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Confirmatory Factor Analysis of Oppositional Defiant Disorder within Clinically Referred Youth

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CONFIRMATORY FACTOR ANALYSIS OF OPPOSITIONAL DEFIANT DISORDER WITHIN CLINICALLY REFERRED YOUTH

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ABSTRACT

CONFIRMATORY FACTOR ANALYSIS OF OPPOSITIONAL DEFIANT DISORDER WITHIN CLINICALLY REFERRED YOUTH

by

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Each new edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; APA, 2013) has been met with substantial criticism. Particularly, in DSM-5, two disorders were defined by very similar criteria. Oppositional defiant disorder (ODD) was defined as consisting of three dimensions - irritability, noncompliance, and spiteful/vindictive. Additionally, ODD has duration criteria that indicate its symptoms must be present for at least 6 months suggesting the presence of chronic irritability. DSM-5 also included disruptive mood dysregulation disorder (DMDD) as a disorder marked by the presence of chronic irritability in childhood and adolescence. The question of whether chronic irritability (i.e., DMDD) can be separated from ODD in clinical settings is a substantial question. Most studies indicate that DMDD and ODD have significant overlap (Freeman et al., 2016; Mayes et al., 2016). An alternate method is to examine whether ODD consists of independent or correlated dimensions. The factor structure of ODD can inform questions regarding whether irritability is a distinct dimension within ODD. Therefore, examining competing models of the factor structure of ODD in a clinical sample and externally validating the resulting dimensions should inform whether irritability should be treated as a unique, separate dimension of psychopathology or whether it is subsumed within a broader disruptive behavior dimension. The current study hypothesized that across parent and

clinician ratings, ODD would have a multidimensional factor structure consisting of at least irritability and noncompliance factors. ODD's factor structure would be best explained via a general ODD factor and two specific factors representing irritability and noncompliance. Additionally, the current study hypothesized that irritability and noncompliance will be differentially associated with internalizing symptoms and psychopathology and externalizing symptoms and psychopathology respectively. The hypotheses were partially supported. Implications for clinical decision making are discussed.

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CHAPTER 1

LITERATURE REVIEW

A re-emergence in the study of irritability has taken place in the past 15 years. Irritability is an approach-oriented, negative affective state in which an individual's heightened physiological arousal increases the propensity for subsequent states of frustration, anger, and aggression (Amsel, 1992; Amsel & Ward, 1954; Avenevoli, Blader, & Leibenluft, 2015; Toohey & DiGiuseppe, 2017). Irritability is normative. Almost all preschool age children display temper loss, a behavioral manifestation of irritability (Wakschlag et al., 2007, 2012). Only approximately 10% of those children have severe, clinically significant temper loss on a daily basis (Wakschlag et al., 2012). Severe, chronic irritability is trans-diagnostic (i.e., not specific to a single diagnosis) and has traditionally been considered a sign of emotion regulation difficulties (Reimherr et al., 2005). The non-specific, trans-diagnostic nature of irritability has long been implicitly recognized in diagnostic nosology. Irritability is listed as an associated feature or diagnostic symptom to many psychiatric disorders (American Psychiatric Association (APA), 2013; World Health Organization (WHO), 1992). However, the role of chronic and severe irritability in the demarcation of pediatric bipolar disorder (PBD) and the increased attention towards irritability as an affective dimension of oppositional defiant disorder (ODD) has driven a renewed focus on whether irritability should be thought of as an affective, internalizing symptom or a disruptive, externalizing symptom. DSM-5 introduced disruptive mood dysregulation disorder as a new mood disorder while maintaining the same symptom set as criteria for ODD (APA, 2012). Therefore, whether irritability's widespread presence among clinical psychiatric disorders is indicative of a specific affective dimension or simply a marker of more general emotion dysregulation in clinical populations is critical to diagnostic and treatment decisions.

The purpose of the current study is to evaluate the latent structure of irritability in clinical settings across informants and measures. The literature review is organized into four sections. First, the theoretical and clinical foundations of irritability from multiple disciplines are integrated. Second, current clinical conceptualizations are considered. Third, the developmental trajectories associated with irritability are evaluated. Fourth, treatment considerations and irritability's potential to impact clinical treatment planning are underscored.

Theoretical Foundations

As irritability has come to the forefront of recent research, the manner in which irritability is defined is inconsistent at best (Toohey & DiGiuseppe, 2017). Operationalization of irritability ranges from the causes of irritability (i.e. heightened physiological arousal) to the experience of irritability (i.e. anger) and to the consequences of irritability (i.e. frustration; aggression). While some argue that current definitions of irritability are difficult to distinguish from similar constructs such as anger and aggression (e.g., Toohey & DiGiuseppe, 2017), others have noted that this might be a reflection of the history of the study of irritability being closely intertwined with aggression research (Deveney et al., 2013). The separation of irritability from aggression is most clearly seen through behavioral tasks that induce irritability without inducing aggression. The following section reviews the early literature on irritability and predominantly concerns the definition of irritability pertaining to the consequences of irritability such as frustration and aggression.

Frustrative Non-Reward. Early work in irritability predominantly focused on a behavioral definition of irritability as a frustrative response to nonreward in the examination of the extinction process. Early learning theory viewed nonreward (i.e., extinction) as having no inherent motivational properties (Amsel & Ward, 1954). In these early conceptualizations, the

extinction process was a passive process that resulted in link between the stimuli and the conditioned behavior eroding over time. However, Amsel and Ward (1954) demonstrated that nonreward (i.e., early phase of the extinction process) holds strong motivational properties of its own. For example, rats can be conditioned to expect reward to a specific stimulus (i.e., reward expectancy). When rewards to a conditioned response to a specific stimuli were withdrawn, the rats initially displayed vigorous approach behaviors and little frustration. The continued absence of the reward resulted in the rats exhibiting aversive emotional reactions as marked by conflict-like behavior. As nonreward continued, rats reverted back to vigorous approach behavior. As more time elapsed, the conditioned behavior would eventually stop. The process of behavioral and affective responding was labeled the frustration effect (Amsel & Ward, 1954). The frustration effect altered the conceptualization of nonreward from a passive process to an active process and suggested that nonreward was also important to learning (Amsel, 1962). In modern clinical writings on behavioral therapy, this process is often referred to as an "extinction burst" (e.g. Miltenberger, 2001) and can be dampened by the inclusion of other practice elements in addition to extinction (Lerman & Iwata, 1995).

The frustration effect was translated into young children and adolescents early in the study operant learning. For example, researchers would condition children to pull a lever to receive a reward by a partial reinforcement schedule. On trials in which reward was withheld, the children's subsequent lever-pulling behavior was significantly faster than when compared to trials in which they were immediately rewarded (Watson & Ryan, 1966). However, children display individual differences in reaction to nonreward. Younger children and those with low expectancies for success had immediate reactions to nonreward, whereas older children and those children with high reward expectancies more frequently demonstrated a delayed reaction to

nonreward, taking longer to pull the lever subsequent to nonreward trials (Watson, 1970). The frustration effect and the presence of individual differences or variability has been supported consistently in children (Davidson & Fitzgerald, 1970; Deur & Parke, 1970; Libb & Serum, 1974; Watson, 1970). Both the immediate reaction to nonreward seen in the younger children and the delayed responses from the older children provide evidence for the motivational and inhibitory properties of nonreward. Most importantly, these early studies indicate a developmental trajectory of normative irritability as well as the presence variability in who is and who is not at high risk for irritability.

Frustration Aggression Hypothesis. The early studies of the frustration effect in children were initially conceptualized as demonstrations of the relevance of behavioral theory to humans. Early aggression researchers attempting to identify what causes aggressive behavior had already identified similar circumstances as a risk for future aggressive behavior (Dollard, Miller, Doob, Mowrer, & Sears, 1939). Blending the early theoretical work with behavioral theory resulted in a series of studies demonstrating that behaviorally induced frustration can result in aggression (e.g. Buss, 1963). The frustration aggression hypothesis posited that the frustration effect evokes negative affect (i.e. irritability) and the negative affect elicits aggressive cognitions and behaviors (L. Berkowitz & Devine, 1989; R. Berkowitz, 1988). Early work focused on frustration and irritability as emotional states that increase an individual's susceptibility to aggressive behavior (Caprara, Paciello, Gerbino, & Cugini, 2007; Caprara, Renzi, Alcini, Imperio, & Travaglia, 1983; Caprara, Renzi, Amolini, D'Imperio, & Travaglia, 1984). In contrast to frustrative non-reward line of work that elucidated individual differences in frustration, work in the frustration-aggression hypothesis line clarified the situational parameters necessary for frustration to occur as well as lead to aggression. First, frustration and aggression

are most likely to occur when an individual is near to obtaining a goal (Harris, 1974). Second, frustration is more likely to lead to aggression if the situation either primes aggression or makes available opportunities for aggression (e.g. Leyens, Camino, Parke, & Berkowitz, 1975). Third, frustration is likely to produce a state tendency toward aggression that dissipates over time if aggressive responses or targets are not made available (Miller, 1941). Therefore, this early line of work helps to set boundaries on irritability. Irritability is typically a temporary, emotional state characterized by a lowered threshold for impulsive, aggressive, and aversive reactions to goal blocking that may increase aggressive tendencies (Caprara & Pastorelli, 1993; Caprara et al., 1984).

General Aggression Model. The general aggression model (GAM) integrates earlier individual versus environment theories of aggression. GAM posits that situational and personal factors interact to influence one's present internal state and subsequent cognitive processes (Anderson & Bushman, 2002). For example, chronically irritable individuals are more likely to be hypersensitive to situations of nonreward, causing them to experience more acute states of irritability and react more sensitively to external provocations with angry rumination, hostility, and aggressive behavior (Anderson & Bushman, 2002; Caprara et al., 2007, 1983, 1984). For example, individuals with both high and low levels of trait irritability were asked to participate in a learning task and a subsequent extra sensory perception task. Immediately following the task, participants were either given positive or negative feedback. During the following extra sensory perception task, participants were given the opportunity to punish a confederate with electric shocks. Those individuals higher in trait irritability were significantly more likely to punish the confederates after negative feedback. Additionally, they were significantly more likely to punish the confederate at a higher intensity than those individuals low in trait irritability (Caprara,

1982). This example study highlights the integration of within person differences (i.e., trait irritability), environmental risk (i.e., access to shock as a punishment), and frustrations role in aggression.

Reactive and Impulsive Aggression. In contrast to theories attempting to model the process of aggression in general, clinically oriented theorists proposed a taxonomy of reactive and proactive aggression to distinguish youth at risk for conduct disorder from other youth with disruptive behavior disorders (Kempes, Matthys, de Vries, & van Engeland, 2005). Reactive aggression occurs in response to perceived threat or provocation. Proactive aggression occurs to aid in goal obtainment. The proactive and reactive taxonomy relies heavily on social information processing theory (Crick & Dodge, 1994). According to social information processing theory, an individual engages in the following series of steps in response to social cues: (a) encoding of cues, (b) interpretation of cues, (c) clarification of goals, (d) accesses responses and (e) decides on a response (Crick & Dodge, 1994). Negative affective states tend to narrow information processing (Bolte & Goschke, 2010) resulting in deficits in the encoding and interpretation of cues (Crick & Dodge, 1994; Dodge & Coie, 1987). As a result, individuals with higher levels of irritability tend to react more aggressively when provoked under frustrating and ambiguous circumstances (Caprara et al., 2007, 1983) because emotional regulation difficulties predispose individuals to more narrowly interpret the world around one resulting in a tendency to react defensively with aggression (Calvete & Orue, 2012; de Castro, Bosch, Veerman, & Koops, 2003; Fite et al., 2016; Hubbard et al., 2002; Kaynak, Lepore, Kliewer, & Jaggi, 2015; Sullivan, Helms, Kliewer, & Goodman, 2010; Zeman, Shipman, & Suveg, 2002). In fact, the endorsement of irritability is positively associated with reactive aggression (Smeets et al., 2017). Individuals with chronic irritability often display poor emotion regulation strategies, low tolerance for

provocation, and greater access to aggressive cognitions (Smeets et al., 2017). Thus, individuals with chronic irritability are at an increased risk for reactive aggression.

Similar to reactive aggression, impulsive aggression is angry, retaliatory aggression arising out of frustration, annoyance, or hostility to real or perceived provocations. Impulsive aggression represents an unplanned and immediate response that reflects out-of-control emotionality that satisfies immediate emotional pressures (Saylor & Amann, 2016). Individuals with chronic and severe irritability are prone to overly angry, aggressive reactions in response to provocation, and thus, these individuals are at risk for impulsive aggression as well (Caprara et al., 1984). Angry rumination and reduced self-control, both of which are seen within severely irritable individuals, are likely the mechanisms linking irritability to impulsive aggression (Denson, Pedersen, Friese, Hahm, & Roberts, 2011). For example, provoked participants demonstrate reduced self-control capacities on subsequent tasks unrelated to aggression (Denson et al., 2011). When given time to engage in angry rumination, provoked participants reported feeling more emotionally depleted and engaged in more aggressive behavior compared to participants not given time to ruminate (Denson et al., 2011). Therefore, individuals with chronic and severe irritability are more likely to be more sensitive to external provocations as well as feeling irritability.

Integrating Aggression and Irritability. The predominant focus of most literature in regards to irritability has been as a precursor to anger and aggression. Definitions of irritability in this context rely heavily on irritability's association with these constructs, so much so that it is often difficult to separate out irritability from the behavioral consequences of aggression and frustration. However, each of these approaches provided clues to irritability. First, irritability is a physiological response to nonreward (Amsel & Ward, 1954). Second, individuals differ in their

susceptibility to frustration (Davidson & Fitzgerald, 1970; Deur & Parke, 1970; Libb & Serum, 1974; Watson, 1970). Third, situational circumstances have a causal effect on the onset of irritability (Anderson & Bushman, 2002; Caprara et al., 2007, 1983, 1984). Fourth, irritability results in narrowed information processing marked most frequently by deficits in encoding and interpretation of situational characteristics (Bolte & Goschke, 2010; Crick & Dodge, 1994; Dodge & Coie, 1987). Fifth, environmental manipulations affect the type and strength of irritabilities consequences (Caprara et al., 1984; Denson et al., 2011). In summary, these disparate traditions that used irritability in both experimental and observational paradigms point toward a distinct, meaningful construct of irritability that is closely related to anger and aggression.

Clinical Foundations

Irritability holds an almost ubiquitous role among psychiatric disorders, as it is listed as a symptom, associated feature, or descriptor to a vast majority of disorders included within the DSM (Safer, 2009). Irritability's pervasive presence among disorders, including both internalizing and externalizing disorders, raises nosological concerns and begs the question as to whether irritability is best conceptualized as a general marker of psychopathology or is itself representative of its own unique pathology. In context, this represents the debate between the analogies of irritability is like a "fever" (i.e., a general marker; Youngstrom, 2013) and irritability is like hypertension (i.e., unique pathology; Stringaris & Goodman, 2009b). As reviewed in the following sections, much of this debate has been driven by the role of irritability in the classification of disorders.

Pediatric Bipolar Disorder. Current clinical concern and conceptualizations of irritability trace back to the controversies surrounding childhood mania that emerged in the early

1990s (Leibenluft & Stoddard, 2013). Disagreement regarding the phenomenology of pediatric mania dominated the early literature and irritability played a significant role in the varying definitions of childhood mania (Carlson, 1990; Carlson & Klein, 2014; Galanter & Leibenluft, 2008; Harrington & Myatt, 2003; Kent, 2003; Klein, Pine, & Klein, 1998). Three definitions of PBD emerged from the literature with each emphasizing a distinct features of mania. First, the "narrow" definition of pediatric mania requires a symptom profile that includes the symptoms of elevated mood and grandiosity (Leibenluft, Charney, Towbin, Bhangoo, & Pine, 2003). Second, the "broad" phenotype defined pediatric mania as consisting of chronic emotional dysregulation accompanied by severe irritability and temper outbursts characterized by rage (Carlson & Klein, 2014; Mick, Spencer, Wozniak, & Biederman, 2005; D. Papolos, Mattis, Golshan, & Molay, 2009). The "broad" phenotype suggests that pediatric mania presents more chronically and primarily as "irritable or affective storms" without clearly distinguishable episodes (Biederman et al., 2004; Davis, 1979; Mick et al., 2005; D. Papolos et al., 2009). Between these two extremes is the "DSM" or "intermediate" phenotype that emphasizes episodic change and the presence of DSM consistent symptoms (i.e., elated mood or irritability). For prototypical cases of PBD, the definitions are likely minimally important. However, youth presentations of mania are more likely to be mixed episodes and be longer in duration but with subthreshold symptom presentations. In this light, the intermediate definition can be divided into two subcategories: individuals presenting with the hallmark symptom of elation who do not meet the duration criterion and those individuals meeting the full duration criterion but who present with irritable mania or hypomania (Leibenluft et al., 2003). Thus, irritability's role within pediatric bipolar disorder spurred a surge of research focused on how to best classify irritability because of its role in potentially defining "border" cases of PBD.

A driving force behind this surge in interest was the concern raised by dramatically increasing rates of diagnosis of PBD in the late 1990s and early 2000s (Blader & Carlson, 2007; Case, Olfson, Marcus, & Siegel, 2007; Moreno et al., 2007). Increases in the rate of clinical diagnoses of PBD could be attributed to the clinical use of the "broad" phenotype as popularized in *The Bipolar Child* (Papolos, 2003). Similarly, adults with labile mood are more likely to be misdiagnosed as having bipolar disorder when other disorders such as borderline personality disorder are more appropriate (Ruggero, Zimmerman, Chelminski, & Young, 2010). Accurate diagnosis matters because treatments for bipolar disorder require ongoing medication management (Connolly & Thase, 2011). A consequence of labeling more youth with PBD was an increase in the number of youth being treated with medications approved for the treatment of bipolar disorder. Typically, medications such as Aripiprazole, Lithium, or Quetiapine are used in the treatment of PBD and these medications come with significant adverse side effects (Díaz-Caneja et al., 2014; Liu et al., 2011). Therefore, irritability's role within PBD was and remains center to the debate regarding appropriate diagnosis of PBD.

Severe Mood Dysregulation. Severe mood dysregulation (SMD) is a syndrome defined to encompass youth experiencing the severe, chronic irritability and hyperarousal that comprise the core symptomatology and presentation of the "broad" phenotype of bipolar disorder (Leibenluft et al., 2003). Criteria for SMD include severe negative affect, hyperarousal, markedly increased reactivity to negative emotional stimuli as compared to peers, and the presence of frustration or temper tantrums. SMD is primarily defined as a chronic presentation of irritability and hyperarousal without other symptoms of mania (e.g., grandiosity), whereas PBD is an episodic illness with manic symptoms present (Leibenluft, 2011; Towbin, Axelson, Leibenluft, & Birmaher, 2013). In introducing SMD, Leibenluft and colleagues were

operationalizing the broad phenotype with the explicit purpose of examining its boundaries with a narrow phenotype of bipolar disorder.

As the result of investigating whether nonepisodic, severe irritability was similar to or different from episodic moods with potential changes in irritability, much of the prior work contrasts youth with SMD to youth with PBD or healthy controls. For example, youth with SMD have lower conversion rates to bipolar I disorder in adulthood relative to youth with PBD (Axelson et al., 2012; Birmaher et al., 2009, 2006; Brotman et al., 2006; Stringaris et al., 2010). Familial history of BD is significantly higher in youth with PBD relative to youth with SMD (Birmaher et al., 2009; Brotman et al., 2007; Perich et al., 2016; Rende et al., 2007). Furthermore, youth with SMD show different neuropsychological functioning relative to youth with PBD. Compared to youth diagnosed with PBD, youth with SMD are shown to display left amygdala hyper-activation in response to facial emotion processing tasks (Brotman, Rooney, Skup, Pine, & Leibenluft, 2009). Additionally, youth with SMD tend to exhibit different attentional biases in comparison to youth diagnosed with PBD (Rich et al., 2010, 2008). Both longitudinal and cross-sectional examinations indicate that the broad phenotype as operationalized in SMD is not the same thing as PBD. However, the SMD literature is extremely young and has weaknesses. Cross-sectional studies examining the neural mechanisms related to SMD youth's processing of emotional stimuli have resulted in conflicted findings. While youth presenting with SMD have similar face emotion labeling deficits as youth diagnosed with bipolar disorder (Deveney et al., 2013; Guyer et al., 2007; Kim et al., 2013; Rich et al., 2008), youth with SMD differ from youth with bipolar disorder by displaying no attentional bias towards positive or negative images (Rich et al., 2010). On the whole, the evidence supports SMD as different from PBD.

Current Conceptualizations

DMDD, a disorder characterized by severe and chronic irritability, was introduced into the depressive disorder category of the DSM-5 based on SMD findings coupled with associations between chronic childhood irritability and later depressive disorders (Roy, Lopes, & Klein, 2014). The symptomology of this disorder includes a) severe, recurrent temper outbursts that are considered out of proportion in intensity or duration to the situation or provocation and b) the temper outbursts must be considered inconsistent with the developmental level of the child. Specific criteria for the disorder include a) the temper outbursts must occur on average three or more times per week, b) the child's mood between temper outbursts must be persistently irritable for most of the day, nearly every day and be observable by others, c) these symptoms must have been present for at least one year and must not have had a period lasting more than 3 months within this time during which these symptoms were not present, d) these symptoms must have been present within two of the following settings: either the home, school, or with peers; and must be severe in at least one of these settings, e) the diagnosis must be made between the ages of 6 and 18, and f) the symptoms must have begun before the age of 10, g) there must never had been a distinct period lasting more than 1 day during which the full symptom criteria for a manic or hypomanic episode have been met, h) the symptoms of DMDD cannot be limited to an episode of major depressive disorder and cannot be better explained by autism spectrum disorder, posttraumatic stress disorder, separation anxiety disorder, or persistent depressive disorder and i) lastly, DMDD cannot be concurrently diagnosed with ODD, intermittent explosive disorder, or bipolar disorder (APA, 2013). Though DMDD's inclusion into the DSM-5 was almost completely based on the SMD literature, DMDD's symptomology and diagnostic criteria differ from SMD in important ways. Criteria for SMD include severe negative affect,

hyperarousal, markedly increased reactivity to negative emotional stimuli as compared to peers, and the presence of frustration or temper tantrums. Thus, SMD's core features of hyperarousal and increased reactivity are not present in the proposed DMDD symptomology.

Few empirical prospective studies of DMDD have been conducted, retrospective secondary analysis of studies fitting DMDD to existing measures have been completed. These studies indicate that prevalence rates for DMDD range from .8% to 30.5% depending on factors such as population, informant, and how strictly criteria are applied (Axelson et al., 2012; Copeland, Angold, Costello, & Egger, 2013; Freeman, Youngstrom, Youngstrom, & Findling, 2016; Margulies, Weintraub, Basile, Grover, & Carlson, 2012). In both clinical and community samples, youth with DMDD are more likely to receive mental health services, exhibit greater functional impairments, more suicidality, and higher rates of learning disabilities (Copeland et al., 2013). In longitudinal studies, youth with DMDD are more likely to be of lower socioeconomic and educational statuses, as well as to report poorer health outcomes (Copeland, Shanahan, Egger, Angold, & Costello, 2014). However, youth with DMDD have extremely high rates of comorbidity which calls into question the diagnostic specificity of the disorder (Copeland et al., 2013). Therefore, understanding the diagnostic boundaries of chronic irritability in clinical populations is critical.

Chronic irritability has historically been largely ignored as a distinct characteristic of psychopathology. Some effort to distinguish phasic and tonic irritability has existed. Phasic irritability is most often associated with affective disorders and tonic irritability most often associated with oppositional defiant disorder (ODD). Recent work in ODD suggests that there are at least two related symptom dimensions. Classically, ODD is defined by noncompliant behavior (e.g., talking back, not following rules); however, recent work suggests that youth with

ODD also experience clinically significant irritability. Therefore, a critical, unanswered question is whether chronic irritability should be an externalizing or internalizing disorder as well as whether irritability should be separated into its own disorder in general.

Oppositional defiant disorder (ODD) has the highest degree of overlap with DMDD. The two disorders share core symptoms of temper outbursts and irritability. Perhaps it is not unexpected that ODD and DMDD should frequently be comorbid. However, the degree to which the two have been shown to overlap within community and epidemiological samples raises concern. For example, multiple clinical and community studies have indicated that youth with DMDD and youth with ODD display similar levels of impairment (Althoff et al., 2016; Axelson et al., 2012; Dougherty et al., 2014; Freeman et al., 2016; Mayes, Waxmonsky, Calhoun, & Bixler, 2016). Similarly, questions regarding whether DMDD can be differentiated from ODD based on symptomology question the validity and utility of the DMDD diagnosis (Mayes et al., 2016). Therefore, the lack of evidence for reliably differentiating DMDD, a mood disorder, from ODD, a disruptive behavior disorder, highlights the problem of how irritability is conceptualized across psychopathology.

In summary, irritability is a transdiagnostic symptom or associated feature of almost all disorders in childhood and adolescence. Substantial debate regarding whether irritability is a more general marker of psychopathology or a marker of a unique disorder continues.

Developmental Trajectories of Irritability

Irritability is prevalent across childhood both as a normative developmental experience as well as a marker for childhood psychopathology, making it an almost ubiquitous phenomenon. The prevalence of irritability during childhood and adolescence has been found to be as high as 50% (Copeland, Brotman, & Costello, 2015). Children and adolescents experience both phasic

and tonic irritability at separate times as well as concurrently (Copeland et al., 2015). Severe and chronic irritability is a much less common circumstance, with prevalence rates among children between .8 and five percent (Althoff, Verhulst, Rettew, Hudziak, & van der Ende, 2010; Brotman et al., 2006; Copeland et al., 2013). Severe and chronic irritability is far less stable than the typical irritability found in childhood and adolescence. Of youth with DMDD, only 20% will continue to meet a diagnosis of DMDD over a three-year period (Axelson et al., 2012) and approximately 30% of children will meet criteria over a longitudinal course of eight years (Mayes et al., 2016). Therefore, understanding the longitudinal associations of the presence of chronic irritability might shed light onto the utility of studying irritability as a unique entity.

Historically, irritability has been closely associated with aggression, delinquency, and more externalizing symptoms (Aebi, Plattner, Metzke, Bessler, & Steinhausen, 2013; Amsel & Ward, 1954; Caprara et al., 2007, 1983; Ezpeleta, Granero, de la Osa, Penelo, & Domènech, 2012). Chronic irritably likely causes hypersensitivity to perceived provocations which leads to more acute states of irritability (Anderson & Bushman, 2002; Bolte & Goschke, 2010; Caprara et al., 2007, 1983, 1984). State (or phasic) irritability results in a higher propensity towards angry rumination, hostility, and aggressive behavior (Anderson & Bushman, 2002; Caprara et al., 2007, 1983, 1984). However, childhood irritability is associated with the development of both anxiety and depression in adolescence and early adulthood have garnered greater attention (J. Burke & Loeber, 2010; Kuny et al., 2013; Stringaris & Goodman, 2009). Therefore, irritability potentially represents a junction between externalizing and internalizing symptoms (Leadbeater & Homel, 2015; Stringaris & Goodman, 2009).

Investigation into the angry and irritable mood dimension of ODD has resulted in a clear demarcation of divergent pathways between the irritability and noncompliance (Althoff, Kuny-

Slock, Verhulst, Hudziak, & van der Ende, 2014; Ezpeleta et al., 2012; Lavigne, Gouze, Bryant, & Hopkins, 2014; Leadbeater & Homel, 2015; Stringaris, Rowe, & Maughan, 2012; Whelan, Stringaris, Maughan, & Barker, 2013). Defiance and oppositional behavior predicts future conduct problems more strongly than internalizing symptoms (Althoff et al., 2014; J. Burke & Loeber, 2010; Leadbeater & Homel, 2015; Whelan et al., 2013). However, irritability displays developmentally distinct outcomes when compared to defiant and oppositional behavior associated with ODD. Irritability is more strongly associated with internalizing symptoms than those conduct problems (Stringaris et al., 2012). Additionally, genetic studies indicate that irritability in adolescence displays a significantly stronger association with depression than it does with conduct problems (Stringaris et al., 2012). Therefore, irritability might play a causal role in the development of later depression and anxiety.

Treatment

Significant to the rationale behind the introduction of DMDD into the latest revision of the DSM was growing concern regarding increased diagnosis of bipolar disorder in youths and adolescents presenting with severe and chronic irritability. Beyond nosological concerns, the question of whether these youths were receiving the correct treatment for their symptoms became central to this debate. Childhood irritability is predictive of the development of both anxiety and depression in adolescence and early adulthood (J. Burke & Loeber, 2010; Kuny et al., 2013; Stringaris & Goodman, 2009). The best treatment course for severe and chronic irritability if it is a mood disorder should be analogous to established treatments for affective disorders. However, irritability has also been shown to be closely associated with aggression, delinquency, and more externalizing symptoms (Aebi et al., 2013; Amsel & Ward, 1954; Caprara et al., 2007, 1983; Ezpeleta et al., 2012). Evidence-based treatments for mood disorders

and disruptive behavior disorders are quite distinct. Pharmacologically, depressive disorders in children and adolescents are typically treated with an SSRI such as fluoxetine (Cipriani, Geddes, Furukawa, & Barbui, 2007). In contrast, disruptive behavior disorders are often treated with stimulants and atypical antipsychotics (Gurnani, Ivanov, & Newcorn, 2016). From a psychosocial treatment perspective, evidence supported treatments (EST) for unipolar depressive disorders typically consist of pleasant activity scheduling and challenging cognitions (Weersing, Jeffreys, Do, Schwartz, & Bolano, 2017). ESTs for disruptive behavior disorders typically rely on contingency management approaches (Kaminski & Claussen, 2017; McCart & Sheidow, 2016). Therefore, the question is whether a disorder marked by severe and chronic irritability should be treated as a mood disorder or a disruptive behavioral disorder.

Within the limited literature base, treatments for SMD and DMDD range from psychotherapeutic to medication-based to a combination of the two (Benarous et al., 2017). Psychotherapeutic treatment trials have been completed in which investigators attempted to treat severe, chronic irritability as a disruptive behavior disorder with social skills training, reward based contingency management, affect regulation, parent training, and hostile interpretation therapy (Krieger et al., 2011; Stoddard et al., 2016; Waxmonsky et al., 2013; 2008). Other psychotherapeutic trials have attempted to treat SMD similarly to a unipolar depressive disorder with dialectical behavior therapy and interpersonal psychotherapy (Benarous et al., 2017; Dickstein et al., 2009; Parmar, Vats, Parmar, & Aligeti, 2014; Waxmonsky et al., 2008). Pharmacological studies have also been conducted in which SMD was treated with antidepressants, which is comparable to pharmacological treatments for unipolar depressive disorders, as well as with stimulants and anti-psychotics, which are comparable to pharmacological treatments of disruptive behavior disorders (Dickstein et al., 2009; Krieger et

al., 2011; Parmar et al., 2014; Waxmonsky et al., 2013). While many studies are ongoing, early findings suggest that parent training associated with CBT or behavior therapy may show potential for reduction of irritability symptoms (Waxmonsky et al., 2013; 2008). Similarly, there is evidence to show that interpretation bias therapy may be effective in the treatment of DMDD (Stoddard et al., 2016). Preliminary results suggest support for the use of anti-psychotics or stimulants as treatment for SMD but not lithium (Connor, Glatt, Lopez, Jackson, & Melloni, 2002; Dickstein et al., 2009; Krieger et al., 2011; Waxmonsky et al., 2013). Despite these findings, there are significant limitations to the trials that have been conducted thus far. Many, if not all of the studies, suffer from small sample sizes and nearly 100% comorbidity rate with ADHD. Moreover, not all samples were randomized to treatment group (Benarous et al., 2017). These limitations call into question the utility of these results. Thus, whether DMDD should be treated as an externalizing or internalizing disorder remains to be definitively determined.

CHAPTER 2

PURPOSE OF THE PRESENT STUDY

Each new edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; APA, 2013) has been met with substantial criticism. In DSM-5, two disorders were defined by very similar criteria. Oppositional defiant disorder (ODD) was defined as consisting of three dimensions - irritability, noncompliance, and spiteful/vindictive. Additionally, ODD has duration criteria that indicate its symptoms must be present for at least 6 months suggesting the presence of chronic irritability. DSM-5 also included disruptive mood dysregulation disorder (DMDD) as a disorder marked by the presence of chronic irritability in childhood and adolescence. One line of criticism regarding DMDD was that it was a new name for an already existing phenomena (Freeman et al., 2016; Mayes et al., 2016). The question of whether chronic irritability (i.e., DMDD) is separate from ODD is a substantial question. Most studies indicate that DMDD and ODD have significant overlap (Freeman et al., 2016; Mayes et al., 2016). An alternate method is to examine whether ODD consists of independent or correlated dimensions. The factor structure of ODD can inform questions regarding whether irritability is a distinct dimension within ODD. Therefore, examining competing models of the factor structure of ODD in a clinical sample and externally validating the resulting dimensions should inform whether irritability should be treated as a unique, separate dimension of psychopathology or whether it is subsumed within a broader disruptive behavior dimension.

Historically, the symptoms of ODD have been conceptualized as unidimensional (Bezdjian et al., 2011; Burns, Boe, Walsh, Sommers-Flanagan, & Teegarden, 2001; Evans et al., 2013; Hartman et al., 2001; Molina, Smith, & Pelham, 2001; Pelham, Gnagy, Greenslade, & Milich, 1992). ODD is often considered a stepping stone to more severe future disruptive

behavior disorders such as conduct disorder (R Loeber, Green, Lahey, Frick, & McBurnett, 2000; Nock, Kazdin, Hiripi, & Kessler, 2007) or as a more moderate presentation of conduct problems that could evolve into more severe conduct problems (Loeber, Burke, & Pardini, 2009). However, recent work examining the structure of ODD symptoms in large community and epidemiological samples suggested that ODD is multidimensional (Burke et al., 2014) and may consist of at least an irritable/affective factor and oppositional/noncompliance factor. In trajectory research based on these dimensions, oppositional symptoms predict the onset of future disruptive behavior problems and affective, or irritability, symptoms predict future affective symptoms (J. Burke & Loeber, 2010; Leadbeater & Homel, 2015; Stringaris & Goodman, 2009; Whelan et al., 2013). The transition in the conceptualization of ODD from a disruptive behavior disorder to potentially an affective disorder has the potential to significantly alter existing treatment discussions as they relate to this disorder and more specifically irritable symptoms. Most evidence-based practice recommendations call on clinicians to treat the current presenting problem, but accounting for or attempting to prevent future negative outcomes might result in changes to current practice.

Prior work has relied on both exploratory and confirmatory factor analyses (EFA; CFA) to validate the structure of ODD. These models have supported latent structures consisting of one, two, or three dimensions. Historically, the broadest support was for ODD to be treated as a single dimension (Bezdjian et al., 2011; Burns et al., 2001; Evans et al., 2013; Hartman et al., 2001; Molina et al., 2001;Pelham et al., 1992). In these studies, ODD symptoms were analyzed with other externalizing psychopathology symptoms (e.g., conduct problems, ADHD symptoms). When examined in the context of other psychopathology, EFA consistently finds that ODD symptoms factor together into a single dimension (e.g., Bezdjian et al., 2011; Pelham et al.,

1992). In CFA, the model must be pre-specified and then compared with competing approaches (Kline, 2015). Many studies using CFA tested only a unidimensional structure and did not compare models evaluating multidimensional structures for ODD (e.g., Burns et al., 2001; Evans et al., 2013; Hartman et al., 2001; Molina et al., 2001; Pelham et al., 1992). In the context of other externalizing psychopathology, ODD symptoms form a unidimensional structure. Factor analyses of ODD symptoms including multiple disorders might bias ODD towards unidimensionality. Therefore, ODD symptoms should be evaluated alone in order to understand the factor structure of ODD.

Figure 1 displays the series of models used to define the latent structure of ODD. These models consist of a number of one, two- and three- simple factor structure models as well as bifactor models. Of these different models, a two-factor model consisting of two correlated, specific factors has been supported (Burke, Loeber, Lahey, & Rathouz, 2005; Rowe, Costello, Angold, Copeland, & Maughan, 2010). In Model B and Model C, the behavioral and affective factors are present but with slightly different symptom sets. Model B consists of an oppositional behavior and a negative affect factor, while Model C consists of an irritable and a headstrong/spiteful factor. In addition to two-factor models, three-factor models have found broad support in the literature (Aebi et al., 2013; Burke & Loeber, 2010; Krieger et al., 2013; Stringaris & Goodman, 2009). In particular, two competing models have been identified. Model D consists of correlated oppositional behavior, antagonistic, and negative affect specific factors (Burke & Loeber, 2010). Similar to Model D, Model E consists of correlated irritable, headstrong, and hurtful specific factors (Aebi et al., 2013; Krieger et al., 2013; Stringaris & Goodman, 2009). Model E, consisting of correlated irritable, headstrong, and hurtful factors, was adopted by DSM-5 (APA, 2013). Even in these more detailed approaches to ODD, there remains

inconsistencies regarding multidimensional models of ODD. An outstanding question is precisely how many meaningful factors are present. For example, the meaningfulness of the hurtful dimension identified in Model E is questioned because it fails to predict meaningful outcomes in longitudinal studies and often becomes untestable when spitefulness and vindictiveness are treated as a single item (Ezpeleta et al., 2012; Rowe et al., 2010). Additionally, the proposed factors tend to display extremely high correlations with each other. Therefore, while the evidence is supportive of multidimensional models, questions remain regarding the best fitting model for ODD.

The inconsistencies in structure and the high correlation among factors may suggest that simple factor structure is not sufficient to fully explain variance among ODD items. Bifactor analysis is one hierarchical modeling strategy that may be beneficial to identifying more complicated factor structures. Bifactor analysis models a general, overarching dimension and specific subdimensions. The overarching dimension reflects the common variance among all the items within a construct and the specific subdimensions reflect the unique remaining shared variance. Given that prior work has focused either on ODD being a single unidimensional set of symptoms or multiple dimensions, CFA modelling including bifactor models provides a framework in which a unidimensional structure, multidimensional structure with simple structure, or multidimensional with bifactor structure is best fitting. Burke et al. (2014) examined ODD in a series of community samples using this approach. A modified bifactor model in which a general ODD factor in conjunction with correlated specific irritable and oppositional factors (Model G in Fig 1) displayed the best fit across multiple community-based samples. However, the best fitting model could reflect over-fitting (Rodriguez, Reise, & Haviland, 2016). For example, when additional indicators of model quality are applied beyond model fit the bifactor

model presented no longer appears to always be the best fitting model. Also, the theoretical implications of the models varies from sample to sample. Some of the models indicate a strong general factor with weak, uninterpretable specific factors, while some indicate a weak general factor with strongly interpretable specific factors. Additionally, prior analyses reflect work in community samples in which youth are less impaired and have lower rates of comorbidity. Berkson's Paradox refers to sampling hospital patients to identify risk factors and that for that selection bias in sampling that these risk factors might not generalize to the general population (Berkson, 1946). The reverse is also likely true in that risk factors in a public health center (e.g., a community sample) might not apply to a selected sample (e.g., a clinical sample) in a meaningful manner due to filtering effects in the treatment seeking process. Therefore, determining the structure of ODD in the context of clinical settings is critical because youth in clinical settings represent a small subset of youth with psychopathology that might be different from youth in the general population.

There are disadvantages to bifactor modeling. First, bifactor modelling explicitly defines the relationship between indicators and factors. The partitioning of variance so precisely typically results in the loss of reliability in the specific factors as variance is attributed to the general factor (Gignac, 2016; Rodriguez et al., 2016). Second, because bifactor models model more relationships, fit statistics, even those with penalties for model complexity, tend to be biased toward identifying bifactor models over simple structure models (Rodriguez et al., 2015). To account for the potential for over-fitting, many commentators suggest evaluating additional model-based reliability indices (e.g., $\omega,\ \omega_H, \omega_S$) to help determine whether bifactor models are necessary (e.g., Rodriguez et al., 2015). As seen in Table 1, these additional fit statistics applied to Burke and colleagues (Burke et al., 2014) bifactor analysis of ODD indicate that within three

of the five samples tested the specific irritability and headstrong subdimensions do not represent reliable subfactors. This suggests that a more parsimonious approach to these samples would be a simpler model (Rodriguez et al., 2015). Therefore, to fully evaluate the factor structure of ODD one must also investigate the meaningfulness of the factors beyond simply the best fitting model.

Determining the significance and meaningfulness of a specific irritability factor is critical because the way that this dimension is conceptualized directly informs clinical treatment planning and outcomes of interest. The classical understanding of ODD as a unidimensional disorder suggests an overarching behavioral dysfunction should be the primary focus of treatment and longitudinal outcomes of interest include more severe conduct symptoms. In contrast, multidimensional models consisting of separate oppositional behavior and irritability dimensions suggest a different set of outcomes. Both behavioral and affective outcomes become critical. Additionally, each may have its own etiology leading to different treatment foci and clinical outcomes. Thus, identifying the latent structure of ODD in a clinical population could help inform treatment planning.

Aims and Hypotheses.

Aim 1. Evaluate the factor structure of ODD within a clinical sample.

Hypothesis 1. Across parent and clinician ratings, ODD will have a multidimensional factor structure consisting of at least irritability and noncompliance factors. ODD's factor structure will be best explained via a general ODD factor and two specific factors representing irritability and noncompliance.

Hypothesis 2. The general ODD factor will be reliable and account for most of the explained variance in ODD. The specific factors of ODD (i.e., irritability, noncompliance) should display reliable variance.

Aim 2. Evaluate the convergent validity of irritability and noncompliance in a clinical sample.

Hypothesis 3. Irritability will be positively associated with anxiety and depressive symptoms after controlling for noncompliance and the general ODD factor.

Hypothesis 4. Noncompliance will be positive associated with more severe conduct problems after controlling for irritability and the general ODD factor.

Hypothesis 5. Irritability will predict depression and anxiety disorder diagnoses after controlling for noncompliance and the general ODD factor.

Hypothesis 6. Noncompliance will predict conduct disorder diagnoses after controlling for irritability and the general ODD factor.

CHAPTER 3

METHOD

Participants

Participants were youth and caregiver dyads presenting to an urban community mental health center or academic medical center in the Midwest (n = 828). At the community mental health center, all new intakes of youth ages 5-18 years were offered the opportunity to participate regardless of presenting symptoms and/or concern. The academic medical center included specialty clinics in pediatric mood disorders but was running treatment trials for a variety of pediatric concerns (e.g., bipolar disorder, unipolar depression, schizophrenia, PTSD, ADHD). Additionally, offspring of parents with bipolar disorder being treated in an adult clinic were also included in the study resulting in an enriched rate of bipolar disorder at the academic medical center. Inclusion criteria for both sites were: (a) youths were between the ages of 5 and 18 years, (b) both the caregiver and youth provided written and/or verbal consent or assent, (c) both the caregiver and youth presented for the assessment, and (d) both the caregiver and youth were conversant in English. As seen in Table 2, participants were primarily male (60%), African-American (70%), 10.9 years old (SD = 3.42), and had high rates of comorbidity.

Measures

Child Behavior Checklist (CBCL). The CBCL is a caregiver-reported measure of emotional and behavioral problems across 8 empirically-derived dimensions and 6 DSM-oriented dimensions (Achenbach & Rescorla, 2001). The CBCL consists of 118 problem items that caregivers answer using a Likert scale ranging from 0-2 (not true – sometimes true - very true or often true). Caregivers of youth aged 6-18 completed the CBCL for 6-18 years and caregivers of 5-year-olds completed the CBCL 1.5-5.5 years. As displayed in Table 3, ODD

dimensions in the current study are defined in the following ways. Irritability is defined as a negative affective state characterized by heightened physical arousal. Items from the CBCL (items #37, #45, #68, #86, #87, and #95), were selected to measure irritability. Noncompliance is defined as the refusal to act in accordance with an instruction or command. Items from the CBCL (items # 3, #22, #23, #28, and #39) were selected to measure noncompliance. Spiteful/vindictive is defined as deliberately causing harm or hurting another for the purpose of revenge or getting back at someone. Items were selected from the CBCL (items #15, #16, #21, #25, #34, #48, #57, and #97) to measure spitefulness/vindictiveness. Items were chosen based on previous literature as well as theory.

Schedule for Affective Disorders and Schizophrenia for Children (KSADS). Highly trained research assistants administered the KSADS to youth and caregiver. The KSADS is a semi-structured interview that queries about the presence of symptoms from common disorders in childhood and adolescence. The KSADS–PL–Plus amalgamates the mood modules from the Washington University KSADS (Geller et al., 2001) and the KSADS Present and Lifetime Version (Kaufman et al., 1997). The Washington University KSADS includes additional symptoms and associated features of depression and mania beyond those included in the KSADS Present and Lifetime Version. Research assistants were highly trained: Symptom level ratings for new raters were compared with those of a reliable rater for at least five interviews rating along and then five interviews leading. A new rater passed a session if he or she achieved an overall $\kappa >= .85$ at the symptom level and a $\kappa = 1.0$ at the diagnostic level. A new cohort of raters was trained each year, and videotaped interviews were used to avoid rating drift across cohorts. Research assistants were primarily pre-doctoral psychology interns or research staff with a master's degree or PhD in psychology or a master's degree in social work. The following items

were selected from the KSADS to measure ODD: easily annoyed, angry/resentful, spiteful/vindictive, annoys people on purpose, blames others, loses temper, argues a lot, disobeys/defies.

Diagnoses. Final diagnoses were assigned by a licensed psychologist using the Longitudinal Evaluation of All Available Data (LEAD) procedure (Spitzer, 1983). During the LEAD meeting, the research assistant presented the KSADS symptoms and diagnoses, family history, and information available from intake (e.g., intake diagnoses, chart review of diagnoses, prior treatment history, and school history). Both the licensed clinical psychologist and the research assistant were blind to the questionnaire results.

Procedure. All study procedures were approved by the Case Western Reserve University and Applewood Centers, Inc. IRBs. Intake clinicians invited all intakes to participate in the study. At the time of the study assessment, caregivers provided written consent for the youths to participate in the study. Youths provided written/verbal assent to participate in the study. The same research assistant interviewed both the caregiver and youth individually with the KSADS. Questionnaires were completed as part of an additional battery while the opposite informant was being interviewed. Assistance was provided by an additional research assistant to both the caregiver and youth as necessary.

Data Analytic Plan.

Primary analyses were conducted in R (R Core Team, 2013). Given that the unit of analysis in the current study is both item and scale level, all data was screened for missingness and distributional assumptions. Approximately 30% of data were missing across all types of data. Most of the missing data was due to design of the KSADS items. Supplemental items were administered only if screening items were scored as clinically significant. These items were

treated as a missing at random. Other missing data appear to be missing completely at random. Multiple imputation by chained equations using the R-package MICE (Van Buuren and Groothuis-Oudshoorn, 2011) was used to create thirty, item-level imputed datasets for analysis. Multiple imputation is currently regarded as a state-of-the-art technique because it improves accuracy and statistical power relative to other missing data techniques (Akande, Li, & Reiter, 2017). Imputation models included demographics, diagnoses, and item level responses for scales used in the planned analyses. Variables that correlated greater than .1 in the available data were included in the imputation model.

Analyses. *Aim 1.* Evaluate the factor structure of ODD within a clinical sample.

Confirmatory factor analysis was used to systematically test a set of unidimensional, multi-dimensional simple structure, and multidimensional bifactor factor structures. Figure 1 displays the set of models that were tested. Specifically, the following models were tested: Model A, a single factor (General ODD); Model B, a model with two correlated factors (ODD behavior and ODD negative affect); Model C, a model with two correlated factors (ODD irritable and ODD headstrong/spiteful); Model D, a model with three correlated factors (ODD behavior, ODD headstrong, and ODD negative affect); Models E and F, a model with three correlated factors (ODD irritable, ODD headstrong, and ODD hurtful); Model G, a bifactor model with two orthogonal specific factors (irritability and oppositional behavior) and a general ODD factor; Model H, a modified bifactor model with two correlated specific factors (irritability and oppositional behavior) and a general ODD factor.

Within Model A, all 8 indicators were specified to load onto the general ODD factor (i.e. “angry”, “defies”, “annoys”, “blames”, “touchy”, “angry”, “spiteful/vindictive”, and “temper”). In Model B, the indicators “argues”, “defies”, and “temper” were specified to load onto the ODD

behavior factor and the indicators “touchy”, “angry”, and “spiteful/vindictive” were specified to load onto the ODD negative affect factor. In Model C, the indicators “temper”, “touchy”, and “angry” were specified to load onto the ODD irritable factor and the indicators “argues”, “defies”, “annoys”, “blames”, and “spiteful/vindictive” were specified to load onto the ODD headstrong/spiteful factor. In Model D, the indicators “temper”, “argues”, and “defies” were specified to load onto the ODD behavior factor. The indicators “annoys” and “blames” were specified to load onto the ODD antagonistic factor. The indicators “touchy”, “angry”, and “spiteful/vindictive” were specified to load onto the ODD negative affect factor. In Model E, the indicators “temper”, “touchy”, and “angry” were specified to load onto the ODD headstrong factor. The indicators “argues”, “defies”, “annoys”, and “blames” were specified to load onto the ODD headstrong factor. The indicator “spiteful/vindictive” were specified to load onto the ODD hurtful factor. In Model F, the indicators “temper”, “touchy”, and “angry” were specified to load onto the ODD irritable factor. The indicators “argues”, “blames”, and “defies” were specified to load onto the ODD headstrong factor. The indicators “annoys” and “spiteful/vindictive” were specified to load onto the ODD Hurtful factor. In both Model G and Model H, all 8 indicators were specified to load onto the general ODD factor (i.e. “angry”, “defies”, “annoys”, “blames”, “touchy”, “angry”, “spiteful/vindictive”, and “temper”). Indicators “temper”, “touchy”, and “angry” were specified to load onto the irritability subfactor. Indicators “argues”, “defies”, “annoys”, “blames”, and “spiteful” were specified to load onto the oppositional behavior subfactor.

Confirmatory factor analyses were fit using the R-packages lavaan (Rosseel, 2012) and semTools (Jorgensen, Pornprasertmanit, Schoemann, & Rosseel, 2018). The semTools package provides functions that wraparound lavaan for multiply imputed data including pooled likelihood

ratio test statistics (Li, Meng, Raghunathan, & Rubin, 1991). The item-level data violated assumptions of conventional approaches to confirmatory factor analysis (Wirth & Edwards, 2007). For example, item level data violated the assumption of multivariate normality necessary for more traditional CFA estimation algorithms (e.g., maximum likelihood). Following current recommendations, the polychoric correlation matrix was estimated and then the polychoric correlation matrix was factor analyzed using a diagonally weighted least squares estimator (WLSMV; Jöreskog & Aish, 1990; Muthén, 1984). Results are presented using a standardized latent variable with mean of 0 and variance of 1 (Kline, 2015).

While there are no universally accepted fit indices or cutoff values for the fit indices (McDonald, 2010), simulation studies indicate that an evaluation of the Akaike Information Criteria (AIC), Bayesian Information Criteria (BIC), Tucker Lewis Index (TLI; also known as the non-normed fit index (NNFI)), Comparative Fit Index (CFI), Root Mean Square Error of Approximation (RMSEA) and χ^2 are useful in identifying global fit. From an overall model fit perspective, the criteria presented in Table 4 were initially used (Hu & Bentler, 1999; MacCallum, Browne, & Sugawara, 1996) as well as an examination of the residuals correlation matrix. Overall model fit was determined by examining all fit indices together as well as ensuring minimal remaining correlations in the residual correlation matrix. Nested models were compared via $\Delta\chi^2$, AIC, BIC, ΔCFI, ΔRMSEA and the amount of variance explained by the model. Models indicated as significantly different by $\Delta\chi^2$, having lower AIC & BIC, ΔCFI > .01, ΔRMSEA > .015 and explaining more variance than alternative models were preferred (Chen, 2007; Cheung & Rensvold, 2002).

The quality of the factor solution was also evaluated by the internal consistency of the Irritability and Headstrong factors. Model-based reliabilities were estimated with coefficient

omega (ω; Raykov, 2001). Omega can be calculated in multiple manners. The ω presented is an estimate of reliability controlling for the other factors (similar to partial eta-squared in ANOVA).

Aim 2. Evaluate the convergent validity of irritability and noncompliance in a clinical sample.

Irritability and noncompliant dimensions were expected to uniquely predict different outcomes. A series of multiple linear regressions were fit to the data. Regressions were crossed by informant such that caregiver-report was predicted by clinician rated variables and vice-versa. From the clinician-reported variables, dependent variables were KSADS diagnoses of depressive disorders, anxiety related disorders, and disruptive behavior disorders and independent variables consisted of the identified irritability and noncompliance symptoms. From the caregivers, dependent variables consisted of the Internalizing and Externalizing subscales less the items being modeled and the independent variables consisted of scales created from the irritability and noncompliance items. Consistent with best practices, models were initially fit consisting of both the IVs and an interaction term (Laird & De Los Reyes, 2013). An examination of the partial regression coefficients allowed for a determination of whether a single predictor (e.g., irritability) accounted for more variance in the DVs after controlling for the presence of another predictor (e.g., noncompliant symptoms). We conducted a brief simulation study to estimate power using the software package, R (R Core Team, 2013). Although we have a total sample of 828 dyads, we initially expected missing data to be present. Therefore, for the simulation study we varied sample size from n=50 to n=600 in increments of 50 and entered a three variable equation (X_1, X_2, and X_1*X_2) into the model. The effect sizes for the IVs ranged from $\beta = .01$ to $\beta = .75$ in increments of .05. The alpha level used for this analysis was $p < .05$. The power analyses revealed statistical power for this study to be greater than 99% across sample sizes for

large effects and across effect sizes for largish samples (e.g., n > 400). Thus, power should be adequate for the proposed study. For the presented analyses, complete data via multiple imputation resulted in the full sample being utilized.

CHAPTER 4

RESULTS

Aim 1. Evaluate the factor structure of ODD within a clinical sample.

KSADS. A series of CFA models were fit to the clinician-reported ODD items. Table 3 and Figure 1 display the items included in each of the models and competing factor structures. As seen in Table 5, all the simple structure models demonstrated excellent fit. The bifactor model demonstrated poor fit and the modified bifactor model did not converge. Nested models were compared with each other in order to find the best fitting simple structure model. χ^2 difference tests were performed to contrast the unidimensional model with the two-factor models. In both cases, the unidimensional model was inferior to the two two-factor models. Furthermore, the two two-factor models were contrasted with the three-factor model displaying the best indices of fit. In both cases, χ^2 difference tests indicate that the three-factor model was superior to each of the other two models. However, given the fit indices of the two-factor and three-factor models are so similar and in the interest of parsimony, the two-factor model was chosen as the best fitting model.

Model C consists of an irritability factor (items: odd1sc, odds1sc, and odds2sc) and a headstrong factor (items: odd2sc, odd3sc, odds5sc, odds6sc, and odds3sc). On the irritability factor, all items were uniformly significant and all greater than .82. Similarly, on the headstrong factor all items were uniformly significant and all greater than .50. The Omega reliability coefficient was .83 for the irritability factor and .80 for the headstrong factor. The irritability and headstrong factors were strongly correlated, r = .90, 95% CI [.86, .94].

CBCL. A similar series of CFA models were fit to caregiver-reported data. As seen in Table 7, the two-factor model and the traditional bifactor model both displayed excellent fit. The

unidimensional model and the three factor model both displayed poor fit. The modified bifactor model did not converge. Nested models were contrasted in order to determine the best fitting model. χ^2 difference tests were performed to contrast the unidimensional model with the two-factor model. The two-factor model fit significantly better than the unidimensional model. Furthermore, the two-factor model was significantly better than the three-factor model, statistics $\chi^2 = 6099.33$, $p < .001$. Lastly, the two-factor model was significantly better fitting than the traditional bifactor model, $\chi^2 = 1461.48$, $p < .001$.

As seen in table 8, the two-factor model consisted of an irritability factor (items: cbc86, cbc95, cbc87, cbc68, cbc37, cbc45) and a Headstrong factor (items: cbc28, cbc22, cbc23, cbc03, and cbc39). On the irritability factor, all items were uniformly significant and all greater than .29. Similarly, on the headstrong factor all items were uniformly significant and all greater than .28. The Omega coefficient was .73 for the irritability factor and .81 for the headstrong factor. The irritability and headstrong factors were strongly correlated, r = .80, 95% CI [.76, .84].

Aim 2. Evaluate the convergent validity of irritability and noncompliance in a clinical sample.

A series of hierarchical linear regressions were fit to the data. Regressions were crossed by informant to account for potential within rater variance. Caregiver-report CBCL syndrome scales were predicted by clinician-report irritability and headstrong scales. The caregiver-report dependent variables were the Anxious/Depression, Withdrawn/Depression, Somatization, Social Problems, Attention Problems, Rule-Breaking, Aggression, Affective Problems, Anxiety Problems, ADHD Problems and ODD Problems scales less the items included in the irritability and headstrong subscales. The clinician-reported independent variables consisted of the identified irritability and noncompliance symptoms. Clinician-reported diagnoses were predicted

by caregiver-report irritability and headstrong scales. The clinician-reported dependent variables were diagnoses (0 = No diagnosis, 1 = Diagnosis present) of depressive disorders, anxiety-related disorders, and disruptive behavior disorders. The caregiver-reported independent variables were the irritability and headstrong factors identified in aim 1. A series of hierarchical models were fit with the irritability factor entered first, the headstrong factor second, and the interaction third.

Internalizing DV.

Caregiver-reported Internalizing Symptoms Predicted by Clinician-reported Irritability and Headstrong. Table 9 displays the results. Higher levels of clinician-reported irritability were expected to predict more caregiver-reported internalizing symptoms, such as depression and anxiety. Additionally, the Clinician-reported Headstrong dimension was not expected to predict internalizing symptoms. After controlling for gender and age, clinician-reported Irritability predicted a significant increase in caregiver-reported Anxious/Depression, b = .21, 95% CI [.02, .41], p = .03, R^2 = .04. Even after controlling for clinician-reported Headstrong, this association held, b = .32, 95% CI [.04, .59], p = .03. Once controlling for clinician-reported Irritability, clinician-reported Headstrong was not significantly associated with caregiver-reported Anxious/Depression, b = -.10, 95% CI [-.30, .09], p = .31, ΔR^2 = .00 The interaction between Irritability and Headstrong was not significant, b = .05, 95% CI [-.03, .12], p = .21, ΔR^2 = .00.

After controlling for gender and age, clinician-reported Irritability was not significantly associated with caregiver-reported Withdrawn/Depression, b = .05, 95% CI [-.07, .18], p = .38, R^2 = .01. However after controlling for clinician-reported Headstrong, increases in clinician-reported Irritability were associated with increases in caregiver-reported Withdrawn-Depression,

b = .21, 95%CI [.03, .38], *p* = .02. Once controlling for clinician-reported Irritability, increases in clinician-reported Headstrong were associated with increases in caregiver-reported Withdrawn/Depression, b = -.15, 95% CI [-.28, -.03] *p* = .02, ΔR^2 = .00.The interaction was not significant, b = .01, 95% CI [-.04, .05] *p* = .78, ΔR^2 = .00.

After controlling for gender and age, clinician-reported Irritability predicted a significant increase in caregiver-reported Affective Problems, b = .21, 95% CI [.01, .42], *p* = .04, R^2 = .01. Controlling for clinician-reported Headstrong, this association was no longer significant, b = .29, 95% CI [-.00, .58], *p* = .05. Once controlling for clinician-reported Irritability, clinician-reported Headstrong was not associated with caregiver-reported Affective Problems, b = -.08, 95% CI [-.28, .13], *p* = .47, ΔR^2 = .00. Additionally, the interaction not significant, b = .03, *p* = .51, ΔR^2 = .00.

After controlling for gender and age, clinician-reported Irritability was not significantly associated with caregiver-reported Somatization, b = .07, 95% CI [-.01, .15], *p* = .11, R^2 = .01. However after controlling for clinician-reported Headstrong, increases in clinician-reported Irritability were associated with increases in caregiver-reported Somatization, b = .12, 95%CI [.01, .24], *p* = .04. Once controlling for clinician-reported Irritability, clinician-reported Headstrong was not associated with caregiver-reported Somatization, b = -.06, 95%CI [-.14, .03], *p* = .18, ΔR^2 = .00.The interaction was not significant, b = .02, *p* = .15.

After controlling for gender and age, clinician-reported Irritability was not significantly associated with caregiver-reported Anxiety Problems, b = .12, 95% CI [-.01, .24], *p* = .08, R^2 = .04. This held true even after controlling for clinician-reported Headstrong, b = .16, 95% CI [-.02, .34], *p* = .09. Once controlling for clinician-reported Irritability, clinician-reported Headstrong was not associated with caregiver-reported Anxiety Problems, b = -.05, 95%CI [-.18,

.08], p = .49, ΔR^2 = .00.The interaction was not significant, b = .03, p = .16. In summary, clinician-reported Irritability was associated with increases in caregiver-reported Anxious/Depression, Withdrawn/Depression, and Somatization. Clinician-reported Headstrong was associated with increases in caregiver-reported Withdrawn/Depression.

Clinician-reported Diagnoses predicted by Caregiver-reported Irritability and Headstrong. Table 10 displays the results. After controlling for gender and age, caregiver-reported Irritability significantly increased the odds of a youth receiving a Bipolar Disorder diagnosis, OR = 1.23, 95% CI [1.15, 1.31], p < .001, Cox & Snell R^2 = .06. Even after controlling for caregiver-reported Headstrong, this association remained significant, OR = 1.27, 95% CI [1.17, 1.37], p < .001. Once controlling for caregiver-reported Irritability, caregiver-reported Headstrong was not significantly associated with clinician-reported Bipolar Disorder, OR = .95, 95% CI [.87, 1.03], p = .24, , ΔR^2 = .00. There was not a significant interaction effect, OR = .99, p = .37, ΔR^2 = .00.

After controlling for gender and age, caregiver-reported Irritability significantly increased the odds of clinician-reported Suicide risk, OR = 1.11, 95% CI [1.05, 1.16], p < .001, Cox & Snell R^2 = .10. Even after controlling for caregiver-reported Headstrong, this association remained significant, OR = 1.18, 95% CI [1.10, 1.25], p < .001. Once controlling for caregiver-reported Irritability, caregiver-reported Headstrong significantly increased the odds of clinician-reported Suicide risk, OR = .90, 95% CI [.84, .96], p < .002, , ΔR^2 = .01. The interaction effect between caregiver-reported Irritability and caregiver-reported Headstrong was not significant, OR = 1.01, p = .29, ΔR^2 = .00.

After controlling for gender and age, caregiver-reported Irritability did not significantly increase the odds of clinician-reported Post-Traumatic Stress Disorder (PTSD), OR = 1.06, 95%

CI [.98, 1.15], p = .16, R^2 = .02. Even after controlling for caregiver-reported Headstrong, this remained true, b = 1.03, 95% CI [.93, 1.15], p = .54. Once controlling for caregiver-reported Irritability, caregiver-reported Headstrong was not significantly associated with clinician-reported PTSD, OR = 1.05, 95% CI [.93, 1.12], p = .44, ΔR^2 = .00. The interaction effect was not significant, OR = .99, p = .51, ΔR^2 = .00.

After controlling for gender and age, caregiver-reported Irritability did not significantly increase the odds of clinician-reported Generalized Anxiety Disorder (GAD), OR = 1.01, 95% CI [.91, 1.11], p = .91, R^2 = .004. Even after controlling for caregiver-reported Headstrong, this remained true, b = 1.09, 95% CI [.96, 1.24], p = .18. Once controlling for caregiver-reported Irritability, caregiver-reported Headstrong significantly increased the odds of clinician-reported GAD, OR = .86, 95% CI [.75, .99], p = .04, , ΔR^2 = .005. The interaction effect was not significant, b = 1.01, p = .50, ΔR^2 = .00.

After controlling for gender and age, caregiver-reported Irritability did not significantly increase the odds of clinician-reported Unipolar Depression, OR = 1.00, 95% CI [.95, 1.05], p = .89, Cox & Snell R^2 = .06. Even after controlling for caregiver-reported Headstrong, this remained true, OR = 1.06, 95% CI [.99, 1.13], p = .09. Once controlling for caregiver-reported Irritability, caregiver-reported Headstrong significantly increased the odds of clinician-reported Unipolar Depression, OR = .90, 95% CI [.84, .97], p = .004, , ΔR^2 = .01. The interaction was not significant OR = 1.00, p = .74, ΔR^2 = .00.

Irritability, as reported or rated by caregivers and clinicians, was expected to predict more internalizing psychopathology while Headstrong was not expected to predict more internalizing psychopathology. This hypothesis was supported in that clinician-reported irritability was associated with caregiver-reported internalizing problems across domains. Additionally,

caregiver-reported irritability predicted more suicide risk and higher odds of bipolar disorder diagnoses. However, caregiver-reported irritability did not predict diagnoses associated with internalizing disorders. Headstrong was associated with increases in Withdrawn/Depression, increased suicide risk, higher odds of GAD, and high odds of Unipolar Depression. Therefore, partial support for the hypothesis was found.

Externalizing DV.

Caregiver-reported Externalizing Symptoms Predicted by Clinician-reported Irritability and Headstrong. Table 11 displays the results of the regression models for these analyses. Clinician-reported Irritability was not expected to predict more caregiver-reported externalizing symptoms such as rule-breaking and aggression. Instead, the clinician-reported Headstrong dimension was expected to predict externalizing symptoms. After controlling for gender and age, clinician-reported Irritability significantly predicted increases in caregiver-reported Aggression, b = .84, 95% CI [.68, .99], $p < .001$, $R^2 = .18$. Even after controlling for clinician-reported Headstrong, this held true, b = .25, 95% CI [.03, .46], $p = .02$. Once controlling for clinician-reported Irritability, clinician-reported Headstrong significantly predicted increases in caregiver-reported Aggression, b = .59, 95% CI [.44, .74], $p < .001$, $\Delta R^2 = .05$. The interaction was not significant, b = .03, $p = .23$.

After controlling for gender and age, clinician-reported Irritability significantly predicted increases in caregiver-reported ODD problems, b = .59, 95% CI [.50, .68], $p < .001$, $R^2 = .25$. Even after controlling for clinician-reported Headstrong, this relationship remained significant, b = .24, 95% CI [.12, .36], $p < .001$, $\Delta R^2 = .01$. Once controlling for clinician-reported Irritability, clinician-reported Headstrong significantly predicted increases in caregiver-reported ODD

Problems, b = .35, 95% CI [.26, .43], $p < .001$, $\Delta R^2 = .01$. The interaction was not significant, b = -.03, $p = .05$.

After controlling for gender and age, clinician-reported Irritability significantly predicted increases in caregiver-reported Rule-Breaking, b = .51, 95% CI [.30, .72], $p < .001$, $R^2 = .04$. Once controlling for clinician-reported Headstrong, this relationship was not significant b = .22, 95% CI [-.08, .51], $p = .15$. Once controlling for clinician-reported Irritability, clinician-reported Headstrong significantly predicted increases in caregiver-reported Rule Breaking, b = .29, 95% CI [.09, .50], $p < .01$, $\Delta R^2 = .01$. The interaction was not significant, b = .04, $p = .32$.

After controlling for gender and age, clinician-reported Irritability significantly predicted increases in caregiver-reported ADHD Problems, b = .42, 95% CI [.30, .55], $p < .001$, $R^2 = .08$. After controlling for clinician-reported Headstrong, this relationship was no longer significant, b = -.11, 95% CI [-.28, .06], $p = .22$. . Once controlling for clinician-reported Irritability, clinician-reported Headstrong significantly predicted increases in caregiver-reported ADHD Problems, b = .53, 95% CI [.41, .65], $p < .001$, $\Delta R^2 = .08$. The interaction was not significant, b = -.02, $p = .35$.

After controlling for gender and age, clinician-reported Irritability significantly predicted increases in caregiver-reported Attention Problems, b = .38, 95% CI [.24, .52], $p < .001$, $R^2 = .07$. After controlling for clinician-reported Headstrong, this relationship was not significant, b = -.07, 95% CI [-.27, .13], $p = .48$. Once controlling for clinician-reported Irritability, clinician-reported Headstrong significantly predicted increases in caregiver-reported Attention Problems, b = .45, 95% CI [.31, .59], $p < .001$, $\Delta R^2 = .04$. Additionally, the interaction was not significant, b = -.01, $p = .84$. In summary, clinician-reported Irritability was associated with increases in Aggression and ODD, but not with Rule-Breaking, Attention Problems, or the ADHD subscale.

Clinician-reported Headstrong was associated with increases in caregiver-reported Aggression, ODD Problems, Rule Breaking, ADHD Problems, and Attention Problems.

Clinician-reported Externalizing Diagnoses Predicted by Caregiver-reported Irritability and Headstrong. Table 12 displays the results of the logistic regressions. After controlling for gender and age, caregiver-reported Irritability significantly increased the odds of clinician-reported Conduct Disorder, OR = 1.28, 95% CI [1.18, 1.39], $p < .001$, Cox & Snell $R^2 = .09$. Even after controlling for caregiver-reported Headstrong, this held true, OR = 1.16, 95% CI [1.05, 1.27], $p < .004$. Once controlling for caregiver-reported Irritability, caregiver-reported Headstrong significantly increased the odds of clinician-reported Conduct Disorder, OR = 1.22, 95% CI [1.09, 1.36], $p < .001$, Cox & Snell $\Delta R^2 = .02$.The interaction was not significant, b = 1.01, $p = .37$, Cox & Snell $\Delta R^2 = .00$.

After controlling for gender and age, caregiver-reported Irritability significantly increased the odds of clinician-reported ADHD, OR = 1.24, 95% CI [1.17, 1.31], $p < .001$, Cox & Snell $R^2 = .21$. Even after controlling for caregiver-reported Headstrong, this held true, OR = 1.09, 95% CI [1.02, 1.17], $p = .01$. Once controlling for caregiver-reported Irritability, caregiver-reported Headstrong significantly increased the odds of clinician-reported ADHD, OR = 1.29, 95% CI [1.19, 1.39], $p < .001$, Cox & Snell $\Delta R^2 = .04$. The interaction was not significant, OR = .98, $p = .06$, Cox & Snell $\Delta R^2 = .01$.

After controlling for gender and age, caregiver-reported Irritability significantly increased the odds clinician-reported ODD, OR = 1.23, 95% CI [1.17, 1.30], $p < .001$, Cox & Snell $R^2 = .11$. Even after controlling for caregiver-reported Headstrong, this held true, OR = 1.15, 95% CI [1.08, 1.22], $p < .001$. Once controlling for caregiver-reported Irritability, caregiver-reported Headstrong significantly increased the odds of clinician-reported ODD, OR = 1.15, 95% CI

[1.07, 1.23], $p < .001$, Cox & Snell $\Delta R^2 = .01$. The interaction was not significant, OR = .96, $p = .001$, Cox & Snell $\Delta R^2 = .05$.

After controlling for gender and age, caregiver-reported Irritability did not significantly increase the odds of clinician-reported Substance Use, b = 1.02, 95% CI [.91, 1.14], $p = .72$, Cox & Snell $R^2 = .08$. Even after controlling for caregiver-reported headstrong, this association remained not significant, OR = .97, 95% CI [.84, 1.11], $p = .61$, Cox & Snell $\Delta R^2 = .00$. Once controlling for caregiver-reported Irritability, caregiver-reported Headstrong was not significantly associated with clinician-reported substance-use. Similarly, the interaction was non-significant, OR = 1.02, $p = .37$, Cox & Snell $\Delta R^2 = .00$.

Irritability, as reported or rated by caregivers and clinicians, and after controlling for headstrong was expected to not significantly predict more externalizing symptoms such as aggression and disruptive behavior disorders, while Headstrong was expected to only predict externalizing symptoms. This prediction was partially supported by the results as both clinician-reported and caregiver-reported irritability significantly predicted externalizing scales from the CBCL and externalizing disorders from the KSADS. Irritability, as reported by caregivers and rated by clinicians, predicted the Social Problems, Aggression, and ODD Problems scales from the CBCL as well as ADHD, CD, and ODD diagnoses from the KSADS, even after controlling for headstrong behaviors. Headstrong did significantly predict caregiver-reported Aggression, ODD Problems, Rule Breaking, ADHD, and Attention Problems and clinician-reported CD, ADHD, and ODD.

CHAPTER 5

DISCUSSION

Accurately identifying whether irritability exists as a dimension of ODD has important implications on the phenomenology of psychopathology in children and adolescents. If irritability is a distinct factor within ODD, then not only might clinical treatment planning and outcomes of interest need to account for the presence of irritability, but this also may provide support for a separate DMDD diagnosis. If irritability were simply a set of symptoms of a single ODD dimension, then the current efforts to characterize irritability as a separate, unique phenomena might be inappropriate. Conventional understanding of ODD is that it is a unidimensional disorder characterized by an overarching behavioral dysfunction that predicts longitudinal outcomes of more severe conduct symptoms (Bezdjian et al., 2011; Burns et al., 2001; Evans et al., 2013; Hartman et al., 2001; Molina et al., 2001; Pelham et al., 1992). In contrast, recent multidimensional models separate oppositional behavior and irritability dimensions that longitudinally predict different outcomes (Burke & Loeber, 2010; Leadbeater & Homel, 2015; Stringaris & Goodman, 2009; Whelan et al., 2013). Therefore, the results of our study, in either support or contradiction to this previous literature, can potentially add insight into how best to conceptualize chronic irritability in clinical settings.

Consistent with factor analysis in community samples, the results of our factor analyses support a multidimensional factor structure for ODD in a clinical sample (Spencer et al., 2017). Contrasting some community studies (Aebi et al., 2013; Bezdjian et al., 2011; J. D. Burke et al., 2014; J. Burke & Loeber, 2010; Burns et al., 2001; Evans et al., 2013; Krieger et al., 2013; Stringaris & Goodman, 2009) and in line with other community studies (Burke et al., 2005;

Rowe et al., 2010), ODD in a clinical setting appears to consist of Irritability and Headstrong Behaviors. While separate factors were present from both informants, the factors were strongly correlated in both sets of analyses. Strong correlations between Irritability and Headstrong dimensions were also found in most examinations of these dimensions (e.g., Aebi et al., 2012; Ezpeleta et al., 2012; Krieger et al., 2013). Even in clinical settings an affective component (i.e., irritability) and noncompliant behavior component (i.e., Headstrong Behaviors) can be distinguished but they appear to be highly correlated. Therefore, the uniqueness of a disorder characterized solely by severe and chronic irritability is questionable given that the strength of the correlation suggestions one should typically expect high levels of noncompliant symptoms as well.

Proponents of a DMDD diagnosis posit that the affective dimensions and behavioral dimensions of ODD longitudinally and cross-sectionally differentially predict more internalizing and more externalizing symptoms respectively. The irritability dimension of ODD is associated with emotional problems and lability (Aebi et al., 2013), depression (Burke & Loeber, 2010; Hipwell et al., 2011; Stringaris et al., 2012; Whelan et al., 2013) and more internalizing problems (Leadbeater & Homel, 2015). The behavioral dimension of ODD is associated with substance use disorders (Rowe et al., 2010), delinquency (Stringaris et al. 2012), ADHD, disruptive disorders, externalizing scales, callous-unemotional traits, and conduct disorder (Burke & Loeber, 2010; Lavigne et al., 2014; Stringaris & Goodman, 2009). Therefore, whether Irritability or Headstrong Behaviors displayed differential predictions is critical to informing the debate regarding whether these two dimensions should be treated separately in a clinical setting.

Our study indicated that both clinician- and caregiver-reported irritability partially align with the prior literature. Clinician-reported irritability significantly predicted increases in

caregiver-reported internalizing symptoms (i.e. Anxious/Depression, Withdrawn/Depression, Somatization, and Affective Problems). This finding lends support to previous studies that have concluded that irritability is associated with depression and anxiety. However, clinician-reported irritability was also associated with more externalizing symptoms such as social problems, aggression, and ODD problems. Moreover, our results indicate that caregiver-reported irritability predicts some internalizing-related pathologies (e.g., Bipolar Disorder, Suicide risk) as well as externalizing pathology (e.g., ADHD, CD, ODD). Irritability provided incremental utility in predicting these psychopathologies even after controlling for headstrong behaviors. Of particular note, the internalizing-related psychopathologies that irritability was associated with (e.g., Bipolar Disorder) are also marked by substantial externalizing features (Freeman, Youngstrom, Freeman, Youngstrom, & Findling, 2011). Therefore, irritability was associated with internalizing symptoms and psychopathology that proponents of a DMDD diagnosis have posited (Aebi et al., 2013; J. Burke & Loeber, 2010; Hipwell et al., 2011; Leadbeater & Homel, 2015; Stringaris et al., 2012; Whelan et al., 2013) but irritability in clinical samples was also consistent with externalizing symptoms and psychopathology as critics of this diagnosis have posited (Althoff et al., 2016; Axelson et al., 2012; Dougherty et al., 2014; Freeman et al., 2016; Mayes et al., 2016).

Both clinician- and caregiver-reported Headstrong only partially align with the previous literature. While clinician-reported Headstrong significantly predicted increases in caregiver-reported externalizing symptoms, clinician-reported Headstrong also significantly predicted increases in caregiver-reported internalizing symptoms (i.e. Withdrawn/Depression,). This finding contradicts previous studies that have concluded that Headstrong is not associated with depression and anxiety. Furthermore, our results indicate that caregiver-reported Headstrong

predicts some internalizing-related pathologies (e.g., Suicide risk, GAD, and Unipolar Depression) as well as externalizing pathology (e.g., ADHD, CD, ODD). Therefore, Headstrong was associated with internalizing symptoms and psychopathology which does not align with those who propose that DMDD, and internalizing disorder, is separate from ODD, an externalizing disorder.

The current study has substantial limitations. First, the sample consists of clinical referrals to a community mental health clinic. Clinical samples often have selection pressures that make their findings potentially biased when applied to the general population or used to directly inform theory. However, the question of whether irritability is distinct is highly relevant to clinical decision-making because of suggestions on how treatment should proceed for youth with severe irritability (Benarous et al., 2017). Second, clinician-reported irritability was constrained to irritability symptoms rated in the context of ODD. This methodology could have potentially increase the clinician-reported irritability association with caregiver-reported externalizing symptoms because the clinician-reported irritability symptoms were filtered (Findling et al., 2010). However, research assistants were trained to rate chronic irritability outside of the context of mood episodes in this section suggesting that these symptom ratings might be more transdiagnostic than the methodology might otherwise suggest. More importantly, caregiver-reported Irritability was unfiltered and the factor structure findings were consistent with the clinician-reported symptoms. Third, the data used in this study were cross-sectional in nature. Much of the prior literature on differential predictions between Irritability and Headstrong comes from longitudinal studies (Burke & Loeber, 2010; Kuny et al., 2013; Stringaris & Goodman, 2009). However, clinicians are often required to make initial clinical decisions based on cross-sectionally available data (e.g., current presenting symptoms). The

current study was predominantly concerned with clinical decision making as it pertains to the DMDD diagnosis and, more specifically, the clinical utility of a DMDD diagnosis over an ODD diagnosis within cross-sectional data. Cross-sectional data allow this line of inquiry because cross-sectional data come from the same time point, versus longitudinal data that come from different times points and are more concerned with how disorders unfold over time. For this reason, cross-sectional data were more appropriate for the current study. Due to differences between the two methodologies, the results of the current study cannot speak towards findings from longitudinal studies that suggest differential predictions between Irritability and Headstrong. While the current results indicate that noncompliant and irritability dimensions of ODD exist, they are highly correlated and patterns of comorbidity do not substantially help differentiate the two dimensions.

In the context of clinical practice, the current study indicates that irritability and headstrong behaviors are highly correlated but distinct. They demonstrate some differences in the prediction of internalizing and externalizing symptoms but also displayed significant overlap with each other that is somewhat contradictory to previous literature. Irritability, which has been proposed as an internalizing disorder (i.e. DMDD) predominantly associated with anxiety and depression, was also found to be associated with externalizing psychopathology. Headstrong, which has been conceptualized as the noncompliant dimension of ODD, has been predominantly found to be associated with more externalizing psychopathology and yet was found to be associated with internalizing symptoms and disorders. These findings coupled with published treatment trials that indicate that chronic irritability may respond well to treatments traditionally associated with externalizing psychopathology (Krieger et al., 2011; Stoddard et al., 2016; Waxmonsky et al., 2013; 2008) call into questions the meaningfulness of a disorder

characterized solely by severe and chronic irritability. Therefore, a DMDD diagnosis continues to be questionable.

Appendix A

Table 1

Omega Hierarchical and Omega Subscale Hierarchical Applied to Burke et al., 2014

Sample	ALSPAC	TTS	GTS	PYS	PGS
Omega Hierarchical					
General ODD Factor	.75	.01	.41	.75	.77
Omega Subscale Hierarchical					
Irritability Subfactor	.36	.82	.60	.20	.26
Headstrong Subfactor	.35	.90	.68	.26	.16

Appendix B

Table 2

Demographic Characteristics of Total Sample

Variable	
Gender *(%)*	
Male	60
Female	40
Ethnicity *(%)*	
African American	70
White	22
Age in years, *mean* (*SD*)	10.90 (3.42)
Number of diagnoses, *mean* (*SD*)	2.7 (1.4)

Appendix C

Table 3
Items Used to Define ODD Constructs

	KSADS	CBCL
Oppositional Defiant Disorder		
Irritability	S2. Angry or resentful	45. Nervous, high strung, or tense
		86. Stubborn, sullen, or irritable
	1. Loses Temper	87. Sudden changes in mood or feelings
		68. Screams a lot.
	S1. Easily Annoyed	95. Temper tantrums or hot temper
Noncompliance	2. Argues a lot with Adults	3. Argues a lot
		37. Gets in many fights.
	3. Disobeys Rules	22. Disobedient at home
		23. Disobedient at school
		28. Breaks rules at home, school, or elsewhere
	S4. Uses Bad Language	
		39. Hangs around with others who get in trouble
	S5. Annoys people on purpose	
	S6. Blames others	

Appendix D

Table 4
Criterion Values for Fit Indices

	Global Fit	
Index	Value	Interpretation
Tucker Lewis Index (TLI)	< .80	Bad
	> .80 & < .90	Possibly permissible
	> .90 & < .95	Adequate
	> .95	Good
Comparitive Fit Index (CFI)	< .80	Bad
	> .80 & < .90	Possibly permissible
	> .90 & < .95	Adequate/Good
	> .95	Excellent
Root Mean Square Error of Approximation (RMSEA)	> .10	Bad
	> .08 & < .10	Adequate
	> .05 & < .08	Good
	< .05	Excellent

Appendix E

Table 5

Confirmatory Factor Analysis Fit Indices for Clinician-reported ODD Symptoms

Number of Factors	Model	χ^2	df	TLI	CFI	RMSEA (90% CI)	RMR	$\Delta\chi^2$	Δdf	*p*
One	A	77.43	27	.97	.98	.05 (.04 - .06)	.05			
Two	B	72.78	26	.97	.98	.05 (.03 - .06)	.05	6.25	1	.01
Two	C	49.83	26	.99	.99	.03 (.02 - .06)	.04	30.37	1	<.001
Three	D	39.46	17	.99	.99	.04 (.02 - .05)	.04	4.47	9	>.05
Three	E	35.11	18	.99	.99	.03 (.02 - .05)	.04	13.59	8	>.05
Three	F	21.46	17	1.00	1.00	.02 (.00 - .04)	.03	28.49	9	.001
Traditional Bifactor	G	589.56	15	.56	.76	.22 (.20 - .23)	.27	-		-
Modified Bifactor	H	-	-	-	-	-	-	-	-	-

Note. Model H did not converge. Fit indices suggest Model C to be best fitting.

Appendix F

Table 6
Standardized Factor Loadings for Best Fitting Model from Table 5

	Irritability		Headstrong Behavior	
ODD item	β	*SE*	β	*SE*
Odds2sc. Angry	.91	.02		
Odd1sc. Temper	.86	.02		
Odds1sc. Touchy	.82	.02		
Odd2sc. Argues			.87	.02
Odd3sc. Defies			.81	.02
Odds3sc. Spiteful			.71	.03
Odds6sc. Blames			.69	.03
Odds5sc. Annoys			.65	.03
Odds4sc. Swearing			.50	.04

Note. Factor correlation between Irritability and Headstrong = .90

Appendix G

Table 7

Confirmatory Factor Analysis Fit Indices for Caregiver-Reported ODD Symptoms

Number of Factors	Model	χ^2	df	TLI	CFI	RMSEA (90% CI)	RMR	$\Delta\chi^2$	Δdf	*p*
One	A	6309.05	152	.71	.74	.22 (.22 - .23)	.23			
Two	B/C	403.46	43	.95	.96	.10 (.09 - .11)	.13	6099.33	109	<.001
Three	D/E/F	3072.19	149	.86	.88	.15 (.15 - .16)	.19	3229.30	106	<.001
Traditional Bifactor	G	1502.06	133	.93	.94	.11 (.11 - .12)	.14	1925.70	16	<.001
Modified Bifactor	H	-	-	-	-	-	-	-	-	-

Note. Model H did not converge. Nested model comparisons suggest the two-factor model to be the best fitting.

Appendix H

Table 8

Standardized CFA Loadings for the Best Fitting Model from Table 7

		Irritability		Headstrong Behavior	
CBCL item	Item Content	β	*SE*	β	*SE*
28	Breaks rules at home, school, or elsewhere			.92	.01
22	Disobedient at home			.87	.02
23	Disobedient at school			.83	.02
03	Argues a lot			.78	.02
39	Hangs around with others who get in trouble			.29	.04
86	Stubborn, sullen, or irritable	.88	.02		
95	Temper tantrums or hot temper	.85	.02		
87	Sudden changes in mood or feelings	.77	.02		
68	Screams a lot	.44	.04		
37	Gets in many fights	.41	.04		
45	Nervous, high-strung, or tense	.29	.04		

Note. Factor correlation between Irritability and Headstrong = .80

Appendix I

Table 9

Hierarchical Regression Models for Clinician Predicted Internalizing Scales

Dependent Variable	Model	Predictor	b [95% CI]	*p*	ΔR^2	Model Test
Anxious/Depression	1	Gender	1.19 [.33, 1.04]	.01	.04	<.001
		Age	-.32 [-.44, -.19]	<.001		
		Irritability	.21 [.02, .41]	.03		
	2	Gender	1.16 [.30, 2.02]	.01	.00	.31
		Age	-.32 [-.45, -.20]	<.001		
		Irritability	.32 [.04, .59]	.03		
		Headstrong	-.10 [-.30, .09]	.31		
	3	Gender	1.13 [.27, 1.99]	.01	.00	.21
		Age	-.32 [-.45, -.20]	<.001		
		Irritability	-.18 [-.99, .63]	.67		
		Headstrong	-.41 [-.93, .11]	.13		
		Irritability by Headstrong	.05 [-.03, .12]	.21		
Affective Problems	1	Gender	.93 [.03, 1.83]	.04	.01	.01
		Age	-.12 [-.25, .01]	.06		
		Irritability	.21 [.01, .42]	.04		
	2	Gender	.91 [.01, 1.81]	.05	.00	.47
		Age	-.13 [-.26, .00]	.05		
		Irritability	.29 [-.00, .58]	.05		
		Headstrong	-.08 [-.28, .13]	.47		
	3	Gender	.89 [-.01, 1.79]	.05	.00	.51
		Age	-.13 [-.26, .00]	.05		
		Irritability	.02 [-.83, .86]	.97		
		Headstrong	-.25 [-.79, .30]	.38		
		Irritability by Headstrong	.03 [-.05, .10]	.51		
Withdrawn/Depression	1	Gender	.53 [-.02, 1.07]	.06	.01	.19
		Age	.03 [-.05, .11]	.48		
		Irritability	.05 [-.07, .18]	.38		
	2	Gender	.49 [-.06, 1.03]	.08	.00	.02
		Age	.02 [-.06, .10]	.65		
		Irritability	.21 [.03, .38]	.02		
		Headstrong	-.15 [-.28, -.03]	.02		
	3	Gender	.48 [-.07, 1.03]	.09	.00	.78
		Age	.02 [-.06, .10]	.65		
		Irritability	.14 [-.38, .65]	.60		
		Headstrong	-.19 [-.52, .14]	.25		
		Irritability by Headstrong	.01 [-.04, .05]	.78		

Table 9 (continued).

Dependent Variable	Model	Predictor	b [95% CI]	*p*	ΔR^2	Model Test
Somatization	1	Gender	.38 [.01, .74]	.04	.01	.02
		Age	-.04 [-.09, .01]	.10		
		Irritability	.07 [-.01, .15]	.11		
	2	Gender	.36 [-.00, .73]	.05	.00	.18
		Age	-.05 [-.10, .01]	.08		
		Irritability	.12 [.01, .24]	.04		
		Headstrong	-.06 [-.14, .03]	.18		
	3	Gender	.35 [-.02, .71]	.06	.00	.15
		Age	-.05 [-.10, .01]	.08		
		Irritability	-.11 [-.45, .23]	.52		
		Headstrong	-.20 [-.42, .01]	.06		
		Irritability by Headstrong	.02 [-.01, .05]	.15		
Anxiety Problems	1	Gender	.71 [.14, 1.28]	.01	.04	<.001
		Age	-.24 [-.32, -.15]	<.001		
		Irritability	.12 [-.01, .24]	.08		
	2	Gender	.70 [.13, 1.27]	.02	.00	.49
		Age	-.24 [-.32, -.16]	<.001		
		Irritability	.16 [-.02, .34]	.09		
		Headstrong	-.05 [-.18, .08]	.49		
	3	Gender	.68 [.10, 1.25]	.02	.01	.16
		Age	-.24 [-.32, -.16]	<.001		
		Irritability	-.20 [-.73, .34]	.47		
		Headstrong	-.27 [-.61, .07]	.12		
		Irritability by Headstrong	.03 [-.01, .08]	.16		

Appendix J

Table 10

Hierarchical Regression Models for Clinician Predicted Externalizing Scales

Dependent Variable	Model	Predictor	b [95% CI]	*p*	ΔR^2	Model Test
Aggression	1	Gender	.02 [-.66, .71]	.94	.18	<.001
		Age	-.39 [-.49, -.29]	<.001		
		Irritability	.84 [.68, .99]	<.001		
	2	Gender	.18 [-.49, .84]	.60	.05	<.001
		Age	-.36 [-.45, -.26]	<.001		
		Irritability	.25 [.03, .46]	.02		
		Headstrong	.59 [.44, .74]	<.001		
	3	Gender	.15 [-.51, .82]	.65	.01	.23
		Age	-.36 [-.45, -.26]	<.001		
		Irritability	-.11 [-.74, .51]	.73		
		Headstrong	.37 [-.04, .77]	.08		
		Irritability by Headstrong	.03 [-.02, .09]	.23		
ODD Problems	1	Gender	-.15 [-.54, .24]	.45	.25	<.001
		Age	-.01 [-.07, .04]	.61		
		Irritability	.59 [.50, .68]	<.001		
	2	Gender	-.06 [-.44, .32]	.75	.01	<.001
		Age	.01 [-.05, .06]	.78		
		Irritability	.24 [.12, .36]	<.001		
		Headstrong	.35 [.26, .43]	<.001		
	3	Gender	-.04 [-.42, .34]	.84	.01	.05
		Age	.01 [-.05, .06]	.77		
		Irritability	.57 [.22, .92]	.001		
		Headstrong	.55 [.33, .77]	<.001		
		Irritability by Headstrong	-.03 [-.06, -.00]	.05		
Rule Breaking	1	Gender	-.14 [-1.06, .78]	.77	.04	<.001
		Age	-.25 [-.38, -.11]	<.001		
		Irritability	.51 [.30, .72]	<.001		
	2	Gender	-.06 [-.98, .86]	.90	.01	.01
		Age	-.23 [-.36, -.09]	.001		
		Irritability	.22 [-.08, .51]	.15		
		Headstrong	.29 [.09, .50]	.01		
	3	Gender	-.09 [-1.01, .83]	.85	.01	.32
		Age	-.23 [-.36, -.10]	<.001		
		Irritability	-.20 [-1.07, .68]	.66		
		Headstrong	.04 [-.51, .59]	.90		
		Irritability by Headstrong	.04 [-.04, .12]	.32		

Table 10 (continued).

Dependent Variable	Model	Predictor	b [95% CI]	*p*	ΔR^2	Model Test
ADHD Problems	1	Gender	-.64 [-1.19, -.10]	.02	.08	<.001
		Age	-.16 [-.24, -.08]	<.001		
		Irritability	.42 [.30, .55]	<.001		
	2	Gender	-.51 [-1.03, .02]	.06	.08	<.001
		Age	-.12 [-.20, -.05]	.002		
		Irritability	-.11 [-.28, .06]	.22		
		Headstrong	.53 [.41, .65]	<.001		
	3	Gender	-.49 [-1.02, .03]	.07	.00	.35
		Age	-.12 [-.20, -.05]	.002		
		Irritability	.11 [-.38, .60]	.66		
		Headstrong	.67 [.35, .98]	<.001		
		Irritability by Headstrong	-.02 [-.07, .02]	.35		
Attention Problems	1	Gender	-1.14 [-1.76, -.52]	<.001	.07	<.001
		Age	-.14 [-.23, -.05]	.002		
		Irritability	.38 [.24, .52]	<.001		
	2	Gender	-1.02 [-1.63, -.42]	<.001	.04	<.001
		Age	-.11 [-.20, -.03]	.01		
		Irritability	-.07 [-.27, .13]	.48		
		Headstrong	.45 [.31, .59]	<.001		
	3	Gender	-1.02 [-1.63, -.41]	.001	.00	.84
		Age	-.11 [-.20, -.03]	.01		
		Irritability	-.02 [-.58, .55]	.96		
		Headstrong	.49 [.12, .85]	.01		
		Irritability by Headstrong	-.01 [-.06, .05]	.84		

Appendix K

Table 11

Hierarchical Regression Models for Caregiver Predicted Internalizing Disorders

Dependent Variable	Model	Predictor	Odds ratio [95% CI]	*p*	Δ Pseudo-R^2	Model Test
Bipolar Disorder	1	Gender	1.26 [.87, 1.82]	.23	.06	<.001
		Age	1.03 [.98, 1.09]	.28		
		Irritability	1.23 [1.15, 1.31]	<.001		
	2	Gender	1.22 [.84, 1.78]	.30	.00	.24
		Age	1.03 [.98, 1.09]	.26		
		Irritability	1.27 [1.17, 1.37]	<.001		
		Headstrong	.95 [.87, 1.03]	.24		
	3	Gender	1.22 [.84, 1.78]	.99	.00	.63
		Age	1.03 [.98, 1.09]	<.001		
		Irritability	1.35 [1.02, 1.79]	.32		
		Headstrong	1.03 [.74, 1.43]	.67		
		Irritability by Headstrong	.99 [.97, 1.02]	.37		
Suicide	1	Gender	1.36 [1.00, 1.83]	.05	.10	<.001
		Age	1.20 [1.14, 1.25]	<.001		
		Irritability	1.11 [1.05, 1.16]	<.001		
	2	Gender	1.27 [.93, 1.72]	.13	.01	.002
		Age	1.20 [1.14, 1.25]	<.001		
		Irritability	1.18 [1.10, 1.25]	<.001		
		Headstrong	.90 [.84, .96]	.002		
	3	Gender	1.26 [.93, 1.71]	.14	.00	.29
		Age	1.20 [1.14, 1.25]	<.001		
		Irritability	1.07 [.88, 1.29]	.52		
		Headstrong	.80 [.64, 1.00]	.05		
		Irritability by Headstrong	1.01 [.99, 1.03]	.29		

Table 11 (continued).

Dependent Variable	Model	Predictor	Odds ratio [95% CI]	*p*	Δ Pseudo-R^2	Model Test
PTSD	1	Gender	2.78 [1.63, 4.72]	<.001	.02	<.001
		Age	1.04 [.96, 1.12]	.33		
		Irritability	1.06 [.98, 1.15]	.16		
	2	Gender	2.86 [1.68, 4.88]	<.001	.01	.44
		Age	1.04 [.96, 1.12]	.33		
		Irritability	1.03 [.93, 1.15]	.54		
		Headstrong	1.05 [.93, 1.12]	.44		
	3	Gender	2.87 [1.68, 4.90]	<.001	.00	.51
		Age	1.04 [.96, 1.12]	.31		
		Irritability	1.15 [.82, 1.64]	.42		
		Headstrong	1.19 [.80, 1.76]	.39		
		Irritability by Headstrong	.99 [.96, 1.02]	.51		
Generalized Anxiety Disorder	1	Gender	1.70 [.90, 3.20]	.10	.004	.36
		Age	1.02 [.93, 1.11]	.74		
		Irritability	1.01 [.91, 1.11]	.91		
	2	Gender	1.55 [.82, 2.94]	.18	.005	.04
		Age	1.01 [.93, 1.11]	.75		
		Irritability	1.09 [.96, 1.24]	.18		
		Headstrong	.86 [.75, .99]	.04		
	3	Gender	1.54 [.82, 2.94]	.19	.00	.51
		Age	1.01 [.92, 1.11]	.78		
		Irritability	.97 [.68, 1.39]	.89		
		Headstrong	.75 [.48, 1.17]	.20		
		Irritability by Headstrong	1.01 [.98, 1.05]	.50		

Table 11 (continued).

Dependent Variable	Model	Predictor	Odds ratio [95% CI]	*p*	Δ Pseudo-R^2	Model Test
Unipolar Depression	1	Gender	1.49 [1.08, 2.04]	.02	.06	<.001
		Age	1.16 [1.11, 1.22]	<.001		
		Irritability	1.00 [.95, 1.05]	.89		
	2	Gender	1.39 [1.00, 1.92]	.05	.01	.004
		Age	1.16 [1.11, 1.22]	<.001		
		Irritability	1.06 [.99, 1.13]	.09		
		Headstrong	.90 [.84, .97]	.004		
	3	Gender	1.39 [1.00, 1.92]	.05	.00	.74
		Age	1.16 [1.11, 1.22]	<.001		
		Irritability	1.02 [.84, 1.25]	.81		
		Headstrong	.87 [.69, 1.09]	.22		
		Irritability by Headstrong	1.00 [.99, 1.02]	.74		

Appendix L

Table 12

Hierarchical Regression Models for Caregiver Predicted Externalizing Disorders

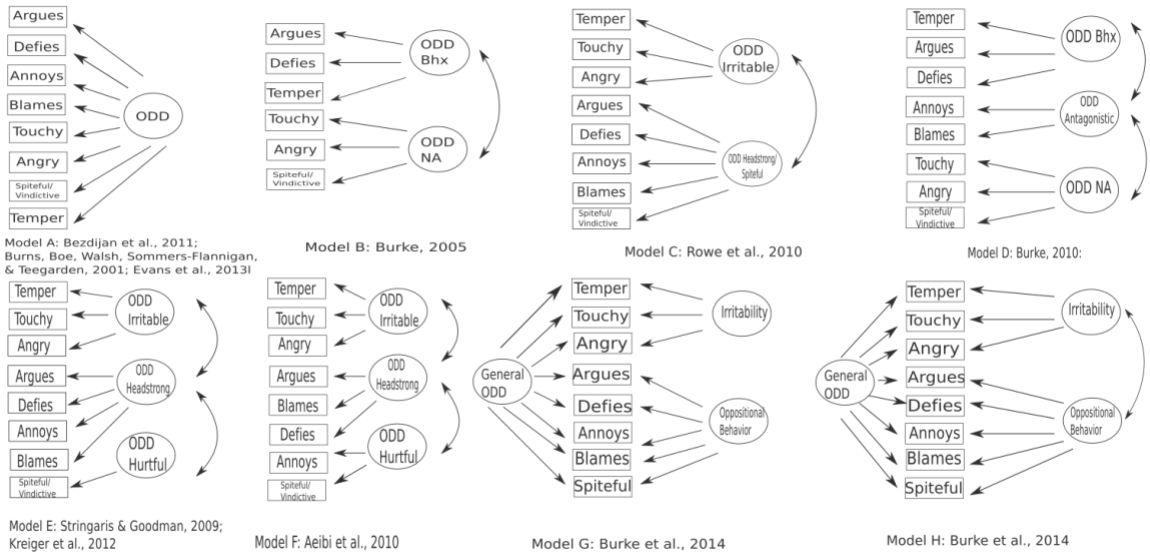
Dependent Variable	Model	Predictor	Odds ratio [95% CI]	*p*	Δ Pseudo-R^2	Model Test
Conduct Disorder	1	Gender	.69 [.43, 1.09]	.11	.09	<.001
		Age	1.28 [1.18, 1.38]	<.001		
		Irritability	1.28 [1.18, 1.39]	<.001		
	2	Gender	.77 [.48, 1.24]	.29	.02	<.001
		Age	1.28 [1.19, 1.38]	<.001		
		Irritability	1.16 [1.05, 1.27]	.004		
		Headstrong	1.22 [1.09, 1.36]	<.001		
	3	Gender	.77 [.48, 1.24]	.29	.00	.38
		Age	1.28 [1.19, 1.38]	<.001		
		Irritability	.98 [.68, 1.42]	.93		
		Headstrong	1.02 [.69, 1.51]	.90		
		Irritability by Headstrong	1.02 [.98, 1.04]	.37		
ADHD	1	Gender	.27 [.19, .38]	<.001	.21	<.001
		Age	.84 [.79, .88]	<.001		
		Irritability	1.24 [1.17, 1.31]	<.001		
	2	Gender	.29 [.21, .41]	<.001	.04	<.001
		Age	.83 [.79, .87]	<.001		
		Irritability	1.09 [1.02, 1.17]	.01		
		Headstrong	1.29 [1.19, 1.39]	<.001		
	3	Gender	.29 [.21, .41]	<.001	.01	.06
		Age	.83 [.79, .87]	<.001		
		Irritability	1.35 [1.07, 1.71]	.01		
		Headstrong	1.65 [1.25, 2.17]	<.001		
		Irritability by Headstrong	.98 [.96, 1.00]	.06		
ODD	1	Gender	.80 [.59, 1.09]	.16	.11	<.001
		Age	.91 [.87, .96]	<.001		
		Irritability	1.23 [1.17, 1.30]	<.001		
	2	Gender	.87 [.63, 1.19]	.37	.01	<.001
		Age	.91 [.87, .95]	<.001		
		Irritability	1.15 [1.08, 1.22]	<.001		
		Headstrong	1.15 [1.07, 1.23]	<.001		
	3	Gender	.87 [.63, 1.19]	.38	.01	.001
		Age	.91 [.87, .95]	<.001		
		Irritability	1.75 [1.34, 2.29]	<.001		
		Headstrong	1.82 [1.35, 2.45]	<.001		
		Irritability by Headstrong	.96 [.94, .99]	.001		

Table 12 (continued).

Dependent Variable	Model	Predictor	Odds ratio [95% CI]	*p*	Δ Pseudo-R^2	Model Test
Substance Use	1	Gender	.95 [.49, 1.87]	.89	.08	<.001
		Age	1.60 [1.37, 1.87]	<.001		
		Irritability	1.02 [.91, 1.14]	.72		
	2	Gender	1.02 [.52, 2.02]	.95	.00	.22
		Age	1.61 [1.38, 1.89]	<.001		
		Irritability	.97 [.84, 1.11]	.61		
		Headstrong	1.10 [.94, 1.27]	.23		
	3	Gender	1.00 [.51, 1.99]	.99	.00	.37
		Age	1.61 [1.37, 1.89]	<.001		
		Irritability	.81 [.53, 1.23]	.32		
		Headstrong	.91 [.58, 1.41]	.67		
		Irritability by Headstrong	1.02 [.98, 1.05]	.37		

Appendix M

Figure 1. Competing Models for Analysis



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Curriculum Vitae

Breanna Garcia

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Education

University of Nevada, Las Vegas, Las Vegas, NV — Expected 2021

Doctor of Philosophy in Psychology

Advisor: Andrew Freeman, Ph.D.

San Jose State University, San Jose, CA — 2015

Bachelor of Arts in Psychology

Psychology GPA: 4.0, Cumulative GPA: 3.95

Awards/Grants

Travel Award | UNC Mood and Sleep Conference — 2015

Travel Award | ABCT — 2018

Access Grant | University of Nevada, Las Vegas — 2016; 2017; 2018

Research Experience

UNLV Department of Psychology

Advisor: Andrew Freeman, Ph.D.

Graduate Assistant 2015-Present

- Projects: Factor structure of oppositional defiant disorder in clinical samples.
- Responsibilities: Assist Dr. with running the DIME Lab and conducting research. Specific responsibilities include administrative duties within the lab, data management, statistical analyses of the data, programming, running participants, and supervision of undergraduate research assistants.

SJSU Department of Psychology

Advisor: Joanna Fanos, Ph.D.

Undergraduate Research Assistant 2013-2015

- Projects: Implementation of a peer support group for college football freshmen; Parental response to diagnosis of their child of Congenital Critical Heart Disease via newborn screening
- Responsibilities: Assist with literature reviews, development of interview guides, coding interviews, and data analysis

Clinical Experience

Mobile Crisis June 2018- Present

Las Vegas, Nevada

Primary Supervisor: Megan Freeman, Ph.D.

Doctoral Practicum Student

- Provided evidence-based assessment and treatment to children and adolescents experiencing acute crises. A cognitive-behavioral orientation was utilized along with motivational and problem-solving techniques. Services were provided to a diverse demographic. Diagnoses included severe mood, disruptive behavior, and schizophrenic spectrum disorders. Assessments focused on determination of whether acute hospitalization was necessary. Treatment team meetings were held twice per week. Received weekly individual supervision.

Desert Willow Treatment Center August 2017-June 2018

Las Vegas, Nevada

Primary Supervisor: Caron Evans, Ph.D.

Doctoral Practicum Student

- Provided evidence-based assessment and treatment to children and adolescents within an inpatient psychiatric setting. A cognitive-behavioral orientation was utilized along with

motivational and problem-solving techniques. Services were provided to a diverse demographic. Diagnoses included severe mood, disruptive behavior, and schizophrenic spectrum disorders. Comprehensive assessments focused primarily on assessing intelligence, personality, and adaptive functioning so as to inform treatment planning and referral placements. Integrated treatment team meetings were held twice per week. Received weekly individual supervision.

The PRACTICE: A UNLV Community Mental Health Center August 2016-August 2017

University of Nevada, Las Vegas

Primary Supervisor: Andrew Freeman, Ph.D.

Doctoral Practicum Student

- Provided evidence-based assessment and manualized intervention to a caseload of 5-9 clients. A cognitive-behavioral orientation was utilized along with motivational interviewing and problem-solving techniques. Services were provided to diverse populations. The majority of clients were children and adolescents between the ages of 2-16 years and their families. Diagnoses included both externalizing (attention-deficit/hyperactivity disorder and oppositional defiant disorder) and internalizing (major depressive disorder, persistent depressive disorder, disruptive mood dysregulation disorder, posttraumatic stress disorder, obsessive-compulsive disorder, trichotillomania, generalized anxiety disorder, and social anxiety disorder) disorders. Comprehensive assessments focused on differential diagnosis, developing treatment plans, and providing

applicable referrals. Case presentations were given during group supervision. Received weekly individual supervision.

Professional Training

Linear Regression Workshop Andrew Freeman, Ph.D. May 2017

4-day training on regression analyses, utilization of R technology, and interpreting results

Interprofessional Education Various Presenters March 2017

1-day training on interprofessional patient care and poverty simulation

Interprofessional Education Various Presenters March 2018

1-day training on interprofessional patient care and poverty simulation

Teaching Experience

Graduate Student Instructor August 2017-Present

University of Nevada, Las Vegas

Foundations of Developmental Psychology: Infant and Child

- Teaching two sections of undergraduate foundations of developmental psychology: infants to childhood. Educational goals of the class include developing an understanding

of human development from conception to middle childhood. Topics include physical, cognitive, and social/emotional development.

General Psychology PSY 101

- Teaching two sections of an undergraduate introductory psychology course per semester. Educational goals of the class include developing an understanding of the discipline of psychology, developing scientific values and skills, fostering personal growth, and enhancing library and computer skills. Duties include developing lecture, lecturing weekly, developing examinations, grading, providing student feedback, linking students to applicable services, and providing at least two office hours a week.

Teaching Assistant January 2017-May 2017

University of Nevada, Las Vegas

Professor: Andrew Freeman, Ph.D.

Child Assessment PSY 712

- Grading student assignments and performing miscellaneous administrative duties.

Teaching Assistant August 2015-May 2016

University of Nevada, Las Vegas

Professor: Kristen Culbert, Ph.D.

Health Psychology PSY 412

- Administered exams, grading exams, grading student assignments, and performing miscellaneous administrative duties.

Mentoring Experience

Graduate Student Mentor 2015-2016

University of Nevada, Las Vegas

Outreach Undergraduate Mentoring Program (OUMP)

- The purpose of OUMP is to provide mentorship to undergraduate psychology students from under-represented backgrounds in order to increase student retention and graduate school applications. Duties include one-on-one mentoring, linking students to resources (e.g., faculty, contacts, research experience, etc.), providing CV development, editing application materials, guiding career planning, and attending mentoring training. Workshops are also provided by mentors for the entire program.

Service to the University

- **Peer Mentor**| Student-Athlete Success Services at SJSU 2014 – 2015

Professional Development

- **Writing Workshop** | Department of Psychology; University of Nevada, Las Vegas 2016

- Semester long writing workshop aimed at enhancing professional writing skills pertaining to grants and manuscripts submitted to peer-reviewed journals.

Presentations

Freeman, A. J., Garcia, B., A., Findling, R. L., & Youngstrom, E. A. (2017, November). *Irritability and noncompliant symptoms reduce quality of life.* Symposium submitted to the Annual Convention of the Association of Behavioral and Cognitive Therapies, San Diego, CA.

Garcia, B. A., & Freeman, A. J. (2016, October). *Aggression to depression: Examining moderators of emotion dysregulation.* Poster session presented to the Annual Convention of the Association of Behavioral and Cognitive Therapies, New York City, NY.

Garcia, B. A., Sherwood, S. N., & Freeman, A. J. (2018, November). *Mood symptoms to aggression: Irritability as a moderator.* Poster session submitted to the Annual Convention of the Association of Behavioral and Cognitive Therapies, Washington, DC.

Millwood, S. N., Saucedo, M., Garcia, B. A., & Freeman, A. J. (2016, October). *Bipolar disorder substance use: Examining drug preference and frequency.* Poster session presented at the Annual Convention of the Association of Behavioral and Cognitive Therapies, New York City, NY.

Sherwood, S.N., Garcia, B.A., Cachero, A., & Freeman, A.J. (2018), *Sleep chronotype, mood, and irritability.* Poster session submitted to the Annual Convention of the Association of Behavioral and Cognitive Therapies, Washington, DC.

Work Experience

- **Graduate Assistant** | Department of Psychology 2015-Present
- **Peer Mentor**| Student-Athlete Success Services at SJSU 2014 – 2015

Professional Affiliations

- Association of Behavioral and Cognitive Therapies 2015-Present
- Nevada Psychological Association 2015-Present
- American Psychological Association, Division 18 2015-Present