatment, ECF, and ER as risk factors for problematic AU and AUD; (b) exploration of differences in quality and type of these individual risk factors using person-centered analyses; and (c) examination of concurrent and prospective relations among patterns of these risk factors and AU across development using variable-centered analyses.

Results indicated that (a) childhood maltreatment, ECF deficits, and ER deficits concurrently and prospectively predicted higher levels of AUD symptoms and a greater frequency of AU; (b) youth placed in classes that were distinguished by experiencing abuse and neglect, experiencing neglect only, having ECF deficits, or having ER deficits did not differ on both concurrent and prospective AUD symptoms or AU frequency; (c) experiencing childhood maltreatment was associated with higher rates of ECF and ER

deficits; (d) child maltreatment, even when examined in conjunction with ECF and ER, placed individuals at a greater risk for problematic AU; and (e) ECF and ER deficits, even when considered concurrently with childhood maltreatment, placed individuals at greater risk for problematic AU. In sum, the current project was the first to examine childhood maltreatment, ECF, and ER concurrently; investigate subgroups differing in terms of childhood maltreatment, ECF, and ER and their relation to problematic AU; and explore how the association between childhood maltreatment and problematic AU are affected by ECF and ER. Findings clarify and extend previous research regarding the roles of childhood maltreatment, ECF, and ER on developmental pathways to AUD symptoms and AU frequency from middle childhood through early adulthood.

Aim 1: Examining childhood maltreatment, ECF, and ER as predictors of AU frequency and AUD symptoms

Experiences of childhood maltreatment, ECF at ages 10-12, and ER at ages 10-12 were investigated as predictors of AU frequency and AUD symptoms both cross-sectionally and prospectively. Prior research has indicated that higher rates of childhood maltreatment, ECF deficits, and ER deficits are associated with higher levels of AU frequency and AUD symptoms (e.g., Giancola & Moss, 1998; Lown et al., 2011; Simons et al., 2004). Thus, I hypothesized that the current study would replicate these prior findings.

Regarding childhood maltreatment, consistent with my hypotheses, reportedly experiencing (a) sexual abuse predicted higher levels of AUD symptoms at ages 16 and 22, (b) sexual abuse or emotional neglect predicted a greater frequency of AU at age 16, and (c) physical or sexual abuse predicted a greater frequency of AU at age 19. However,

in contrast to prior research and my hypothesis, experiencing physical neglect predicted a lower frequency of AU at age 16. Additionally, physical or emotional abuse did not predict AU frequency at any time point, and physical and emotional abuse, and physical and emotional neglect, did not predict AUD symptoms at any time point.

Regarding ECF, consistent with my hypotheses, higher levels of ECF at ages 10-12 predicted (a) lower levels of AUD symptoms at ages 12-14, and (b) lower levels of AU frequency at age 19. ECF at ages 10-12 did not predict AUD symptoms or AU frequency at any other time point assessed.

Regarding ER, consistent with my hypotheses, higher levels of AU frequency were predicted (a) at ages 10-12 by higher levels of concurrent, self-reported behavioral dysregulation; (b) at ages 12-14 by higher levels of parent-reported behavioral dysregulation, reported at ages 10-12; and (c) at age 16 by higher levels of self-reported behavioral dysregulation, reported at ages 10-12. However, in contrast to prior research and my hypothesis, higher levels of self-reported affective dysregulation at ages 10-12 predicted a lower frequency of concurrent AU. No measures of ER at ages 10-12 predicted AUD symptoms. Additionally, self-reported cognitive dysregulation and mood quality, as well as parent-reported affective and cognitive dysregulation and mood quality, did not predict AU frequency at any time point.

In summary, AUD symptoms and AU frequency were predicted by some aspects of childhood maltreatment, ECF at ages 10-12, and ER at ages 10-12. However, results of the present analyses indicate that, contrary to hypotheses, these associations do not appear to be as robust as in prior research (e.g., Deckel & Hesselbrock, 1996; Herrenkohl et al., 2013; Mohr et al., 2008). Several types of childhood maltreatment and the majority

of measures of ER did not predict either problematic AU outcome. Additionally, ECF was only predictive of problematic AU or AUD outcomes at two time points. The development of problematic AU or AUD is a complex, multivariate process; thus, this lack of findings may have stemmed from my consideration of single areas (e.g., ER or ECF independent of the other construct), which cannot account for interactions or other inter-relations among risk variables and/or the exacerbation of risk by experiencing multiple risk factors. As such, other approaches that can capture this complexity (e.g., examining profiles of individuals) may be necessary for accounting for AU outcomes. Finally, although significant associations between AU frequency and AUD symptoms with childhood maltreatment, ECF, and ER were not have as plentiful as hypothesized, findings do indicate that, at various ages, these constructs likely enhance vulnerability for engaging in problematic AU.

Aim 2: Identification of Risk Classes

Use of a person-centered approach to identify classes or subtypes of childhood maltreatment, ECF, and ER allowed for delineation of qualitatively distinct risk groups. I hypothesized that I would identify four groups of youth based on their history of childhood maltreatment, ECF, and ER. Consistent with hypotheses, four subtypes of risk groups that vary in type and severity of risk factors were identified. However, these groups of youth exhibited different patterns and levels of childhood maltreatment, ECF, and ER than expected.

Patterns of risk subtypes suggest that risks that have been empirically demonstrated to enhance vulnerability for AU or AUD were also found in the current sample. Rather than identifying subgroups of youth that exhibited high levels of multiple

risk factors as expected, the four classes each exhibited high levels of a distinct risk factor, with other risk factors at relatively low or average levels. Specifically, the following risk groups were identified: (1) history of both childhood abuse and neglect, (2) history of childhood neglect only, (3) ECF deficits, and (4) ER deficits. In contrast to my hypotheses, there was not a group of youth that exhibited high levels of childhood maltreatment and low levels of both ECF and ER. An additional unexpected finding was that, rather than the hypothesized groups of youth with low levels of childhood maltreatment and low levels of ECF and ER, groups of youth were identified that exhibited low levels of maltreatment and either low levels of ECF or low levels of ER.

Finally, results of the LCA analysis did not identify any groups of youth that exhibited protective levels of all assessed constructs (e.g., average or high levels of both ECF and ER, low levels of childhood abuse and neglect). Each group of youth identified exhibited high levels of a distinct risk factor, which was an unexpected finding. Thus, the hypothesis that LCA results would identify groups of youth at lowered risk for AU and AUD, specifically children with low levels of maltreatment and high levels of both ECF and ER, was unconfirmed.

Aim 3: Examination of AU frequency and AUD symptoms among the latent classes

Following the identification of latent risk classes, omnibus between-group analyses comparing AU frequency and AUD symptoms among the classes were conducted. I hypothesized that a multiple risk group (with high rates of maltreatment and low levels of ECF/ER) would have higher levels of AU and AUD at ages 10-12. This hypothesis was unconfirmed, as (a) all study participants endorsed having zero symptoms

of AUD at ages 10-12 across study participants and (b) there were no significant differences across groups in levels of AU frequency at that time point.

Regarding prospective prediction of AU and AUD, I hypothesized that there would be significant differences across groups, such that individuals with low levels of ECF/ER and high or low levels of maltreatment would have higher levels of AU and AUD, and individuals with high levels of ECF/ER would have lower levels of AU and AUD. Not surprisingly, given thatdifferent classes than those hypothesized were found, there were no significant differences among groups regarding prospective levels of problematic AU.

It is likely that no differences were found between classes regarding both cross-sectional and longitudinal levels of AU frequency and AUD symptoms because of the high rates of a specific risk factor for each of the four groups; consequently, classes were characterized more by qualitative (e.g., type of risk) than quantitative (e.g., frequency) differences. These results nevertheless support that equifinality operates in the development of problematic AU, as varied starting points (e.g., membership in classes characterized by different risk factors) were associated with similar AU levels over time, rather than all individuals in a specific class following a single progression to an outcome (Cicchetti & Rogosch, 1996, 2002).

Aim 4: Evaluation of transactional-ecological model linking childhood maltreatment to longitudinal AU through ECF and ER

Structural equation modeling was utilized to evaluate a transactional-ecological model linking childhood maltreatment to longitudinal AU frequency and AUD symptoms through ECF and ER. Specifically, analyses investigated how ER and ECF might

function as risk and/or protective processes to influence pathways to AU and AUD (Cicchetti & Rogosch, 2002).

A structural equation model predicting AU frequency was investigated. As hypothesized, there was longitudinal continuity in ER, as ER at ages 10-12 predicted ER at age 22. Regarding longitudinal continuity for AU frequency, AU frequency at (a) age 16 predicted AU frequency at age 19, and (b) at age 19 predicted AU frequency at age 22. Consistent with hypotheses, child maltreatment predicted both ECF and ER. However, though maltreatment negatively predicted ER at ages 10-12, higher rates of maltreatment were associated with higher levels of ECF, which was unexpected based on prior research (e.g., Pechtel & Pizzagalli, 2011). Childhood maltreatment positively predicted AU frequency at age 19, which was consistent with hypotheses and previous research (e.g., Young-Wolff et al., 2011). AU frequency was negatively predicted at age 16 by ER at ages 10-12 and at age 19 by ECF at ages 10-12. However, maltreatment, ER at ages 10-12 and age 22, and ECF at ages 10-12 did not predict AU frequency at any other time points. Finally, I hypothesized that there would be pathways from maltreatment to AU through ECF or ER. This hypothesis was confirmed as maltreatment predicted both ECF and ER, which, in turn, predicted AU frequency.

A structural equation model predicting AUD symptoms also was investigated. As in the previous model, there was longitudinal continuity in ER, as ER at ages 10-12 predicted ER at age 22. Regarding longitudinal continuity for AUD symptoms, AUD symptoms (a) at ages 12-14 predicted AUD symptoms at age 16, and (b) at age 19 predicted age 22 AUD symptoms. Consistent with hypotheses and similar to the AU model, childhood maltreatment predicted both ECF and ER. However, directionality of

these relations was similar to the AU model, with maltreatment negatively predicting ER at age 22 and positively predicting ECF at ages 10-12. Maltreatment did not significantly predict ER at ages 10-12. As in the AU model, maltreatment positively predicted AUD symptoms at age 19. AUD symptoms were negatively predicted at age 19 by ER at ages 10-12 and by ECF at ages 10-12. AUD symptoms at age 22 were negatively predicted by ER at age 22, which is in contrast to the AU model where ER at age 22 did not predict the AU frequency. However, maltreatment, ER at ages 10-12, and ECF at ages 10-12 did not predict AUD at any other time points. Finally, as in the prior model, I hypothesized that there would be pathways from maltreatment to AUD symptoms through ECF or ER. This hypothesis was confirmed as maltreatment predicted both ECF and ER, which, in turn, predicted AUD symptoms.

In summary, there were many similarities between the AU and AUD models. Maltreatment predicted ER negatively and ECF positively in both models, though ER was predicted at different time points between the two models. In both models, child maltreatment was predictive of the age 19 AU or AUD outcome. At age 16, ER at ages 10-12 predicted AU frequency, but AUD at that age was not predicted by either ECF or ER. In both models, ECF at ages 10-12 negatively predicted the age 19 AU outcome. However, the AUD model had additional significant pathways between ER and AUD symptoms at ages 19 and 22. Finally, both models indicated pathways from maltreatment to the AU or AUD outcomes through ECF or ER.

Results of the present analyses indicate quite clearly the importance of considering ECF and ER in conjunction with childhood maltreatment to better understand pathways to enhanced risk for both AUD and higher levels of AU. Childhood

maltreatment, as well as ECF and ER deficits, jointly serve as risk factors for AU and AUD. Even when accounting for ECF and ER through their inclusion in the structural equation models, childhood maltreatment was predictive of both more frequent AU and higher levels of AUD symptoms in early adulthood. Furthermore, when accounting for experiences of childhood maltreatment, ER and ECF deficits during middle childhood were associated with more symptoms of AUD in early adulthood, and ER deficits also were predictive of higher AU during adolescence. Thus, results suggest that having intact or higher levels of ECF or ER may protect against problematic AU among individuals with a history of childhood maltreatment. Results of the present analyses also provide evidence that ER and ECF can promote resilience, as higher levels of ER and ECF were associated with lower levels of AU and AUD. Conversely, having ECF or ER deficits may function to enhance risk for problematic AU among these individuals, which is consistent with prior research (e.g., as reviewed by Brown et al., 2009; Klanecky & McChargue, 2013; Sher & Grekin, 2007) suggesting that ECF and ER deficits are risk factors for AU.

Findings also provided support for the conceptualization developed by prior researchers that development among individuals who experience childhood maltreatment functions as a transactional-ecological model, whereby maltreatment generates risk factors that function more proximally to increase the likelihood of AU (e.g., Cicchetti & Lynch, 1993; Cicchetti & Toth, 2005; Cicchetti & Valentino, 2006). Specifically, experiencing childhood maltreatment was associated with more emotion dysregulation, which was then associated with higher levels of AUD or AU. Within the developmental psychopathology perspective of examining risk and resilience (e.g., Drabick & Steinberg,

2011), more emotion dysregulation may function to exacerbate risk for AUD or AU, whereas greater ER may confer resilience or buffer risk for AUD or AU.

The current study expands upon prior work that utilized a dynamic cascades framework to examine linkages between child maltreatment and SU (e.g., Oshri et al., 2011; Rogosch et al., 2010) by utilizing this framework to examine processes by which maltreatment confers risk or influences other factors linked more proximally to the development of AU or AUD specifically. The current findings also build upon the preponderance of evidence indicating childhood maltreatment (e.g., Lown et al., 2011), ER deficits (e.g., Klanecky & McChargue, 2013), and ECF deficits (e.g., Stavro, Pelletier, & Potvin, 2013) enhance vulnerability for AU and the development of AUD. Additionally, examining AU and AUD as outcomes allows for consideration of both dimensional and categorical approaches to understanding AU behaviors and thus can speak to generalizability of the findings based on the assessment approaches used. Using a dimensional approach (e.g., AU frequency) provides more information regarding severity and the emergence and diminishment of AU (Beauchaine, 2003), whereas using a categorical approach (e.g., AUD symptoms, based on the *DSM-IV*) allows for making determinations regarding the clinical significance of AU problems. Interestingly, although results indicated that the four classes of youth identified in LCA analyses are distinct in having high levels of a specific risk factor, rather than experiencing multiple risks for problematic AU, results of these variable-centered SEM analyses demonstrate that experiencing maltreatment is associated with having ER deficits. In the person-centered, LCA analyses, connections among multiple risk factors may have been minimized in the pursuit of homogeneous classes. Thus, it is possible that the variable-

centered approach better delineated how specific factors, independently and in combination with one another, function to enhance vulnerability for engaging in problematic AU.

Strengths, Limitations, and Future Directions

Among the strengths of this study were the racial and ethnic diversity of the sample, as well as the heterogeneity among study participants regarding paternal psychiatric background. These features suggest that the findings hold true among children from families with varying levels of risk due to paternal psychiatric status. An additional strength of this study was the longitudinal data spanning from middle childhood to early adulthood, and the use of repeated AU measures at each time point. The prospective nature of the sample afforded examination of the roles of child maltreatment, ECF, and ER in risk processes leading to problematic AU across development. Use of both person- and variable-centered approaches enabled risk processes for AUD to be examined through both identification of classes that differed in terms of their risk profiles and investigation of developmental pathways to problematic AU. Finally, the use of a transactional-ecological model enabled investigation of risk processes involving maltreatment that might in turn generate risk factors that function more proximally to increase the likelihood of AU (Rogosch et al., 2010). Examining these processes of risk and resilience, as well as continuity and discontinuity across development, are central components of a developmental psychopathology perspective (e.g., Cicchetti & Rogosch, 1996, 2002; Drabick & Steinberg, 2011).

Despite these strengths, there are several factors that may limit the generalizability of the findings. There was a preponderance of boys within the sample, so

findings may not generalize to girls. As being male predicted higher AU frequency and more AU symptoms at age 22, future research should investigate the generalizability of the current models to females by conducting multiple group structural equation modeling analyses with males and females. Additionally, the current study is limited by the available index of childhood maltreatment. It should be noted that childhood maltreatment was measured by a frequently used retrospective self-report and thus results could differ when predicting maltreatment as measured by official records, observational methods, and/or clinical interview either concurrently or prospectively, which could mitigate concerns related to self report and retrospective biases. Future research might address these issues by utilizing a combination of well-validated self-reported measures, official records, and observational methods to index childhood maltreatment. Relatedly, there was a lack of data regarding timing of maltreatment during childhood. Given findings in previous research that timing of maltreatment affects its sequelae (e.g., Anderson et al., 2008; Schoedl et al., 2010), it is probable that maltreatment timing led to different sequelae for participants in the current study, but the methodology precluded examination of these potential differences. As past research has found that experiencing maltreatment generates risk for global deficits in cognitive function and ECF (e.g., van der Kolk, 2003) and ER difficulties (e.g., Tottenham et al., 2010), future research should examine how timing of maltreatment reciprocally influences the development of impaired ECF and ER and then, in turn, problematic AU. Similarly, it was only possible to examine ECF and ER beginning at ages 10-12 in the current study. Given prior findings that ECF and ER development are well underway by middle childhood (as reviewed in Anderson, 2002; Gross & Thompson, 2007), nuances regarding the developmental course

and timing of ECF and ER development, as well as interactions among these processes, were not captured in the current study. Future research should be expanded to include assessments of ECF and ER in early childhood.

Of course, childhood maltreatment, ECF, and ER are only three of many potential predictors of problematic AU. Future research should examine whether and to what extent other individual or contextual factors strengthen or attenuate these relations. Specifically, parenting, neighborhood factors, and peer relations may influence (e.g., buffer, exacerbate) the relations among maltreatment, ECF, and ER. Additionally, future research should consider individuals' psychological responses to childhood maltreatment within models examining ECF and ER. In particular, examining the role of developing post-traumatic stress disorder (PTSD) and related sequelae following experiences of maltreatment in future research may improve our understanding of risk processes linking childhood maltreatment to later problematic AU. Indeed, given prior research finding ECF (Beers & De Bellis, 2002) and ER (Tull, Barrett, McMillan, & Roemer, 2007) deficits among individuals with PTSD, experiencing maltreatment and, as a result, meeting criteria for PTSD may predict a higher likelihood of ECF and ER deficits, which would then enhance risk for problematic AU among individuals with a PTSD diagnosis.

Moreover, future research should examine whether and to what extent the current models of relations among childhood maltreatment, ECF, and ER with AU frequency and AUD can be applied to other SUDs (e.g., marijuana, tobacco, polysubstance use), as the heterogeneity observed in diverse illicit drugs and alcohol regarding onset, escalation, and decline during adolescence and adulthood (e.g., Schulenberg & Maslowsky, 2009) may result in different findings. Similarly, future research should investigate the roles of

ECF and ER as risk or protective factors in the development of other psychiatric difficulties following childhood maltreatment, as maltreatment has been linked to a broad range of psychological symptoms and diagnoses (Andrews et al., 2004; Cicchetti & Toth, 2005; Norman et al., 2012). This research should include investigations of resilience and predictors of positive outcomes following maltreatment and thus provide a more extensive test of multifinality than conducted in the present study that focused on AU behaviors. Future research that considers these issues will advance our understanding of the documented connection between maltreatment during childhood and later development of AUD, as well as alternative outcomes and roles for ECF and ER in understanding sequelae of maltreatment. Findings of these investigations would provide an important framework for etiological and intervention models that could potentially reduce the costs and sequelae associated with childhood maltreatment as well.

Conclusions and Clinical Implications

Several main findings emerged from this study. First, childhood maltreatment, ER deficits, and ECF deficits were independently associated with higher rates of AU and AUD symptoms. Second, comparisons of groups of youth with high levels of a particular risk factor (e.g., abuse/neglect, neglect only, ECF deficits, ER deficits) did not differ on levels of concurrent or prospective AU frequency or AUD symptoms. Third, childhood maltreatment predicted both a higher frequency of AU and a greater number of AUD symptoms in adolescence and early adulthood, and ECF and ER deficits enhanced this vulnerability for prospective problematic AU. Taken together, results highlight the importance of examining risk and protective factors, in particular ECF and ER, for problematic AU among children and adolescents with a history of childhood

maltreatment. These findings can inform prevention and intervention efforts for youth with a history of maltreatment by improving identification of youth at enhanced risk for problematic AU and refining treatment selection.

Assessment, case conceptualization, and treatment selection and implementation should account for not only experiences of childhood maltreatment, but also ECF and ER processes that may influence the development of AU behaviors. These areas may provide additional opportunities for intervention as ECF and ER may affect the development and maintenance of AU among youth with a history of maltreatment. Assessing ECF and ER processes and skills concurrently with childhood maltreatment history will enable youth with deficits in these areas, with and without a history of maltreatment, to be identified for targeted prevention and intervention efforts. ECF and ER also should be incorporated in case conceptualization. For example, deficits in these areas could ameliorate, maintain, or exacerbate psychological difficulties both related and unrelated to childhood maltreatment. Finally, children with a history of maltreatment and concurrent ECR or ER deficits should be engaged in preventive efforts prior to adolescence, when the onset of AU is more likely, so as to mitigate the likelihood of these youth developing problematic AU.

Future research is needed to determine whether interventions for youth with a history of maltreatment can be selected and/or tailored based on the youth's ECF and ER skills. Perhaps more cognitively focused treatments (e.g., trauma-focused cognitive behavioral therapy; Cohen, Mannarino, & Deblinger, 2006) may be better suited for youth with intact or higher levels of ECF. Conversely, treatments that emphasize the development of coping skills and improving ER (e.g., dialectical behavior therapy;

Linehan, 1993) may be particularly useful for maltreated youth with higher levels of emotion dysregulation. Youth with a history of maltreatment with more severe ECF or ER deficits may benefit from greater involvement from a non-perpetrator caregiver in their treatment. Finally, given findings that ECF and ER deficits can enhance vulnerability for AU, it is possible that concurrently addressing these deficits while providing trauma-related interventions would lead to lower rates of AU. Thus, future research should examine poor ECF and ER in the context of intervention efforts to investigate whether ECF and ER profiles predict treatment outcome, whether it is effective to match treatments to youth based on their self-regulation abilities, and whether ECF and ER can be modified by intervention efforts. If ECF and ER are found to predict treatment outcome, further research should determine whether adding modules focusing on ECF and ER to existing treatments improves outcomes compared to typical treatment approaches. Finally, given the malleability of ECF and ER across developmental periods, it is possible that interventions may modify ECF and ER functioning. Thus, research should consider not only outcomes related to AU or other typical targets, but whether ECF and ER change from baseline beyond changes attributable to maturation. This reciprocal relation suggests that enhancing ECF and ER functioning could lead to improved and more sustainable treatment gains.

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