Disruptive Mood Dysregulation Disorder: A Polyvagal Perspective

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Abstract

The present study was designed to contribute to the existing body of knowledge on Disruptive Mood Dysregulation Disorder (DMDD) by exploring the potential association between heart rate variability and the externalising symptoms (i.e. frequent temper tantrums and irritable or angry moods between outbursts) of the disorder. The overarching goal was to establish whether the emotion dysregulation and compromised social behaviour associated with DMDD are a function of a compromised social engagement system, as proposed by the polyvagal theory. This study compared data from a group of children (n = 15) who were diagnosed with DMDD to that of a group of typically developing controls (n = 15) to test four hypotheses derived from the polyvagal theory: 1) Children with DMDD would have significantly lower RSA amplitude than controls; 2) RSA amplitude would be significantly related to speed and accuracy of attention shifting (Affective Posner Cueing Task) and emotion recognition (Dynamic Affect Recognition Evaluation) tasks – lower RSA amplitude was expected to be associated with more errors and reduced reaction time during emotional, frustrating and emotion recognition tasks in children with DMDD; 3) RSA amplitude would be significantly related to attachment style (as measured by the Attachment Style Classification Questionnaire) – lower amplitude RSA was anticipated to be related to anxious/ambivalent or avoidant attachment styles; and 4) RSA amplitude would be significantly related to prosody – reduced RSA amplitude was predicted to be correlated with reduced acoustic modulation in children with DMDD. Results did not fully confirm the hypothesis that children who have been diagnosed with DMDD exhibit lower baseline RSA and excessive reductions in RSA in response to emotionally evocative stimuli compared to healthy controls. Although not statistically significant, dissimilar trends emerged for the two groups in terms of RSA trajectories, and warrant further investigation. Results partially supported the hypothesis that participants in the DMDD group would exhibit reduced

speed and accuracy during the Affective Posner Cueing Task and the DARE emotion recognition task. Children in the DMDD group were significantly slower (p = .002) to respond during all three conditions of the Affective Posner Cueing Task and had significantly impaired ability (p = .032) to accurately recognise fear during the DARE task, in comparison with healthy controls. Participants in the DMDD group were found to largely have an avoidant attachment style. Respiratory sinus arrhythmia was, however, related to anxious ambivalent attachment in general. Statistically significant (p = .044) differences also emerged in terms of prosody, with participants in the DMDD group exhibiting more modulation, or instability, than those in the control group. Finally, the number of social interaction problems reported by parents of participants in the DMDD group significantly correlated with vagal tone during the frustration condition (p = .049) of the Affective Posner Cueing Task. These findings are discussed in terms of their implications and relation to findings of other studies. In conclusion, the limitations of the current study are addressed and recommendations for future research are made.

Keywords: Disruptive Mood Dysregulation Disorder, DMDD, RSA, vagal tone, polyvagal

Declaration

I declare that this dissertation is my own, unaided work. It is being submitted for the degree of Doctor of Philosophy at the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at any other university.

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Michelle Leal

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Dedication

To Aeden and Anja. Reach for the sky.

Table of Contents

List of Figures

List of Tables

Chapter 1: Introduction

Disruptive Mood Dysregulation Disorder (DMDD) was included as a new diagnostic category in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*; American Psychiatric Association [APA], 2013a) to attend to concerns regarding the misdiagnosis of paediatric bipolar disorder and overestimation of its prevalence. In 1995, Wozniak and colleagues broached the issue of widespread failure to diagnose paediatric bipolar disorder in clinical practice (Wozniak et al., 1995). Subsequently, clinical attention to the disorder has improved significantly, and data indicates a pronounced rise in the rates at which children have been diagnosed with paediatric bipolar disorder. In the United States of America (USA), paediatric bipolar disorder diagnosis in outpatients increased from 0.42% to 6.67% between 1994 and 2003 (Moreno et al., 2007). Between 1996 and 2004, inpatient diagnosis of paediatric bipolar disorder rose from 1.3 to 7.3 per 10 000 children. Bipolar related discharges in teenagers showed a comparable surge, with an almost 300% increase from 1996 to 2004 (Blader & Carlson, 2007). According to Kessing, Vradi, and Andersen (2014), similar trends have been observed in clinical practice in Europe, but theirs was the first study outside of the USA that examined annual rates of incidence of paediatric bipolar disorder. In Denmark, the yearly rates of paediatric bipolar disorder increased between 100% and 300% during the study interval of 1995 to 2012 (Kessing et al., 2014). This upsurge in diagnoses was accompanied by a proliferation of publications. Listings on PubMed indicate that more articles were published on paediatric bipolar disorder in January 2008 (22 articles) than the decade from 1986 to 1996 (15 articles; Leibenluft, 2008). In the following two decades, the number of publications soared: from 1997 to 2007 it grew to 294 and from 2008 to present, search results list a total of 654 articles.

Statistics obtained from the Council for Medical Schemes (CMS, 2015) suggest that this tendency is mirrored in South Africa. Between 2007 and 2015, the prevalence for treated

bipolar mood disorder (BMD) in females has increased from 0.01 to 0.10 (900%) for ages 5-9, 0.09 to 0.48 (433%) for ages 10-14, and 0.54 to 2.39 (343%) for ages 15-19 per 1000 medical aid beneficiaries (see Figure 1).

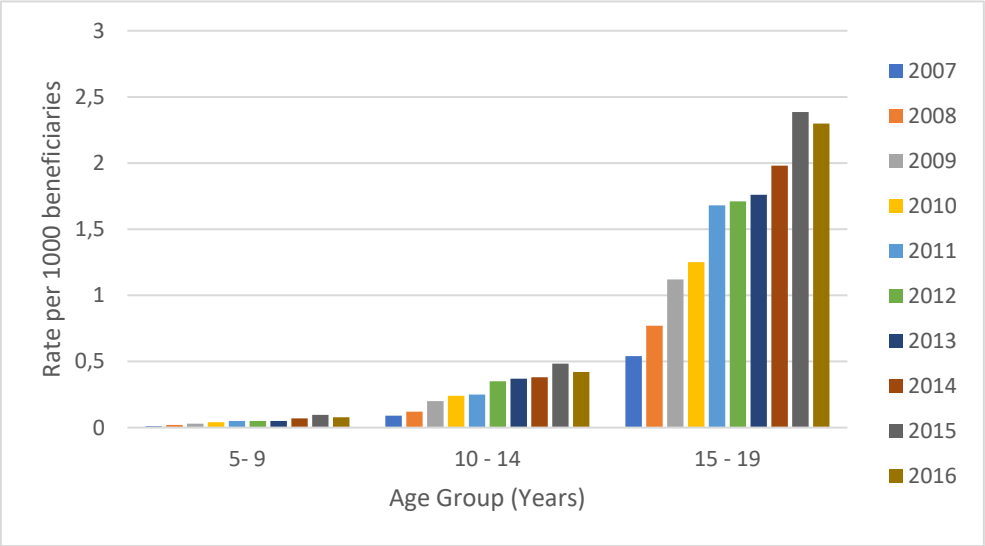

Figure 1. Paediatric bipolar mood disorder prevalence in females on medical schemes in South Africa. *Note:* Statistics obtained from the Council for Medical Schemes

In males, for the same time period, the prevalence for treated BMD increased from 0.19 to 0.26 (36%) for ages 5-9, 0.36 to 0.78 (117%) for ages 10-14, and 0.88 to 1.85 (110%) for ages 15-19 per 1000 beneficiaries (see Figure 2).

Noteworthy is that, in females, the trend in increase continued until 2015 and then dropped only slightly in 2016 (at the time of writing, statistics for 2017 were not available yet). In contrast, in boys, the upward trend peaked in 2014 and then saw a pronounced drop in 2015 (-55%, -44% and -45% for each age group respectively) and a further slight decrease in 2016 (-23%, -12% and -2% respectively).

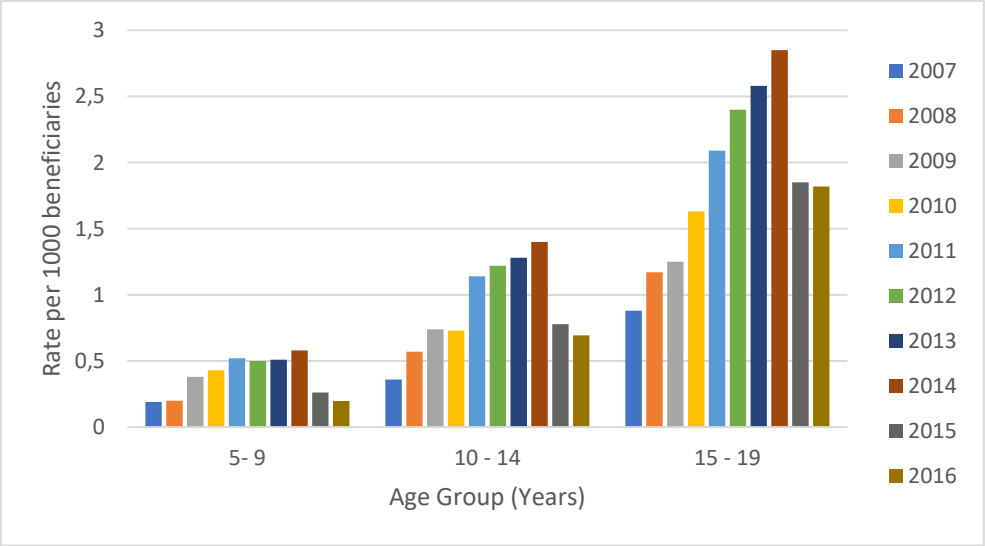

Figure 2. Paediatric bipolar mood disorder prevalence in males on medical schemes in South Africa. *Note:* Statistics obtained from the Council for Medical Schemes

Further research to explore the reason for this sharp decrease would be beneficial to our understanding of the current interpretation and diagnosis of mood and behavioural dysfunction in South African children. Possibilities include an actual decline in prevalence, the result of increased awareness of over- or misdiagnoses among clinicians, a change in inclination towards the diagnosis of behavioural or conduct disorders in boys due to a gender difference in the manifestation of symptoms, and the introduction of the new diagnostic category.

Although percentage increases are higher for child and adolescent females, in the same age groups, more males are being treated for BMD. This is the opposite of trends in adult beneficiaries, where prevalence rates for treatment are higher in adult female beneficiaries than in adult male beneficiaries (CMS, 2015). It should be noted that the trend above may be explained by the increased likelihood of females to seek medical treatment compared to their male counterparts, as lifetime male-to-female prevalence is roughly equal (APA, 2013a).

It is not yet clear whether these trends reflect an actual proliferation in prevalence, improved clinical attention and case identification, or a contentious broadening of the bipolar disorder construct, in particular, the inclusion of children and adolescents who exhibit severe irritability without distinct hypomanic or manic episodes (Leibenluft, 2011).

Evidence suggests that the disorder is sometimes also misdiagnosed. One study of adolescent inpatients found that clinical chart diagnoses of mania in adolescents were far more common than research-quality diagnoses. More than half of paediatric bipolar cases diagnosed clinically were reclassified during the research procedure as either depression or conduct disorder, having presented with depression and hostility rather than elation (Pogge et al., 2001).

Clinical heterogeneity may provide another explanation for the higher prevalence estimation. For example, typical manifestation of attention-deficit/hyperactivity disorder (ADHD), such as distractibility and hyperactivity, are also diagnostic criteria of mania (Moreno et al., 2007). In addition, although not a diagnostic criterion for ADHD, which frequently co-occurs with DMDD, irritability, and difficulties in the regulation of emotional reactivity and behavioural state, is common in children with ADHD (Leibenluft, 2011). The erroneous diagnoses of these severely impaired children who present symptoms similar to that of bipolar disorder and ADHD, but do not meet the exact diagnostic criteria of either disorder, could be a factor in the higher incidence of paediatric bipolar disorder. In South Africa, as in the USA (Blader & Carlson, 2007), the prevalence increase seems to be associated, at least in part, with “up-coding” or prescribed minimum benefits (PMB) creep. Prescribed minimum benefits are a set of defined benefits that medical aid schemes are obliged to cover, regardless of the members’ benefit option (Watson, 2015). As defined by the Department of Health (DOH), PMB creep is the erroneous diagnosis of an illness or disorder to match a specific PMB category that is slightly more severe than the actual case to

ensure payment to the provider and coverage of treatment for the patient. Watson (2015) cites the example, given by the DOH and one of the leading medical aid providers in the country, of the diagnosis of bipolar disorder, which is on the chronic disease list and thus eligible for PMB coverage, instead of other forms of depression, which are not on the PMB list. It is also possible that bipolar disorder is used as diagnosis because a more appropriate diagnostic category for chronic, debilitating irritability, such as DMDD, is lacking in the South African health system. The International Statistical Classification of Diseases and Related Health Problems, 10th Revision code (ICD-10; World Health Organisation [WHO], 1992) does not have a code for DMDD that could be implemented by medical schemes - and without the diagnosis, access to mental health care services and treatment would be limited.

Although the exact reasons for the dramatic increase in the prevalence of paediatric bipolar disorder remain unclear, the distinction between this diagnostic category and DMDD is an important one. While levels of impairment are comparable (Leibenluft, 2011), research suggests there are significant differences between the two disorders, which may have significant implications for clinical practice and treatment. Extensive research has found, for example, that children with severe, chronic irritability are more prone to develop depression and generalised anxiety disorder than bipolar disorder later in life (Brotman et al., 2006; Brotman et al., 2007; Stringaris, Cohen, Pine, & Leibenluft, 2009). Studies also suggest that DMDD shares biological pathways with depression (Savage et al., 2015) and ADHD (Riglin et al., 2017). Medication used to treat these disorders varies significantly from that used to treat bipolar disorder (Dickstein et al., 2009; Kim & Boylan, 2016; Moreno et al., 2007; Tourian et al., 2015; Waxmonsky et al., 2008). Prematurely medicating young individuals may at best be ineffective in treating the debilitating symptoms and at worst be harmful. It is therefore imperative to develop evidence-based treatment and intervention strategies.

Due to its recent inclusion, however, there is a dearth of empirical data on DMDD. In an attempt to help address this gap in the literature, the current research explores the hypothesised relationship between the regulation of emotional reactivity and behavioural state, which is at the core of DMDD, and underlying neurophysiological processes, using the polyvagal theory (Porges, 1995, 1997, 1998, 2001) as a conceptual framework. This theory provides an understanding of social behaviour and, by extension, compromised social behaviour, from a neurobiological perspective (Porges, 2003a). It maintains that several psychiatric and behavioural disorders are characterised by insufficiencies in tasks related to social engagement, such as avoidant gaze and reduced facial expressiveness, and that these features often present as a cluster. From a psychopathological perspective, these disorders are seen to have different aetiologies, but from a polyvagal standpoint, there is a common neurophysiological component (Porges, 2011). Insight into this system's involvement in adaptive social behaviour and psychological experience allows us to predict relationships between its functioning and atypical social behaviour, which can be tested and used to inform diagnostic and treatment practices.

The polyvagal theory puts forward such a conceptual system, the social engagement system (Porges, 2001, 2003), which depicts the neurophysiological and neuroanatomical link between the regulation of visceral state (including the heart and bronchi) and the muscles of the head and face associated with social engagement behaviour. According to this model, the functioning of specific components of the mammalian autonomic nervous system (i.e., visceromotor and somatomotor) became integrated through evolutionary processes to form an intricate system that supports three distinct biobehavioural strategies – social engagement behaviours, mobilisation and immobilisation - in response to safety, danger and life-threatening cues in the environment. The theory suggests that these strategies follow a phylogenetically ordered hierarchy and each reflects a specialised neurophysiological

substrate to maximise the adaptive response. The visceromotor component includes pathways of the myelinated vagus nerve (also called the 10th cranial nerve) to the heart and inhibits sympathetic and hypothalamic-pituitary-adrenal (HPA) axis activity to calm autonomic state, which is necessary for prosocial behaviour. The somatomotor component includes the special visceral efferent (SVE) pathways that travel through cranial nerves V, VII, IX, X and XI. It regulates the muscles required for mastication and ingestion, head turning (to orient and gesture to others), looking behaviour, facial expression, discrimination of the human voice from background sounds and listening, vocalisation and intonation (to convey a message). An optimally functioning social engagement system supports an autonomic state and behaviours necessary for the successful initiation and maintenance of relations with others, such as voluntary and sustained attention, appropriate eye contact, gestures and facial expressions, listening, vocal communication, and the perception, projection and expression of emotion (Porges, 2001; Quintana, Kemp, Alvares, & Guastella, 2013). A compromised social engagement system, the theory predicts, would result in inadequate physiological state regulation and deficits in affect recognition and regulation, and thus difficulties in behavioural state regulation and fostering and maintaining social relationships.

These deficits are features of numerous psychiatric disorders, such as autism, social anxiety and post-traumatic stress disorder (Porges, 2003a). Attention deficit and conduct disorders, which frequently co-occur with DMDD, are characterised by difficulties in attention, listening, regulation of behaviour and social skills (Denver, 2004). A few studies have found evidence suggesting deficits in some of these areas in DMDD (e.g., Brotman et al., 2010; Deveney, Brotman, Decker, Pine, & Leibenluft, 2012; Guyer et al., 2007; Hommer et al., 2014; Leibenluft, 2011; Rich et al., 2010a), but data integrating these findings are lacking. Given the similarities of certain predicted characteristics of an impaired social engagement system and the core features of DMDD, the question whether DMDD represents

a disorder on the continuum of dysregulation within the social engagement system is a logical path of investigation. The current study was thus designed to test whether the emotion and behaviour dysregulation seen in DMDD is associated with a compromised social engagement system by assessing autonomic regulation, as indexed by the influence of the myelinated vagus on the heart (referred to as vagal tone).

The polyvagal theory (Porges, 1995, 1997, 1998, 2001) offers an alternative explanation of maladaptive social behaviour to that suggested by the symptom-based, nosological conceptualisation of mental health disorders. The latter view is increasingly considered not to reflect burgeoning knowledge of the relationship between neurophysiology and behaviour (Adam, 2013; Craddock & Owen, 2010). The phenotypic classification system is marred by high rates of comorbidity and similar diagnostic criteria as defining characteristics. For instance, in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM-IV*; American Psychiatric Association, 2000), irritability, which is one of the defining symptoms of DMDD, was included as a criterion for at least six childhood diagnostic categories, including major depressive disorder, generalised anxiety disorder, dysthymic disorder, post-traumatic stress disorder, oppositional defiant disorder, and manic episode. Similarly, impediments or atypical behaviour in social situations are characteristic of mood disorders, anxiety disorders and several child and adolescent psychiatric disorders (i.e., pervasive development disorders, learning disorders, communication disorders and attention/disruptive behaviour disorders). Furthermore, individuals suffering from these disorders struggle with tasks necessary to successfully engage with others, such as initiating and maintaining social relationships, expressing social behaviour and appropriate social communication (Denver, 2004). It is therefore conceivable that a single neurophysiological system, such as the social engagement system proposed by

the polyvagal theory, regulates the normal function of these behaviours and that disorders, such as DMDD, lie along a spectrum of dysfunction or dysregulation within that system.

The aims and hypotheses of the current study are directly drawn from the polyvagal theory of emotion and its model of the social engagement system. An interpretation of the diagnostic features of DMDD as emergent properties of deficits in the social engagement system provides an integrated neurobiological perspective of these symptoms. Also, it may inform intervention and treatment strategies to restore normal system function and may also help to identify a physiological variable that may serve as an early predictor of this disorder.

Chapter Two discusses the background of the new diagnostic category, outlines the features and diagnostic criteria of DMDD, and provides an overview of the prevalence, correlates, comorbidity, pathophysiology and treatment algorithms of DMDD.

Chapter Three examines the constructs of emotion and emotion regulation, which are critical to adaptive, healthy psychosocial functioning. It also explains how individuals develop stable styles of emotion regulation, primarily a result of attachment experiences with the primary caregiver, which may later become maladaptive and could lead to emotion dysregulation.

Chapter Four introduces the polyvagal theory, which articulates the mechanisms that mediate social engagement between individuals and provides a plausible explanation of how physiological state restricts the scope of affective and behavioural experience, and hence, determines contingent social behaviour. In conclusion, the research questions and hypotheses of the current study, which were derived from this theory, are discussed.

Chapter Five presents the methodology employed in the present study. The chapter introduces the aims of the research as derived from the review of available literature. The quasi-experimental research design is elaborated upon, followed by the sampling procedure and the demographic details of the final sample. There is a description of the psychometric

instruments utilised as the assessment battery, as well as the bespoke experimental hardware and setup. The procedure that was undertaken is detailed, and the analytic tools used for statistical analysis are briefly presented. The chapter ends with a description of the ethical considerations regarded necessary for the undertaking of this study.

Chapter Six comprises the exploration of the results and documents the statistical outcomes of the analyses. The descriptive statistics are outlined, after which significant and non-significant differences are subsequently explored and theorised.

Chapter Seven is a critical discussion of the most important results of the current study relative to the currently available literature and existing knowledge. The thesis concludes with the theoretical and practical contributions of the results of the current study to existing knowledge and explores the study's limitations with the identification of potential areas for future research.

Chapter 2: Disruptive Mood Dysregulation Disorder

As discussed in Chapter 1, Disruptive Mood Dysregulation Disorder (DMDD) is a new diagnostic category in the *DSM-5* (APA, 2013a) that was created to cater for children with non-episodic irritability and recurrent anger outbursts who fail to fully meet the criteria of existing disorders, but still experience significant impairment. Due to its novelty, and origin based on another disorder, empirical evidence regarding its prevalence, the comorbidity, course, prognosis, neurobiological correlates and potential treatment regimes of DMDD is only now emerging. The current research was designed with the aim to contribute to the limited, yet increasing, body of knowledge on the disorder. This chapter commences with an overview of the rationale that guided the creation of the new category and proceeds with a brief look at its features and diagnostic criteria as listed in the *DSM-5* (APA, 2013a). A thorough review of available literature on DMDD follows, focussing on prevalence and comorbidity, course and outcome, and pathophysiology. It then looks at how the disorder is currently treated and the studies that have tested these and novel approaches to alleviate symptoms in children with DMDD. The chapter concludes with a concise discussion of the criticism that ensued upon the creation of this category and emphasis on the importance of understanding the underlying mechanisms of DMDD.

Background

Disruptive Mood Dysregulation Disorder was introduced in the *DSM-5* (APA, 2013a) in response to the growing concern surrounding the marked increase in prevalence and potential over-diagnosis of bipolar disorder in children and adolescents. The proposal of the new category resulted in much controversy, firstly as some researchers and clinicians view severe, chronic irritability as a presentation of paediatric mania (Biederman et al., 2000; Wozniak et al., 1995); secondly because it was included in the *DSM-5* (APA, 2013a) without published clinical validity trials (Copeland, Angold, Costello, & Egger, 2013); and thirdly, in

field trials, inter-rater reliability varied significantly by setting (Margulies, Weintraub, Basile, Grover, & Carlson, 2012). Furthermore, support for the creation of this diagnosis is derived primarily from scientific studies on a syndrome proposed by the US National Institute of Mental Health (NIMH) called "severe mood dysregulation", which is similar to DMDD, but not identical (Leibenluft, 2011; Stringaris, & Goodman, 2009). In addition, the noteworthy comorbidity and symptom overlap with other psychiatric disorders (Axelson et al., 2012; Brotman et al., 2006; Copeland et al., 2013; Leibenluft, 2011), particularly oppositional defiant disorder, cast doubt as to whether or not DMDD should be a distinct category. The sizeable debate surrounding the inclusion of this proposed new diagnostic category, however, was overshadowed by the alarming increase in the off-label use of antipsychotics in children with severe, non-episodic irritability who had been diagnosed with bipolar disorder.

Both the *DSM-IV* (APA, 2000) and the *DSM-5* (APA, 2013a) specify that both adults and children must experience distinct episodes of mania or hypomania to satisfy the criteria for bipolar I disorder. The broadening of the category of "classic" bipolar to include non-episodic presentations of severe irritability could be a contributing factor in the significant increase in the prevalence of paediatric bipolar disorder (Grimmer, Hohmann, & Poustka, 2014; Moreno et al., 2007). To provide a structure for further research, Leibenluft, Charney, Towbin, Bhangoo, and Pine (2003) distinguished between "narrow" and "broad" phenotypes of bipolar disorder.

The narrow phenotype corresponded to the criteria as defined in the *DSM-IV* (APA, 2000). The broad phenotype was conceptualised as severe mood dysregulation and characterised by chronic, non-episodic irritability with severe rages, chronic hyperarousal (e.g., motor hyperactivity, distractibility) and negatively valenced mood (either sadness or anger) in between temper outbursts. It was anticipated that children who meet the criteria for the broad phenotype may or may not also fulfil the criteria for attention-deficit/hyperactivity

disorder (ADHD), major depressive disorder, oppositional defiant disorder and mania (Lebeinluft et al., 2003). Research did not, however, support the theory of severe mood dysregulation as a possible developmental manifestation of bipolar disorder. Longitudinal (Brotman et al., 2006; Stringaris et al., 2009) and familial (Brotman et al., 2007) studies suggest that non-episodic irritability in childhood predicts unipolar depression and anxiety disorders rather than adult bipolar disorder.

Brotman et al. (2007) postulated that, if severe mood dysregulation is a developmental phenotype of bipolar disorder, the rates of bipolar disorder among parents of children with severe mood dysregulation should be comparable to that of parents of children with narrow phenotype bipolar disorder. Using the Diagnostic Interview for Genetic Studies, parents of youths with bipolar disorder and youths with severe mood dysregulation were interviewed by clinicians who were blind to the children's diagnostic status. Compared to the parents of children with severe mood dysregulation (proband n = 30, parent n = 37), parents of youth with narrow phenotype bipolar disorder (proband n = 33, parent n = 42) were more likely to be diagnosed with bipolar disorder: 33.3% of children with narrow phenotype bipolar disorder had a parent with bipolar disorder, whereas only 2.7% of children with severe mood dysregulation had a parent with bipolar disorder. This study did, however, have various limitations, including small sample size, ascertainment bias, the fact that second-degree relatives were not assessed, the exclusion of comparison to parents of unaffected offspring, and the omission of assessment of ADHD and other disruptive behavioural disorders in the parents that may have differentiated the two groups.

Data extracted from the Great Smoky Mountains Study (GSMS), a longitudinal epidemiological study of children and adolescents in North Carolina, USA, suggested that severe mood dysregulation predicts risk for early adulthood depressive disorders (Brotman et al., 2006). Using the Child and Adolescent Psychiatric Assessment (CAPA), the researchers

determined the prevalence of severe mood dysregulation among the sample of 1420 children, ages 9 to 19 years. Severe mood dysregulation was found to be fairly common in childhood, with a lifetime prevalence of 3.3%. Compared to children who did not meet the criteria for severe mood dysregulation, those with severe mood dysregulation were significantly more likely to be diagnosed with a depressive disorder, such as major depressive disorder, dysthymia or depression not otherwise specified, during early adulthood (M = 18.3 years, SD = 2.1 years). The probability ratio of developing a depressive disorder for those with severe mood dysregulation was 7.2, 95% CI [1.3 – 38.8]. Cases were not significantly more likely than non-cases to be diagnosed with ADHD, conduct disorder, oppositional defiant disorder, substance abuse or dependence, or any anxiety disorder in the last wave of assessment. These findings should, however, be interpreted with caution, due to several limitations. Most notably, the study was a post hoc study of existing data. The severe mood dysregulation criteria as defined by Leibenluft et al. (2003) also had to be adjusted, as the CAPA was not designed to assess severe mood dysregulation. In addition, many of the participants had not yet reached the average age-of-onset for bipolar disorder and may well have been experiencing the onset of bipolar disorder as a depressive episode and gone on to developed mania or hypomania. Finally, participants were primarily Caucasian, thus it is not possible to say whether these findings can be extrapolated to a more diverse population.

Based on the findings of Brotman et al. (2006), Stringaris and Goodman (2009), hypothesised that the Irritability dimension of oppositionality - the combination of temper outbursts, anger, and a low threshold for being annoyed – would be predictive of distress disorders, which included depression and generalised anxiety. The two other dimensions of oppositionality identified were the Headstrong dimension (associated with ADHD and conduct disorders) and the Hurtful dimension (linked to the aggressive symptoms of conduct disorder). In order to investigate this, researchers surveyed a large, cross-sectional

epidemiological sample of 7912 British children and adolescents, aged 8 to 19 years, using The Strengths and Difficulties Questionnaire (SDQ) and The Development and Well-Being Assessment (DAWBA). Results suggested that only a high score on the Irritable dimension was highly predictive of distress disorders at a three-year follow-up, with an odds ratio of 3.34, 95% CI [2.08 - 5.38] at baseline.

The lack of evidence supporting severe, chronic irritability as a developmental phenotype of bipolar disorder prompted the adaptation of severe mood dysregulation to DMDD, with the new category omitting the hyperarousal criterion (Dougherty et al., 2014).

Features and Diagnostic Criteria

The *DSM-5* (APA, 2013a) describes DMDD as a mood disorder that is characterised by chronic, severe irritability. Clinically, the irritability manifests in two ways. The first is frequent temper outbursts that are developmentally inappropriate and not proportionate to the situation. These temper tantrums are usually the result of frustration and may be verbal or behavioural (i.e., aggressive behaviour directed at self, others or property). The second manifestation is a chronic and persistent angry or irritable mood between outbursts, which must be a distinguishing feature of the child and evident to individuals the child's environment.

The DMDD diagnostic criteria (see Table 1) state that temper outbursts have to happen at least three times per week, and the irritable or angry mood has to be present for most of the day, on most days, in at least two settings (school, home or with peers) for a year or more. Symptoms have to be severe in at least one setting. No more than three consecutive months must pass without symptoms. The onset of symptoms has to be before the age of 10 and diagnosis has to be made after the age of 6 or before the age of 18 (APA, 2013b).

Disruptive Mood Dysregulation Disorder cannot be diagnosed with oppositional defiant disorder, intermittent explosive disorder or bipolar disorder. Comorbid disorders

include major depressive disorder, attention-deficit/hyperactivity disorder, conduct disorder, and substance use disorders. When children meet the criteria for both DMDD and oppositional defiant disorder, they should receive a diagnosis of DMDD only. The diagnosis of DMDD should not be given if a child or adolescent has ever had a manic or hypomanic episode lasting more than one day.

Symptoms should not be better explained by a medical disorder, be a manifestation of substance abuse or a medical condition, or only occur during an episode of major depressive disorder (Zepf & Holtmann, 2016).

Table 1

Summary of DMDD Diagnostic Criteria

No.	Criteria
1.	Severe, recurrent temper verbal or behavioural tantrums that are entirely disproportionate in extent and/or duration to the provocation or situation
2.	Outbursts are developmentally inappropriate and occur at least three times per week
3.	Chronic and persistent angry or irritable mood between outbursts
4.	Irritable/angry mood present most of the day, almost daily, and evident to others in the child's surroundings
5.	Symptoms occur in at least two settings (home, school, peers) and are severe in at least one
6.	Duration is at least 12 months, without a symptom-free period of three consecutive months
7.	Onset prior to the age of 10
8.	First diagnosis should be made after age 6 and before age 18
9.	Mania or hypomania have not been present for longer than one day
10.	Symptoms are not restricted to an episode of major depressive disorder or better explained by autism spectrum disorder, posttraumatic stress disorder, separation anxiety disorder, persistent depressive disorder.
11.	Cannot receive a comorbid diagnosis of oppositional defiant disorder, intermittent explosive disorder, or bipolar disorder.

Functionally, these symptoms cause significant disruptions in the child's social relationships and academic career (APA, 2013b). Family life is usually significantly disturbed by the child's rages and persistent irritability.

Children with DMDD also struggle to initiate and maintain friendships. A very low frustration tolerance means that these children struggle to perform in school and often are not able to participate in activities enjoyed by healthy peers. Levels of impairment compare to those of children with paediatric bipolar disorder (Leibenluft, 2011), with severe aggression, dangerous behaviour, suicide ideation and suicide attempts and psychiatric hospitalisation common in both clinical populations (APA, 2013a). Few studies have, however, examined the prevalence of DMDD in clinical settings and/or community samples.

Prevalence and Comorbidity

Emerging evidence suggests that DMDD frequently occurs in clinical samples (Dougherty et al., 2016). Most studies focused on children treated at paediatric mental health clinics in the USA. In a child psychiatric outpatient sample, which was preselected for the presence of noticeable mood instability in participants, Axelson et al. (2012) found that 26% of children (N = 706) participating in the Longitudinal Assessment of Manic Symptoms (LAMS) met the DMDD criteria at intake. In an inpatient sample of 82 children, between the ages of 5 and 12, hospitalised consecutively at a 10-bed university hospital children's inpatient psychiatric unit in the USA, 30.5% of children met criteria for DMDD by parent report, while only 15.9% met the criteria by inpatient unit observation (Margulies et al., 2012). It should be noted that this discrepant inter-rater reliability between outpatient and inpatient populations gives credence to the concerns underlying the issue of the validity of DMDD as a diagnostic entity. A limited number of studies have investigated DMDD prevalence in the community (Copeland et al., 2013; Dougherty et al., 2014).

In three large community samples of children aged 2 to 17 years in North Carolina in the USA, Copeland et al. (2013) found the 3-month prevalence to range from 0.8% to 3.3%, with the highest prevalence in pre-schoolers (who were included despite the *DSM-5* exclusion criteria). The authors note that the rates dropped slightly when the strict exclusion criteria

were applied but were virtually unchanged when the onset and duration criteria were applied. In another large community sample of 6-year olds from the Stony Brook Temperament Study, the 3-month prevalence rate was 8.2% (*n* = 38; Dougherty et al., 2014).

Limited studies have focused on the temporal stability of DMDD, but those that do suggest it is relatively low. Stability over time also may be higher in childhood than adolescence (Zepf, Biskup, Holtmann, & Runions, 2015). Dougherty et al. (2016) found that more than 80% of children who met the criteria for DMDD at age 6 (*N* = 473) also met the criteria when they were 9. Rates did, however decline from 7.6% at age 6 to 1.3% at age 9. As there were methodological differences in clinical assessments at age 6 and age 9 – using the Preschool Age Psychiatric Assessment (PAPA) with parents at age 6 and the Kiddie-Schedule of Affective Disorders and Schizophrenia (K-SADS) with parents and children at age 9 – it is not possible to conclude that these changes were due to a significant decline in DMDD across the developmental period. In marked contrast with these findings, a study of over 200 youths, aged 7-17, with severe mood dysregulation found that less than 50% met criteria at follow-up (Deveney et al., 2015). A possible explanation for this discrepancy in findings could be the difference in the age range of the participants in the studies, with that of Deveney et al. (2015) including much older children. In a slightly larger community sample of 376 children aged 6 to 12 years, stability over time was low as well: 71% of participants with DMDD symptoms at baseline no longer had the symptoms at the 8-year follow-up. Of those who had symptoms at follow-up, only 45% had these symptoms at baseline (i.e. 55% were new cases; Mayes et al., 2015). Lack of temporal stability is also reflected in clinical samples. In one clinical sample, 48.7% and 39.5% of children with severe mood dysregulation continued to meet criteria for severe mood dysregulation at two- and four-year follow-ups, respectively. Of the participants with DMDD at initial assessment, 51.3% and 36.4% continued to meet criteria for DMDD at two and four years, respectively (Dougherty

et al., 2016). Similar decreases in irritability after early childhood are evident from cross-sectional data on the prevalence of DMDD across childhood (Copeland et al., 2013) and longitudinal data on normative irritability across childhood (Wiggins, Mitchell, Stringaris, & Leibenluft, 2014).

Furthermore, findings suggest that the lifetime prevalence dropped when criteria had to be met in two or more waves of assessments. Axelson et al. (2012) demonstrated that, in a child psychiatric outpatient population, with ages ranging between 6 and 12, 52% of participants met the DMDD criteria at first assessments, while 29% met the criteria at the second assessments and only 19% met the criteria for all three assessments across one-year and two-year follow-ups. This finding was consistent with that of Brotman et al. (2006), who applied the criteria for severe mood dysregulation to the GSMS. Among children who met the criteria for severe mood dysregulation, 82.5% did so in only one of four waves of assessments. The lifetime prevalence dropped from 3.3% to 0.4% when criteria had to be met in two consecutive waves.

Comorbidity was the rule in most of the studies: participants with DMDD had a comorbid psychiatric disorder more often than not, most frequently depressive disorders, ADHD, oppositional defiant disorder and conduct disorder. Youths with DMDD also exhibited higher rates of social impairments, suspension from school, service use, and poverty (Axelson et al., 2012; Copeland et al., 2013; Dougherty et al., 2014; Dougherty et al., 2016).

Retrospectively diagnosing DMDD in children who participated in the LAMS study, Axelson et al. (2012) found that almost all (96%) of the participants who met the criteria for DMDD also met the criteria for oppositional defiant disorder or conduct disorder and 77% also met the criteria for ADHD and oppositional defiant disorder or conduct disorder. It should be noted that this study had various limitations which may have skewed the results.

Participants were disproportionately recruited to score highly on the Parent General Behavior Inventory 10-Item Mania Scale (PGBI-10M), and DMDD was associated with increased PGBI-10M scores. Most of the participants also presented to outpatient clinics at academic psychiatric departments in the US, so results cannot be generalised to other clinical or community samples. Furthermore, low numbers of participants with bipolar disorder made it difficult to attain accurate estimations of the association between DMDD and paediatric bipolarity. Adequate operationalising of retrospective DMDD diagnoses may also have influenced results (Axelson et al., 2012; Vassar & Holzmann, 2013).

Using existing data of three large community samples involving 7881 observations of 3258 participants covering a broad age range (2 to 17 years old), Copeland et al. (2013) also found a propensity toward comorbidity extending to emotional disorders (anxiety or depressive disorders) and behavioural disorders (ADHD, oppositional defiant disorder, or conduct disorder), or both. Again, the result of this study should be interpreted bearing several limitations in mind. The data used for this investigation relied upon psychiatric instruments that were designed to assess other disorders, not DMDD. Test-retest reliability for these instruments used to assess DMDD was not available. None of the studies recruited participants with the aim of approximating a sample representative of US children, although strategies were used to minimise selection bias. Moreover, community samples may differ from clinical samples.

Dougherty et al. (2014) studied the epidemiology of DMDD using data from an extensive community-based study that followed children from ages 3 to 6 years. 541 parents were interviewed regarding their 3-year-old child's (M = 3.6 years, SD = 0.3 years) diagnostic information. Of these participants, 462 parents were interviewed again when their child reached the age of 6 years (M = 6.1 years, SD = 0.4 years). Children with DMDD presented with another emotional or behavioural disorder in 60.5% of the cases. At age 6,

DMDD was associated with emotional and behavioural dysregulation and functional impairment, such as depression, oppositional defiant disorder, increased emotional reactivity and intensity, decreased effortful control, poor peer functioning, as well as reduced parental support and marital satisfaction.

Disruptive mood dysregulation disorder is expected to be diagnosed more often in males and school-aged children than females and adolescents (APA, 2013a). According to Leibenluft (2011), children presenting to clinics in the USA with symptoms of DMDD are predominantly male. Brotman et al. (2006) also found this gender bias to be reflected in community samples with severe mood dysregulation and chronic irritability, on which the DMDD category is primarily based.

All the studies reported here were conducted in developed countries in USA and Europe, with none in developing regions such as South America or Africa. Epidemiological studies on DMDD in clinical and community populations in South Africa have not been published at the time of writing and warrant investigation. Another potential direction for further research would be to explore whether the higher prevalence of bipolar disorder seen in child and adolescent medical aid beneficiaries could be explained by the lack of a more appropriate category (i.e., DMDD) at the time of diagnosis or whether concerns about its validity as a diagnostic entity limits its utility.

Course and Outcome

Data on the predictive validity, trajectory and long-term outcome of DMDD are sparse. Replicating and extending the findings of the Stony Brook Temperament Study in the USA (Bufferd, Dougherty, Carlson, Rose, & Klein, 2012; Dougherty et al. 2011), one study identified predictive indicators at age 3. These were childhood ADHD, depression, oppositional defiant disorder, behavioural dysregulation, poorer peer functioning, and child temperament (higher surgency and emotional intensity, with reduced ability to voluntarily

manage attention and adapt behaviour as needed, known as “effortful control”; Dougherty et al., 2014, p. 2342). Other predictive indicators include parental substance use disorder and greater hostility in parents (Dougherty et al., 2014).

These findings are consistent with that of a later study (Dougherty et al., 2016) in a sample of 473 children who lived within a 32-km range of Stony Brook University in New York, USA. Findings suggest that a diagnosis of DMDD at age 6 predicted a DMDD diagnosis at age 9. Disruptive mood dysregulation disorder at 6 years also predicted ADHD at age 9, as well as a current and later diagnosis of depressive disorder. These children were more likely to exhibit disruptive behaviour disorder symptoms as well. A childhood DMDD diagnosis also predicts consequent difficulties with peer relationships, increased peer ostracism and victimisation, and greater relational aggression. In later childhood, between the ages of 10 and 16 years, a DMDD diagnosis predicts long-term, pervasive compromised functioning. Compared to controls without history of childhood psychiatric problems (non-cases) and controls who met the criteria for psychiatric disorders other than DMDD, those who met the criteria for DMDD were at increased risk for poor health and severe illness, sexually transmitted diseases, other psychiatric problems (excluding bipolar disorder), risky or illegal behaviour, police contact, disrupted social functioning (e.g. violent relationships, poor parental relationships and no best friend), being impoverished and low educational achievement as adults (Copeland, Shanahan, Egger, Angold, & Costello, 2014). While these studies highlight the poor prognosis of children with chronic irritability and frequent temper outbursts and the importance of making treatment development and clinical management of these children a priority, additional longitudinal research is also necessary to address the limitations of these studies, such as small sample sizes, lack of test-retest validity, the use of measures and interviews that were not designed or validated for the assessment of DMDD, reliance on reports from parents and teachers who may have different interpretations of the

term “temper tantrums”, a focus on the frequency of outbursts and a lack of qualitative data on the triggers, duration and intensity of temper tantrums.

Similar to the shortage of literature on the epidemiology and course of DMDD, little empirical data exist on the physiological and neurological mechanisms that underlie this clinical diagnosis.

Pathophysiology

There is growing interest in the underlying neurobiology of severe mood dysregulation, most of which focuses on social and emotional information processing and emotion regulation (Zepf et al., 2015). According to Leibenluft (2011), the main symptom of DMDD, chronic irritability, may be explained from a neuroscience systems perspective as a failure to employ top-down mechanisms (i.e. executive functioning and selective attention) to inhibit maladaptive responses to frustration, which is defined as the emotional response to blocked goal attainment. Emerging evidence suggests that individuals with DMDD show deficits in four main processes that contribute to their symptoms (Driver & Thomas, 2018). These are impaired regulation of emotion and attention (Brotman et al., 2010; Rich et al., 2010a; Rich et al., 2007); misinterpretation of social, emotional, and threat stimuli (Brotman et al., 2010; Rich et al., 2008); impaired context sensitivity (Blair, 2010; Dickstein et al., 2007); and reward system dysfunction (Brotman et al., 2007; Dickstein et al., 2010; Leibenluft, 2011).

The skill to accurately process social cues, a key social-emotional function, as defined by Ochsner (2008), that enables emotion regulation and social competence, seems to be lacking in those with severe mood dysregulation (Leibenluft, 2011). Brain structures identified as playing a role in these functions include the amygdala, striatum, and classical limbic regions, such as the medial prefrontal cortex and insula (Ochsner, 2008). Particularly important in the detection and interpretation of nonverbal cues (e.g., eye gaze and facial

expressions) that may directly or indirectly signal the presence of threat in the environment, such as the faces of seemingly untrustworthy people (Adolphs, Tranel, & Damasio, 1998; Winston, Strange, O'Doherty, & Dolan, 2002) and fearful facial expressions (Morris et al., 1996; Whalen et al., 1998), is the amygdala - an almond-shaped structure on the medial temporal lobe that is situated next to, and just in front of, the hippocampus.

The amygdala seems to be reliably activated with the presentation of stimuli that are biologically relevant (e.g., potential threat), particularly ones that evoke strong negative emotional states. Neuroimaging studies show that amygdala signal intensity is greater when participants viewed graphic images and film clips of negative material (such as mutilated bodies) than when they viewed neutral material (Irwin et al., 1996; Reiman et al., 1997) and that the amount of activity within the amygdala is associated with later recall (Cahill et al., 1996). There is also considerable evidence that supports the role of the amygdala in the processing of stimuli that might be considered to be emotional, but which don't necessarily induce an intense emotional state, such as facial expressions (Breiter et al., 1996; Cahill et al., 1996; Irwin et al., 1996; Morris et al., 1996; Morris et al., 1998; Reiman et al., 1997; Whalen et al., 1998). For example, individuals presented with photos of people with fearful expressions, do not report feeling afraid, despite increased activity in the amygdala. This suggests that reported emotion should not be equated with amygdala activation (Whalen, 1998). The amygdala seems particularly crucial in the processing of fear in facial expressions (Phelps, 2006).

Several brain lesion studies (Adolphs, et al., 1994; Anderson & Phelps, 2000b; Broks et al., 1998; Calder et al., 1996) show that patients with bilateral damage to the amygdala have significant impairment in identifying fearful facial expressions. Neuroimaging studies (Breiter et al., 1996, Morris et al., 1996, Whalen et al., 2001) of healthy participants demonstrated greater activation in the amygdala when presented with fearful faces, compared

to happy faces, angry faces and neutral faces. Three adult patients with large lesions to the temporal lobe regions, which included complete bilateral damage to the amygdala, were also found unquestionably impaired on face labelling tasks (Schmolck & Squire, 2001). When presented with images of male and female actors expressing one of six target facial emotions (i.e., anger, happiness, sadness, surprise, fear, or disgust), the patients were particularly poor at labelling faces expressing sadness and fear (58% and 39% correct, respectively) compared to healthy volunteers (88% and 73% correct). Patients also performed worse than controls when anger and disgust were expressed (patients, 61% and 75% versus controls, 73% and 80%), but these differences were not statistically significant. When happiness and surprise were expressed, patients scored a little higher than controls (patients, 99% and 94%; controls, 97% and 80%), but again, not significantly so. For each emotion, the groups made the same most common error. For example, when presented with a sad expression, the most common error by controls and patients was to describe it as disgust (on 22% of the trials for patients and 10% of the trials for controls). Fearful expressions were mostly commonly erroneously described as surprise (in 33% of the trials for patients and in 17% of the trials for controls). This may be explained by the fact that fear and surprise have similar features of wide eyes and an information-gathering appearance. While they have features in common, they convey different messages: a fearful expression indicates shock in reaction to a frightening stimulus, whereas surprise denotes novelty or unexpectedness. (Whalen, 1998; Zhao, Yan, Chen, Zuo, & Fu, X. 2013; Zhao, Zhao, Zhang, Cui, & Fu, 2017).

However, these findings are not definitive. Researchers are yet to fully understand the complexities of the various structures of the brain and their direct or indirect influences on each other, as is evident from studies that did not corroborate the findings discussed above. No association between amygdala activation and expression of fear was found by Sprengelmeyer, Rausch, Eysel, & Przuntek (1998) or Rapcsak et al., (2000). A potential

explanation for this could be either the rapid habituation of the amygdala in response to fearful faces reported by Breiter et al. (1996) or the proposed neuromodulatory effect of the amygdala on the fusiform gyrus – decreased activation in the amygdala was mirrored by increased activity in the fusiform gyrus in reaction to facial expressions of fear (Morris et al., 1998).

Because of its extensive connections to brain structures associated with cognitive functions (e.g. the prefrontal cortex, the hippocampal complex and the sensory cortices), the amygdala is thought to influence cognitive functions in response to emotional stimuli (Phelps, 2006). In fact, the primary function of the human amygdala is suggested to be the modulation of neural structures responsible for cognitive and behavioural responses to emotional stimuli (Anderson & Phelps, 2000a; Whalen, 1998), some of which may bypass the cortex, which has led some researchers (Öhman, 2002; Phelps, 2006; Whalen, 1998) to the conceptualisation of the amygdala as a "surveillance system". This theory purports that the amygdala continually scans the environment for relevant affective stimuli, particularly those that signal an increased potential of threat, and controls the perception and memory processes that detect and translate them. In an extensive review, Phelps (2006) identified five areas that emphasise the role of the amygdala in the interplay between emotion and cognition: emotional learning (i.e., classical fear conditioning, instructed fear and observational fear), emotion and memory, the effect of emotion on attention and perception, the processing of emotion in social stimuli, and adapting emotional responses.

Due to the sensory system's limited processing capacity, the brain has to employ various mechanisms, such as selective attention and executive functions, to detect relevant information in the environment and evaluate subsets of information based on their current relevance or salience (Öhman, 2002; Vuilleumier, 2005). Numerous mechanisms of selective attention exist and function concurrently during perceptual processes and any factors that

influence these early phases of stimulus processing may also affect downstream cognitive functions, such as memory and reasoning (Driver, Vuilleumier, Eimer, & Rees, 2001; Phelps, 2006). This could be linked to Leibenluft's (2011) explanation of irritation as the result of the failure to use these mechanisms to regulate maladaptive responses to frustration when goal attainment is blocked.

One of the mechanisms through which the amygdala modulates attention, perceptual processes and responses to potential threat is its ability to detect fear or threat stimuli early and automatically due to its reciprocal connection to the sensory cortices. Several studies show that the amygdala receives information about the emotional significance of a stimulus promptly and prior to attention or awareness (Anderson, Christoff, Panitz, De Rosa, & Gabrieli, 2003; Morris et al., 1998; Öhman, 2002; Romanski & LeDoux, 1992; Vuilleumier, Armony, Driver, & Dolan, 2001; Whalen et al., 1998). Supporting these finding are studies that show increased heightened activity in the visual cortex of middle-aged subjects in response to aversive stimuli compared to neutral stimuli (Kosslyn et al., 1996), with the extent of this stimulation related to amygdala activation in response to the same stimuli (Morris et al., 1998). An fMRI study conducted with individuals with medial temporal lobe damage provides further evidence that suggests that the amygdala facilitates heightened responses in the visual cortices of the brain for emotional stimuli. Three groups of participants - healthy controls, patients with damage only to the hippocampus, and patients with damage to the hippocampus and amygdala – were presented with facial expressions of fear, as well as neutral expressions (Vuilleumier, Richardson, Armony, Driver, & Dolan, 2004). Control participants exhibited increased activation in the visual cortex for fear compared to neutral faces. These results were mirrored in patients with damage limited to the hippocampus. Participants with damage to the amygdala did not demonstrate significant differences in visual cortex activation for fear compared to neutral faces. These findings

suggest that the amygdala performs a crucial role in facilitating the temporary changes in visual cortex processing that occurs for emotional stimuli. While the amygdala seems particularly critical in the processing of facial cues related to threat and fear, other studies suggest more extensive impairment in recognising various highly arousing emotions that are akin to fear (i.e., negatively valenced emotions), including anger, disgust and sadness (Adolphs, Russell, & Tranel, 1999; Calder et al., 1996; Schmolck & Squire, 2001).

In three experiments, the ability of three patients with bilateral damage to the anterior temporal lobe to process facial emotions was assessed (Schmolck and Squire, 2001). Patients performed consistently poorer than controls in the first and second experiments when having to label emotions of fear and sadness. In the third experiment, each face was accompanied by an adjective that was either consistent or inconsistent with the emotion displayed. Participants had to rate the intensity with which each face conveyed the emotion described by the adjective. As a rule, patients gave higher ratings than controls, particularly for incongruent pairs. The researchers ruled out the possibility of the findings merely reflecting an inclination of patients to give higher ratings by comparing the first and the third experiments. In Experiment 3, patients gave higher ratings to particular incongruent face-adjective pairs (for example, when a sad face was displayed with the adjective ‘disgust’) and in the first experiment, they tended to choose the same incorrect adjective for the emotions displayed (e.g., they repeatedly selected the word ‘disgust’ to describe a sad face). Consequently, the researchers concluded that that this impairment in the patients indicated difficulty in distinguishing between particular emotions. Specifically, they tended to confuse sadness with disgust and anger, and fear with surprise and anger.

Studying identification of two emotional dimensions, arousal and valence (pleasantness-unpleasantness), in a unique case with complete, bilateral damage limited to the amygdala, another study found compromised detection of arousal for facial expressions,

words and sentences that portrayed negative emotions, specifically those related to fear and anger (Adolphs et al.,1999). Compared to control subjects, the patient gave unusually low arousal ratings to negatively valenced emotions. Fear and anger, the two negative emotions that were rated to be the most arousing by controls, were given the lowest rating by the test subject. Recognition of valence, however, was normal. These results confirm earlier findings by Calder et al. (1996), who reported investigations of two adult individuals with bilateral damage to the amygdala: one had partial bilateral amygdala as result of surgery for otherwise unmanageable epilepsy, while the other had extensive right temporal damage, with bilateral damage to the amygdala, caused by encephalitis. Across the tests performed, both subjects showed significant impairment in recognition of the facial expression of fear. One also had severe problems with disgust and anger, while the other one exhibited minor abnormality in recognition of anger. These studies substantiate the theory of a broader role for the amygdala in the processing of emotional expressions, but it is possible that the impairments were the result of not only bilateral damage to the amygdala, but also other regions involved in emotion recognition and processing. Expanding these findings is evidence that suggests the amygdala's role is not limited to that of detecting potential sources of threat and danger (i.e., negatively valenced emotions), but may have a more general purpose in processing salient information from faces. The amygdala also responds to novel (Duan, Dai, Gong, & Chen, 2010; Schroeder et al., 2004) and positive stimuli (Adolphs, 2010; Ball et al., 2009; Breiter et al., 1996; Fitzgerald, Angstadt, Jelsone, Nathan, & Phan, 2006; Sergerie, Chochol, & Armony, 2008; Yang et al., 2002) and receives perceptual inputs from various sources, such as the cortex, thalamus and hippocampus (Phelps & LeDoux, 2005; Weymar & Schwabe, 2016).

Using a backwards masking procedure, Duan et al. (2010) studied neural activity in reaction to surprised faces displayed below the threshold of conscious visual perception using

fMRI. Healthy participants were scanned viewing masked surprised faces, and as control, masked happy and neutral faces too. Results showed that, compared to control conditions, masked surprised faces resulted in significantly greater activation in the parahippocampal gyrus and fusiform gyrus, which have previously been associated with the detection of novelty (Rombouts et al., 1997; Sperling et al., 2001; Stern et al., 1996; Zorrilla, Jeste, Paulus, & Brown, 2003). These fMRI results also confirmed activation of the right amygdala and right thalamus in response to the masked surprised faces, confirming previous work linking these regions to the unconscious processing of emotional stimuli (Bayle, Henaff, & Krolak-Salmon, 2009; Driver et al., 2001; LeDoux, Ruggiero, & Reis, 1985; Liddell et al., 2005; Morris et al., 1998; Morris, Öhman, & Dolan, 1999; Whalen et al., 1998). The researchers also found a correlation between the ability to identify and differentiate one's emotions, as measured by the 20-item Toronto Alexithymia Scale (TAS-20), and the activation of these areas. A negative correlation was found between the subscale, Difficulty Identifying Feelings, and the neural response of these areas to masked surprised faces, which suggests that diminished activation in specific brain regions may indicate greater difficulties in recognising emotions. This study substantiated findings by Schroeder et al. (2004), who scanned healthy participants while they viewed surprised faces. Participants were also presented with faces portraying neutral or disgust expressions as a control. Neural responses during the emotional conditions were compared with each other and with the neutral face condition. In contrast with both control conditions, perception of surprised facial expressions produced consistently increased activation in the parahippocampal area of the brain, a region that is associated with the detection of novelty. An increasing number of neuroimaging studies also confirm the involvement of the amygdala in the processing of positive stimuli (Duan et al., 2010).

A meta-analysis of 114 human functional imaging studies concerned with the amygdala published between 2000 and 2008 (Ball et al., 2009) found that of the 339 amygdala response peaks, 59% were related to positive stimuli. Another study examined the activity in healthy volunteers during rapid presentations of fearful, happy and neutral faces using fMRI. As discussed above, the amygdala was preferentially activated by fearful faces compared to neutral faces, but also to happy faces versus neutral faces, suggesting a possible general response to emotionally valenced stimuli (Breiter et al., 1996). Further challenging the notion that the amygdala has a specialised role in the processing of emotions mostly related to potential threat (i.e., fear or anger), are findings by Fitzgerald et al. (2006) that propose a more general-purpose function in processing all relevant information from faces. This critical component of evolutionary shaped social intelligence, the ability to effectively and automatically decode communicative facial signals, seems to be perturbed in children with severe mood dysregulation, which may contribute to their symptoms of emotional problems.

An investigation into the neural circuitry involved in these deficits revealed that the same behavioural deficiency could be brought about by different forms of neural dysfunctions, with amygdala dysfunction reported as the most common finding in functional magnetic resonance imaging (fMRI) studies (Brotman et al., 2010; Leibenluft, 2011; Thomas et al., 2012). Studies show that children with severe mood dysregulation struggled as much with correctly identifying emotions in facial expressions as children with bipolar disorder and made more mistakes than healthy children and volunteers with mood, anxiety, conduct or attention deficit disorders (Guyer et al., 2007; McClure et al., 2005; McClure, Pope, Hoberman, Pine, & Leibenluft, 2003; Schenkel, Pavuluri, Herbener, Harral, & Sweeney, 2007). It is not possible to compare the results of these studies directly, however, as each of these studies used different paradigms. To date, few neuroimaging studies have focused on

the comparison of amygdala activity in other psychiatric disorders and severe mood disorder or DMDD during face emotion processing (Brotman et al., 2010; Rich et al., 2008; Thomas et al., 2014).

Despite similar difficulties with the facial expression labelling task, children with severe mood dysregulation showed decreased amygdala activity compared to that of children with bipolar disorder and control subjects (Brotman et al., 2010). In another study, subjects with severe mood dysregulation also needed more emotional intensity in facial expressions to correctly label the emotion in the expressions than control subjects and were as compromised as children with bipolar disorder (Brotman et al., 2008; Deveney et al., 2012; Rich et al., 2008). Hommer et al. (2014) examined attentional partiality toward angry (i.e., threat) and happy faces in participants with severe mood dysregulation and healthy control subjects. Children with severe mood dysregulation demonstrated an attention preference towards threat, with greater threat bias correlated with increases in the severity of dysregulation symptoms. In another study, compared to healthy volunteers and those with bipolar disorder, children with severe mood dysregulation showed an over-reactivity to angry faces, which could be seen as evidence for the dysregulation of the behavioural approach system (Thomas et al., 2014) – a conceptually neurological motivation system proposed by several motivation theorists (e.g., Davidson, 1998; Depue & Collins, 1999; Depue & Iacono, 1989; Fowles, 1980; Fowles, 1988; Gray, 1975; Gray, 1976; Gray, 1982; Gray, 1994; Gray & McNaughton, 2000; Lang, Bradley, & Cuthbert, 1990). This flows from an earlier study during which children with severe mood dysregulation reported higher levels of frustration than control subjects in response to blocked goal attainment, possibly also attributable to a failure to regulate the approach system (i.e., the inability to inhibit approach responses when these responses are not successful; Deveney et al., 2013; Thomas et al., 2014).

Motivation theories of behaviour propose that two biologically-based systems underlie most of behaviour (Harmon-Jones, 2003). The first facilitates approach motivation and behaviour in response to appetitive stimuli (i.e. reward and the cessation or omission of punishment), and corresponds with impulsivity (Corr, 2002; Corr, 2004; Gray, 1970). It has been referred to as a behavioural activation system (Fowles, 1980; Fowles, 1988), behavioural approach system (Gray, 1970; Gray, 1982; Gray, 1994; Gray & McNaughton, 2000), and behavioural facilitation system (Depue & Collins, 1999; Depue and Iacono, 1989). It has also been referred to as an approach or appetitive motivational system (Davidson, 1998; Lang et al., 1990). The second, and inverse, system manages aversive motivation (i.e. punishment and omission or cessation of reward) and avoidance and withdrawal behaviours, and is thought to cause anxiety (Corr, 2001; Corr, 2002; Corr, 2004; Gray, 1970; Harmon-Jones, 2003). This system has been referred to as the behavioural inhibition system (Gray 1970; Gray, 1982; Gray, 1994), the aversive/defensive system (Lang et al., 1990), and the withdrawal motivational system (Davidson, 1998). One of the most influential theories in this field, Gray's Reinforcement Sensitivity Theory (RST; Gray, 1970; Gray, 1982; Gray, 1994; Gray & McNaughton, 2000), predicts that an individual with a hyperactive approach system should be more sensitive to cues that signal reward, compared to someone with a hypoactive approach system; and individuals with hyperactive inhibition systems should be most sensitive to signals of punishment, relative to those with hypoactive inhibition systems. According to this model, the comparison between actual reward with expected reward plays an essential role in motivation-driven behaviour. It asserts that only actual reinforcement that is equal to, or greater than, expected reinforcement is an adequate input to the approach system; and only actual punishment equal to or, or greater than, expected punishment is sufficient input to the inhibitory system. Appetitively motivated behaviour is therefore caused by actual punishment lower than expected punishment (i.e. the relief of non-punishment),

which is considered an adequate input to the approach system; while aversively-motivated behaviour is caused by an actual reward that is lower than the expected reward (i.e. frustrative non-reward), which is mediated by the inhibitory system (Corr, 2002).

Some focus has been given to using the RST to explain disinhibitory disorders, such as psychopathy, early-onset alcoholism, childhood hyperactivity and non-pathological impulsivity, specifically an underlying dysfunction in the reward system (Corr, 2001; Gorenstein & Newman, 1980). Several reports indicate that this heightened sensitivity has a number of significant consequences, all of which may be pertinent to DMDD: it results in a reaction set that is reward-focused dominant; it impairs consideration of environmental contingencies; and it leads to a failure to learn from punishment (Newman, 1987; Patterson & Newman, 1993; Wallace & Newman, 1990). These motive systems not only manage approach and withdrawal or aversive behaviour, but are also thought to be involved with the creation and promotion of emotional states that are relevant to such behaviours (Corr, 2001; Corr, 2002; Corr, 2004; Harmon-Jones, 2003).

It is widely accepted in theory that the approach motivational system is involved in the generation of positive affect (e.g., happiness, hope and elation), while the aversive motivational system is involved in the generation of negative affect (e.g., anxiety, fear, frustration, anger, and sadness) and much research support these suppositions (Carver & White, 1994; Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Depue & Iacono, 1989; Gray, 1982; Gray, 1994; Harmon-Jones & Allen, 1997; Lang et al., 1990; Sutton & Davidson, 1997). A few studies suggest that the approach system is exclusively responsible for the generation of positive affect (Cacioppo & Berntson, 1994; Lang et al., 1990; Watson, Wiese, Vaidya, & Tellegen, 1999). While there is evidence of the association of the behavioural approach system with positive effect, one critical shortcoming of these theories is that they fail to account for the relationship between individual differences in (approach

system) reward sensitivity and actual reactions to rewarding stimuli (Corr, 2002). There is reason to believe that the approach system is also linked to negative affect responses, such as sadness, anxiety and anger (Carver, 2001; Carver, 2004; Carver & White, 1994; Finlay-Jones & Brown, 1981; Harmon-Jones, 2003; Higgins, Shah, & Friedman, 1997). Of particular relevance to severe mood dysregulation and DMDD is the suggestion that the approach system is linked, at least to some extent, to frustrative non-reward affective states and behaviour due to its sensitivity to reward (Corr, 2002). Specifically, anger has been found to cause activation in the left anterior cortical region, which has been linked to the approach system (Harmon-Jones & Sigelman, 2001). Evidence suggests that increased left frontal cortical activity and decreased right frontal cortical activity are associated with both trait and state anger (Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001, Harmon-Jones, Sigelman, Bohlig, & Harmon-Jones, 2003). This may be because higher levels of approach behaviour are associated with higher expectancies for rewards, and therefore higher levels of frustration result when reward magnitude is terminated or reduced (Carver, 2001; Corr, 2002; Mikulincer, 1988). Although limited research was available, and findings were tenuous (i.e. methods used were correlational and therefore limited to problems associated with correlational designs, such as determining causality, third variables, etc.), these studies gave some credence to the theory that chronic irritability is the emotional response to blocked goal attainment, i.e. frustrative non-reward (Leibenluft, 2011) and indicated that this might be an avenue to explore in children with severe mood dysregulation, and later DMDD. Evidence is emerging that these children do exhibit abnormalities that set them apart from children with other disorders and children without mental health problems.

Children with severe, chronic irritability were found to have unusually reduced activation in regions implicated in emotion, attention, and reward processing in response to negative feedback received in a frustration context (Deveney et al., 2013). In these children,

frustration appears to impair attention flexibility, which the authors surmised may contribute to emotion regulation difficulties. Congruous with documented facial labelling problems, attentional bias towards angry expressions, and maladaptive reward processing, one study suggests that children with severe mood dysregulation have difficulties in determining the correct tone of verbal speech (called prosody), which could play a role in emotion dysregulation in chronically irritable children (Deveney et al., 2012).

The ability to identify the emotional tone or prosody of spoken words is an important aspect of nonverbal emotional cue processing, competent emotional interactions and emotional communication (Halberstadt, Denham, & Dunsmore, 2001; Scherer, 2003). Misinterpreting, for example, "playful sarcasm as hurtful criticism", can lead to tension in interpersonal relationships, decrease social support, and increase probability and severity of mood symptoms (Deveney et al., 2012, p. 262). Previously an understudied field of psychopathology, particularly in children, more findings are appearing on impaired affective prosodic processing and expression in psychiatric populations, such as adults with major depressive disorder, schizophrenia, bipolar disorder (Bozikas et al., 2007; Kan, Mimura, Kamijima, & Kawamura, 2004; Mitchell, Elliott, Barry, Cruttenden, & Woodruff, 2004; Murphy & Cutting, 1990; Péron et al., 2011; Uekermann, Abdel-Hamid, Lehmkaemper, Vollmoeller, & Daum, 2008). Furthermore, comprehensive nonverbal emotion identification training may be a promising adjunctive treatment for enhancing functional outcomes of people with psychopathology (Deveney et al., 2012; Horan et al., 2011). To date, very few studies have examined the relationship between mood or behavioural disorders in children and receptive and expressive emotional prosody.

Most recently, one study examined the association between internalising symptoms (i.e., depression and anxiety) and adolescents' ability to recognise vocal social-emotional expressions by youth (Morningstar, Dirks, Rappaport, Pine, & Nelson, 2017). Fifty-seven

children between the ages of 8 and 17 (*M* = 12.62, *SD* = 2.66), 28 with anxiety and 29 without, were asked to identify the intended emotion in youth actors' (11 – 15 years, *M* = 13.60 years, *SD* = 1.43) portrayal of seven basic emotions: anger, disgust, fear, friendliness, happiness, meanness, and sadness (Morningstar, Dirks, & Huang, 2017). The sentence content was socially relevant but neutral in emotional content and was designed to apply to all expressions, such as "Why did you do that?", "I was just trying to be nice", "I didn't know about it", and "You shouldn't have done that" (Morningstar et al., 2017, p. 3). Two important findings surfaced from this study. Firstly, prosody recognition improved with age, confirming previous studies that found the ability to accurately identify vocal emotional cues continues to develop throughout childhood (Allgood & Heaton, 2015; Chronaki, Hadwin, Garner, Maurage, & Sonuga-Barke, 2014; Egan, Brown, Goonan, Goonan, & Celano, 1998; Sauter, Panattoni, & Happé, 2013). Secondly, depressive symptoms, but not anxiety symptoms, were related to poorer recognition of vocal expression of happiness and anger. This study confirms the results of an earlier investigation into the accuracy of receptive prosodic speech recognition in 38 school-aged boys, half of whom had received a depression diagnosis (Emerson, Harrison, & Everhart, 1999). Participants were asked to identify happy, sad, angry and neutral prosodies within congruent and incongruent verbal statements, such as "All the puppies are dead" stated in both happy and sad tones. The children with depression consistently had more difficulty identifying both congruent and incongruent affective prosody than the boys without depression. Children with depression not only experience problems with receptive emotional prosody, but also expressive emotional prosody. A recent study (Cummins et al., 2015) suggests that acoustic speech analysis and classification could be used as early identification of adolescents showing early signs of major depression up to two years before they meet the criteria for the full-blown disorder. Receptive emotional prosody problems were not, however, found in children with

externalising disruptive behavioural symptoms. Compared to chronically ill children and typically developing children, children with attention deficit hyperactivity disorder, oppositional defiant disorder, conduct disorder, or comorbid combinations thereof, were not less accurate in decoding verbal and non-verbal emotional stimuli (Egan et al., 1998). These phenotypic differences in expressive and receptive emotional prosody may reflect different brain/behaviour mechanisms in children with mood disorders, such as DMDD, compared to children with externalising disruptive behavioural disorders and may offer more support for the notion that a distinct nosological category is warranted. As DMDD is associated with a current or later diagnosis of depression, this study sought to explore whether differences in expressive prosody exist in children with DMDD compared to controls by analysing and comparing voice recordings of both groups.

Another prerequisite for adaptive social-emotional functioning is the ability to regulate one's assessment of, and behaviour towards, others in a manner that is appropriate to the context, a construct that Ochsner (2008, p. 53) refers to as "context-sensitive regulation". According to Leibenluft (2011, p.8), context-sensitive regulation can be measured by using "response reversal paradigms", in which subjects must alter their reactions in accordance with varying stimulus-reward contingencies. Individuals who are slower to extinguish or change old, no longer rewarded responses (i.e., response reversal) may be more likely to experience frustration, and consequently, to exhibit aggressive or irritable behaviour (Blair, 2010). The neural structures involved in reactive aggression include the amygdala (which, as mentioned earlier, is also central to detection and interpretation of non-verbal cues that may directly or indirectly signal threat in the environment), the hypothalamus, and the periaqueductal grey, which is mediated by the frontal cortex. From this perspective, deficits in response reversal or the underlying neural mechanisms may explain the irritability and anger seen in DMDD. Two studies support this hypothesis. Dickstein et al. (2007) and

Dickstein et al. (2010) found that participants with severe mood dysregulation and bipolar disorder differed from non-cases in response reversal performance, although it is not yet clear whether the neural circuits mediating these differences vary between groups.

Dickstein et al. (2007) examined the cognitive flexibility (i.e. the ability to adapt to changing contingencies) in children with narrow-phenotype bipolar disorder (n = 50, M = 13.1 years, SD = 2.9 years), severe mood dysregulation (n = 44, M = 12.2 years, SD = 2.1 years), and healthy controls (n = 43, M = 13.6 years, SD = 2.4 years). Simple and compound reversal stages of an intra- and extradimensional shift task and change task (which involves inhibiting a prepotent response and substituting a new response) were compared. Children with narrow-phenotype bipolar disorder were significantly more impaired than children with severe mood dysregulation and healthy control participants on the simple reversal. On the compound reversal, both groups performed worse than the control group. On the change task, narrow-phenotype youths were slower to adapt than the children with severe mood dysregulation. This work suggests that children with severe mood dysregulation, and by extrapolation DMDD, are slower to extinguish or change old, no longer rewarded responses, which may explain why they are more prone to experience frustration, and consequently, to exhibit aggressive or irritable behaviour. However, it does not explain the neural basis for this deficit. Furthermore, various limitations, such as age differences between groups (children with severe mood dysregulation were significantly younger than those with bipolar disorder and control participants), the use of psychotropic medication that affect cognitive flexibility, mood state heterogeneity (both patient groups were presented in a variety of mood states), and comorbidity necessitate the need for further comparative studies of larger samples to determine to which degree cognitive flexibility is a trait or state deficit in children with severe mood dysregulation.

Extending this research, reversal learning deficits in children with bipolar disorder (*n* = 35) were also compared to other patient groups, specifically those with anxiety disorders (*n* = 42), major depressive disorder (*n* = 18), and severe mood dysregulation (*n* = 35), as well as healthy controls (*n* = 35; Dickstein et al., 2010). To address the limitations of previous research, the 165 participants, between the ages of seven and 17, in the five groups were age-, IQ- and sex-matched. The main finding was that, compared to typically developing children, impaired probabilistic learning was present in youths with bipolar disorder and major depressive disorder ($p = 0.07$, Cohen's $d = 0.89$). Similar deficits were not seen in children with severe mood dysregulation, however, due to the significant effect size ($p = 0.13$, Cohen's $d = 0.74$) the researchers speculated that this might have been due to a type II error. Anxiety youths did not differ significantly from typically developing controls and because the effect size was small, it was not considered the result of a type II error. These findings are limited by the use of psychotropic medication and small sample size of the major depressive disorder group. While all the children in the control, anxiety and major depressive disorder groups were medication-free, only 20% of the bipolar group and 54% of the severe mood dysregulation youths were not on medication. This means that the researchers could not perform an adequately powered comparison of the unmedicated groups (Dickstein et al., 2010). Further research exploring the effects of psychotropic medication on probabilistic learning in paediatric populations is warranted, as one study showed that differences exist in adults taking the selective serotonin reuptake inhibitor (SSRI) citalopram and those taking the noradrenaline reuptake inhibitor (NRI) atomoxetine (Chamberlain et al., 2006).

Leibenluft (2011) proposed yet another hypothesis, which is that chronically irritable children respond differently to frustrating situations than do their healthy counterparts. Two studies explored the neural and psychophysiological mechanisms of frustration in children with severe mood dysregulation using event-related potentials and neuroimaging techniques.

Rich et al. (2007) found that both children with severe mood dysregulation and bipolar disorder reported considerably more arousal during a frustration task – an affective Posner task, which is an attentional task that was altered to manipulate emotional demands and induce frustration - and showed more impairment in attention than did individuals in the control group. Using electroencephalography (EEG), a non-invasive measure of cortical activity, the event-related potentials (ERPs) of the three groups were compared. Event-related potentials are time-linked measures of the electrical activity in the brain associated with information reception and processing, as well as higher level processing. During the frustration condition, bipolar subjects had significantly lower P300 event-related potential amplitude, while children with severe mood dysregulation exhibited significantly lower N100 ERP amplitude during emotional and non-emotional tasks compared to those with bipolar disorder and control subjects. P300, or P3, has positive-going amplitude and is most strongly measured on the scalp over the parietal areas of the brain. Lower P3 amplitudes suggest problems with attention, working memory and executive functioning (Van Dinteren, Arns, Jongsma, & Kessels, 2014). N100, or N1, is a negative deflection measured over the temporal, central and frontal areas of the brain. Lower N1 amplitude indicates deficiencies in the initial stages of attention (also referred to as attention orienting; Hillyard, Hink, Schwent, & Picton, 1973). In children with severe mood dysregulation, deficits in attention orienting were similar to those reported in children with ADHD, which is not surprising given the high comorbidity between the two disorders. However, little data exists on the nature of these deficits in severe mood dysregulation, particularly in the context of emotional stimuli (Leibenluft, 2011).

Using the Emotional Interrupt Task, which measures how attention is affected by positive, negative or neutral distracters, Rich et al. (2010) found that participants with severe affective and behavioural dysregulation (n = 41, M = 12.6 years, SD = 2.6 years) showed

significantly lower attentional interference from emotional stimuli than those with bipolar disorder (*n* = 57, *M* = 14.4 years, *SD* = 2.9 years) or non-case control comparisons (*n* = 33, *M* = 13.7 years, *SD* = 2.5 years). This blunted response may contribute to the dysregulation of mood and behaviour in these children (Rich et al., 2010a). These findings are contradicted by results from a study by Brotman et al. (2010). When asked to focus on non-emotional aspects instead of emotional aspects (i.e. rating how wide the nose is compared to how hostile the face is or how afraid the subject is of the face on the image) of 32 grey-scale images of adult faces (eight happy, eight angry, eight fearful, eight neutral), participants with severe mood dysregulation (*n* = 29, *M* = 12.94 years, *SD* = 1.91 years) exhibited decreased left amygdala activation relative to bipolar (*n* = 43, *M* = 14.81 years, *SD* = 2.66 years), ADHD (*n* = 18, *M* = 13.87 years, *SD* = 1.91 years) and control groups (*n* = 37, *M* = 13.73 years, *SD* = 2.72 years) when completing subjective fear ratings of neutral faces during the nose-width and fixation trials. This suggests that children with severe mood dysregulation have trouble directing their attention away from emotional stimuli. While the first study suggests the absence of performance modulation by emotional stimuli youth with severe mood dysregulation, the discrepancy in findings may be explained by the fact that the amygdala receives information about the emotional significance of a stimulus promptly and prior to attention or awareness, but that these children also do show an attentional bias to fearful facial expressions in humans (Anderson et al., 2003; Hommer et al., 2014; Morris et al., 1998; Öhman, 2002; Romanski & LeDoux, 1992; Vuilleumier et al., 2001; Whalen et al., 1998). The exact nature and extent of the deficits in attention-emotion interactions merit further research attention, as such interactions may play an integral part in the regulation of emotional and behaviour (Leibenluft, 2011).

Apart from brain studies, the influences of genetics on juvenile irritability have also been explored. Data from a prospective four-wave longitudinal twin study (*n* = 2620) suggest

that genetics effects on irritability are present from middle childhood through to young adulthood, with patterns varying in males and females. Males were more influenced by genetics as they grew older, while genetic contributions in females were quite substantial in early life but lessened in importance with age (Roberson-Nay et al., 2015). Further insight into genetic factors related to irritability may enhance our understanding of the psychopathology and treatment of DMDD.

Treatment

There are no established guidelines for the treatment of DMDD as empirical evidence for the effective treatment of DMDD is limited and several clinical trials are still underway. It is probable that, as with most psychiatric disorders in children, multimodal interventions may produce the best results. Currently, pharmacological treatment recommendations rely on established treatments for disorders with similar symptomology (i.e., anger outbursts and irritability), including ADHD, ODD and depressive disorders. Potential pharmacological treatments include stimulants, anticonvulsants, antidepressants, antipsychotics, and mood stabilisers (Carlson, Potegal, Margulies, Basile, & Gutkovich, 2010; Connor, Glatt, Lopez, Jackson, & Melloni, 2002; Dickstein et al., 2009; Donovan et al., 2000; Driver & Thomas, 2018; Kim & Boylan, 2016; Moreno et al., 2007; Rosato et al., 2012; Tapia & John, 2018; Tourian et al., 2015; Waxmonsky et al., 2008). As DMDD have biological pathways in common with ADHD (Riglin et al., 2017) and anxiety/depressive disorders (Savage et al., 2015), and often are comorbid with these disorders, medication aimed at treating these comorbidities should be considered first (Driver & Thomas, 2018). Selective serotonin reuptake inhibitors (SSRIs) and stimulants are therefore frequently used as first-line pharmacological treatment of DMDD.

The use of stimulants to treat aggression and irritability that are frequently seen in children with ADHD have been studied and a meta-analysis has been done to determine the

effect size thereof. A review of literature from 1970 to 2001 revealed 28 studies meeting inclusion/exclusion criteria for meta-analysis and revealed that the effect size for aggression-related behaviours in ADHD is similar to those for the core symptoms of ADHD (Connor et al., 2002). Long-acting methylphenidate treatment was also found to significantly improve irritability and related emotional symptoms associated with DMDD in children with comorbid ADHD (Winters, Fukui, Leibenluft, & Hulvershorn, 2018). Effect sizes for the use of SSRIs as treatment for irritability in children and adolescents were, however, small (Kim & Boylan, 2016).

Several atypical antipsychotic medications, such as risperidone, aripiprazole and olanzapine, have shown to be efficacious in the treatment of irritability in autism spectrum disorders and severe mood dysregulation in children without autism (Stigler & McDougle, 2008). A recent study in Taiwan found that a combination of aripiprazole and the stimulant methylphenidate is well tolerated and significantly improved irritability, externalising symptoms, depression, anxiety, attention, social problems, and reaction time variability in children with DMDD and comorbid ADHD (n = 24; Pan, Fu, & Yeh, 2018). However, due to the significant side effects of these agents (e.g., weight gain, extrapyramidal symptoms and metabolic syndrome) they should be used with caution (Driver & Thomas, 2018).

Anticonvulsants are also sometimes prescribed to treat irritability and aggression, but only one trial demonstrated the effectiveness of valproic acid as treatment for explosive temper and mood lability in disruptive children and adolescents (N = 20; Donovan et al., 2000). The effect size of this study was underpowered and the study is usually not included in any meta-analysis (Tourian et al., 2015). Lithium did not show to have any beneficial effects in the treatment of children with severe mood dysregulation (Dickstein et al., 2009). While some medications may be useful in the treatment of DMDD, the use of pharmacology alone is not recommended (Gisunterman, Cohn, & Weizman, 2018).

The conceptualisation of chronic irritability as the response to frustrative non-reward and threat, and the subsequent identification of the neurobiological correlates of DMDD, have led to novel treatments aimed at altering the circuitry by changing the responses to such stimuli (Driver & Thomas, 2018). Early studies indicated that behavioural modification treatment (n = 33, M = 8 years, SD = 2.1 years), cognitive behavioural therapy for affect regulation and parent-training intervention for recurring for managing recurrent defiant behaviours (N = 7, M = 8.7 years, SD = 1.6 years) were promising adjunct treatment modalities in children with ADHD and severe mood dysregulation (Waxmonsky et al., 2008; Waxmonsky et al., 2013). Computer training aimed at training hostile interpretation bias to shift judgements of ambiguous facial expressions from predominantly angry to predominantly happy seemed to decrease irritability and changing activation in the lateral orbitofrontal cortex (Stoddard et al., 2016). One pilot randomised trial assessed the preliminary efficacy of interpersonal psychotherapy (IPT) for mood and behaviour dysregulation, an adapted version of IPT for depressed adolescents, and found that IPT reduced outbursts and irritability and improved interpersonal interactions in children with DMDD between the ages of 12 and 17 (N = 19; Miller et al., 2018). Similarly, group therapy focussing on emotion regulation education was found to decrease emotive derangement and irritability in students with DMDD (Sheybani, Mikaeili, & Narimani, 2018). These and other ongoing studies that focus on the neurobiological and social mechanisms that mediate chronic irritability and angry outbursts will hopefully engender novel and effective treatment regimens for the newly defined disorder.

The definition of the new diagnostic category was intended to address the needs of youths that are severely impaired by their chronic, volatile irritability and anger and don't satisfy all the criteria for the *DSM-IV* (APA, 2000) diagnostic categories and thus have not received appropriate treatment (Axelson, 2013; Lochman et al., 2015). Its addition to the

DSM-5 (APA, 2013) was met with criticism from the professional community, who expressed reservations about the limited research upon which the category is based (developed mainly from research on severe mood dysregulation, with which there is only partial overlap), the high degree of similarity and comorbidity between DMDD and other disorders (i.e., oppositional defiant disorder), the possibility of further contributing to increased rates of mental health diagnoses and medication use among children, and emerging evidence of limited reliability (Lochman et al., 2015). Axelson et al. (2012) raised concerns about the utility of the new category when they found that it was difficult, if not impossible, to distinguish DMDD from oppositional defiant disorder or conduct disorder. Disruptive mood dysregulation disorder also had limited diagnostic stability (only 19% of children initially diagnosed with DMDD met the criteria at 1- and 2-year follow-ups) and was not associated with current, future-onset, or parental history of mood or anxiety disorders. Moreover, apart from a few studies on the neural correlates of the disorder (Blair, 2010; Brotman et al., 2006; Brotman et al., 2010; Dickstein et al., 2007; Dickstein et al., 2010; Leibenluft, 2011; Rich et al., 2007; Rich et al., 2008; Rich et al., 2010b; Stringaris et al., 2009), little is known about the aetiology of these symptoms or how the family, social and other environmental factors contribute to the development of DMDD.

While the debate on the use of chronic irritability as a distinct category or rather a symptom or specifier of other disorders continues, it is imperative to understand the mechanisms involved in emotion regulation to find ways to minimise the impairment these children experience. To this end, it is necessary to consider the concept of emotion and emotion regulation.

Chapter 3: Emotion and Emotion Regulation

The ability to regulate emotion and emotion-related behaviour is critical to adaptive, healthy psychosocial functioning and consequently has received significant attention first in developmental and child psychology (e.g., Barret & Campos, 1987; Campos, Campos, & Barrett, 1989; Dodge, 1989; Fox, 1989; Kopp, 1982; Kopp, 1989; Kopp & Neufeld, 2003; Rubin, Coplan, Fox, & Calkins, 1995; Thompson, 1991; Thomson, 1994) and later in adult psychology literature (e.g., Carstensen, Fung, & Charles, 2003; Diamond & Aspinwall, 2003; Gross, 2002; Hay & Diehl, 2011; Lawton, 2001; Kim et al., 2013; Ong & Bergeman, 2004; Tamir, 2011; Zimmermann & Iwanski, 2014). The origins of the scientific inquiry into the regulation of emotion lie in the early and fundamental theories of emotion, such as the James-Lange (James, 1884; Lange, 1887), Cannon–Bard (Cannon, 1927), and Schachter–Singer (Schachter & Singer, 1962) theories of emotion. These theories, which evolved from Darwin's (1872) work, formed the foundation for many subsequent theories that focussed on the management of anxiety and the nature of psychological defences, stress and coping; attachment and the social construction of emotion (Averill, 1980; Davidson, 1984; Ekman, 1984; Fridja, 1986; Izard, 1972; Izard, 1977; Lang, 1979; LeDoux,1984; Panksepp, 1982; Plutchik, 1980; Scherer, 1984). The field of emotion regulation emerged as an independent field in the mid-1990s (Gross, 2002; Gross, 2015; Tull & Aldao, 2015). Extensive research in this area has produced diverse perspectives and an extraordinary body of knowledge on the nature of emotion regulation spanning various subfields of psychology, including developmental, social, and clinical psychology, and, more recently, cognitive and affective neurosciences and psychophysiology (e.g., Aldao, Nolen-Hoeksema, & Schweizer, 2010; Banks, Eddy, Angstadt, Nathan, & Phan, 2007; Chang, Schwartz, Dodge, & McBride-Chang, 2003; Cole, Michel, & Teti, 1994; Cummings & Davies, 1996; Diamond & Aspinwall, 2003; Eisenberg, 2000; Eisenberg et al., 1995; Eisenberg et al., 2001; Goldin, McRae, Ramel, &

Gross, 2008; Gratz & Roemer, 2004; Gross. 2001; Gross, 2015; Gross & Muñoz, 1995; Izard, 1990; Koole, 2009; Lopes, Salovey, Côté, Beers, & Petty, 2005; Rubin et al., 1995; Shields & Cicchetti, 1997; Tamir, 2011). Despite the ubiquitous interest in the nature of emotion and emotion regulation, the conceptualisation and definition of these psychological constructs, and consequently, the research conducted, depend heavily on the paradigm from which it is approached. Any discussion on the nature of emotion regulation, however, presupposes a comprehension of what is denoted by the term emotion. Two of the most influential contributors to contemporary thinking about the nature of emotion are Charles Darwin and William James.

Theories of Emotion

In his book, *The Expression of the Emotions in Man and Animals* (1872), Darwin concluded that emotions, like physical structures, evolved to serve specific functions and helped the organism prepare for action. In particular, Darwin emphasised the communicative function of emotions via a range of postural, facial, vocal and other non-verbal cues. These communications, he argued, motivated and regulated social behaviour in a way that is both adaptive and appropriate to environmental demands and so increased the organism's chances of survival (Darwin, 1872). Darwin's work formed the foundation for many subsequent researchers, most notably Ekman (1957, 1984, 1992a, 1992b, 1999), Izard (1972, 1977), Tomkins (1962), Plutchik (1980) and LeDoux (1984, 1992, 1996), who continued to develop his ideas from an evolutionary perspective. Whereas Darwin was mostly interested in the expression of emotion, James was more concerned with the experience of emotion (James, 1884).

James also acknowledged the evolutionary contributions to the nature of emotion but focused primarily on the relationship between physiology and subjective state. According to James (1884), emotions are adaptive physiological and behavioural response tendencies that

are elicited by evolutionary significant stimuli. The bodily changes, James suggested, followed “directly the PERCEPTION of the exciting fact" (James, 1884, p. 189). While these tendencies are often expressed, they are not always, indicating the individual’s capacity to regulate their emotional response tendencies. James’ view that feeling states are associated with innate and distinct patterns of autonomic nervous system arousal, which today would be considered a psychophysiological perspective, still guides contemporary theories on the nature of emotion, such as non-cognitive, somatic feedback, and perceptual theories of emotion (e.g. Damasio, 1994, 1996, 2001; Prinz, 2004a, 2004b; Robinson, 1995, 2004; Zajonc 1980, 1984).

Up until the early 1980s, the focus of emotion research was primarily on the structural or form aspects of emotional processes (Campos et al., 1989; Holodynski & Friedlmeier, 2006). According to the structuralist paradigm, emotion is a discrete state denoting a “reaction to an emotion-specific cause” (Holodynski & Friedlmeier, 2006, p. 13). Emotional behaviour, in turn, is viewed simply as “a readout of internal feeling states or of central nervous system programs” (Witherington & Crichton, 2007, p.628). This conceptualisation considers emotion to be exclusively intrapersonal. The basic premise of this approach is that each emotion comprises of an objective (i.e., physiological and/or expressive) component and a subjective or experiential component, with the latter depending on the internal perception of the former (Holodynski & Friedlmeier, 2006). Arnold (1960) elaborated James’ concept of perception to include the process of appraisal, which she considered "direct, immediate, non-reflective, non-intellectual, [and] automatic" (Arnold, 1960, p. 174) judgments of events in the environment as good or bad. Researchers in the structuralist tradition are interested in topics such as the identification of primary emotions, how emotions are expressed in the face and body (including posture, gaze, prosody and gestures), the neurophysiological and cognitive underpinnings of emotions, and the cross-cultural universality of emotions (e.g.,

Ekman, Friesen, & Ellsworth, 1972; Izard, 1977; Panksepp, 1982, 1998; Rolls, 2002; Tomkins, 1962). Structuralist theories have advanced our understanding of the constituent processes involved in emotion and emotional development, but have been criticised for reducing emotion to a mere derivative of perceptual and cognitive processes. The critique also included viewing emotion regulation as purely a consequence of physiological and cognitive maturation, as well as underestimating the reciprocal influence of emotion on the internal and external environment (Bornstein, Arterberry, & Lamb, 2013; Campos et al., 1989). These limitations prompted the expansion of emotion research to analyse not only its form but also its function in the individual's activity regulation within his or her environment (Holodynski & Friedlmeier, 2006).

Pivotal to functionalist theories is the conceptualisation of emotions as complex multicomponent, adaptive systems best organised, not around autonomic signatures, neurological patterns or facial prototypes, but rather around the functions they serve (Saarni, Campos, Camras, & Witherington, 2006; Witherington & Crichton, 2007). The components involved depend on the specific functionalist theory, but typically entail the individual's goals and objectives, the context-specific events that affect those goals, the individual's appraisals of the event's significance in terms of his or her goals, and the expressive and behavioural actions used to sustain or alter the relationship between the organism and the environment (Witherington & Crichton, 2007). Functionalists view emotional development as shifts in some or all of these components, such as novel ways of appraising environmental events or the attribution of different or new significance to the organism-environment relationship. Modifications may also include the development of new goals and the new ways of coping with and working towards these goals, and different ways of communicating affect to other individuals (Barrett & Campos, 1987). The functionalist approach views the relationships between the components, rather than the elements themselves, as fundamental

to emotion and the development thereof (Witherington, Campos, & Hertenstein, 2001). Within this view, social encounters offer the most significant contexts for practising the strategies of emotion regulation. The efficiency of these abilities is contingent on the responses of social partners and the demands of the social environment, as they are pertinent to the individual's goals (Thompson, 1994).

Functionalist definitions of emotions stem, in part, from ethological theories, which emphasised the adaptive function of emotion in human evolution (Bornstein et al., 2013). It is not the distinctive facial expression or physiological arousal that distinguishes one emotion from another, but the functional relationship between the environment and the individual (Witherington & Crichton, 2007). The focus is on action and action tendencies, and emotion is seen to have both intrapersonal and interpersonal regulatory effects. For example, joy motivates a person to continue his or her activity, while simultaneously indicating to others the desire to maintain interaction.

Campos et al. (1989, p. 395) succinctly capture the functionalist orientation when they described emotions as "processes of establishing, maintaining, or disrupting the relations between the person and the internal or external environment, when such relations are significant to the individual". According to Gross and Thompson (2007), emotion is a person-situation transaction that demands attention, is evaluated to be immediately relevant to currently active goals, and results in a coordinated yet flexible whole-body, multisystem (i.e., physiological, experiential and behavioural) response to the ongoing person-situation transaction. These responses or changes are what Gross and Jazaieri (2014) refer to as emotional reactivity. The emotion often changes the situation that prompted the response, to begin with, demonstrating the reciprocal relationship between emotion and the environment. Gross and Thompson (2007) refer to this as the modal model of emotion (see Figure 3).

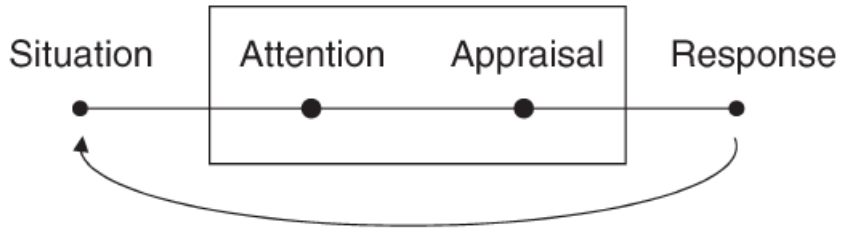

Figure 3. The Modal Model of Emotion. *Note*: From "Emotion Regulation: Conceptual Foundations," by J.J. Gross and R.A. Thompson, 2007, in *Handbook of Emotion Regulation* (p. 6). J.J. Gross (Ed.), New York, NY: Guilford Press. Reprinted with permission of Guilford Press.

From a functionalist perspective, events may become meaningful to the individual in various ways (Campos et al., 1989). The event may be directly relevant to the individual's goals or objectives, which may be diverse and changing, transient or enduring, central or peripheral to one's sense of self. Objectives and goals may be conscious and complex or unconscious and simple, widely understood/shared or idiosyncratic (Gross, 2007; Thompson, 1994). Additionally, events may carry meaning because of the emotional messages from social partners (i.e., facial expressions, prosody or gestures) or the hedonic quality of the stimulus (i.e., people are often, but not always, motivated to avoid pain or seek pleasure or be soothed). Regarding intrapersonal processes, emotion may influence a person's perception, cognition and motivation (i.e., it is linked to executive functions), causing an adjustment in one's evaluation of a situation and therefore altering behaviour. Interpersonally, emotions function to regulate or guide an individual's action tendencies and actions (i.e., facilitating them when appropriate or desirable, redirecting them when needed, and preventing them dangerous or inappropriate), as well as elicit similar emotional states from others in the environment (i.e., social referencing and emotional contagion). Individuals read and interpret emotional cues from others and adjust their emotional expression and behaviour to achieve their social objectives. Emotion can also foster emotional bonding and attunement when

brought about by the approval of the social cues from others or result in repulsion by emotional rejection and abuse (Campos et al., 1989). Finally, emotion also regulates reactions to the environment that may be biologically and/or psychologically adaptive (Bornstein et al., 2013), with each adaptive encounter posing unique demands and bringing about unique "action-in-context processes" (Witherington & Crichton, 2007, p.631).

Functionalist theories of emotion stressed the importance of emotion in the preparation of necessary behavioural responses, regulation of attention and decision-making processes, enhancement of memory for significant events, and the facilitation of interpersonal interactions (Gross & Thompson, 2007). Functionalism was instrumental in the evolution of psychology by shifting the focus from the subjective to the objective in the study of emotion, behaviour and cognitive processes, and still predominates modern research on emotion (e.g., Beauchaine & Zisner, 2017; Farb, Chapman, & Anderson, 2013; Hutcherson & Gross, 2011; Keltner & Gross, 1999; Lench, Bench, Darbor, & Moore, 2015; Panksepp, 2011, 2016; Porges, 1997; Weisfeld & Goetz, 2013), along with constructionism (Beauchaine & Haines, 2019).

Constructionists maintain that what humans perceive as discrete emotions are not determined by specific regions in the brain, but constructed in the moment through highly individualised memory and learning processes. This perspective asserts that emotions and other experiential states, such as perception and cognition, are the products of interactions between core sensory and neural networks, which we interpret, categorise, and infer meaning from based on prior experiences (Barrett, 2009, 2017b; Mandler, 2013; Russell, 2009). Constructionism distinguishes between core affective processes and emotions (Beauchaine & Haines, 2019). Core affective processes refer to general feelings of valence (pleasure or displeasure) and activation (calm to high activation), whereas emotions are more specific feeling states, such as anger, fear and shame, which can be described in terms of valence and

arousal (Barret, 2016; Ekman & Cordaro, 2011; Lindquist & Barrett, 2012). Constructionist theories of emotion are limited by their reliance on research using functional magnetic resonance imaging, which has poor temporal resolution, to falsify their core propositions (Beauchaine & Haines, 2019). Constructionists, on the other hand, have criticised functionalists for non-falsifiable teological arguments regarding the adaptive evolution of emotion, especially given that evolutionary biologists had relinquished the notion of purposeful design more than a century ago (Barrett, 2017a). Further critiques are aimed the locationist basic emotion perspective (functionalists link neural structures and networks to particular emotional states) and the extent to which animal research furthers our understanding of emotional states and processes in humans. While acknowledging that these criticisms warrant considerations when evaluating functionist theories, proponents of functionalism maintain that functionalism is often oversimplified and misconstrued, and that it is in fact compatible with, and informs, constructionism (Beauchaine & Haines, 2019; Farb et al., 2013; Gross & Feldman Barrett, 2011). Both functionism and constructionism have provided key insights into emotion and continue to further our understanding of how emotions either enhance or undermine effective, adaptive psychosocial functioning (Gross, 2015; Thompson, 1994). Emotions are unhelpful when they are inappropriate to the situation, disproportionate in intensity level, or problematic in frequency, providing the impetus to monitor, evaluate and modify our emotional reactions.

Emotion Regulation and Dysregulation

The burgeoning interest in the research of emotion regulation underscores the acknowledgement that emotional responses are rich, complex processes that vary significantly between individuals and of which discrete emotion is only a component. With the paradigm shift from structuralism to functionalism came renewed interest in James' observation that response tendencies are not obligatory. While emotions can be disruptive

and demand awareness, a quality Frijda (1986) calls “control precedence”, they compete with various other processes and can be modulated using executive functions (such as “hot” and “cold” processing) in a number of ways (Hongwanishkul, Happaney, Lee, & Zelazo, 2005; Poon, 2018; Prencipe et al., 2011; Roiser & Sahakian, 2013; Zelazo & Carlson, 2012). Despite the abundance of papers published on this topic, there is a lack of consensus among researchers and theorist on the conceptualisation and definition of the term emotion regulation. Nonetheless, it is possible to extract aspects that various definitions have in common (Thompson, 1994).

Definitions focus either on the role of emotion in regulating internal processes (e.g. attention, memory and action readiness) and social communication, enabling the individual to respond promptly to environmental demands, or they emphasise the way in which emotions are regulated (e.g. cognitive control, internalisation of social expectations). Some definitions stress both aspects. Cole et al. (1994, p.76), for example, define emotion regulation as “the ability to respond to the ongoing demands of experience with the range of emotions in a manner that is socially tolerable and sufficiently flexible to permit spontaneous reactions as well as the ability to delay spontaneous reactions as needed”. Thompson (1994) offers a more comprehensive definition: “Emotion regulation consists of the extrinsic and intrinsic processes responsible for monitoring, evaluating and modifying emotional reactions, especially their intensive and temporal features, to accomplish one’s goals”. Included in this definition are numerous features of emotion regulation processes: emotion regulation may entail the maintenance, enhancement, inhibition, or dampening of emotional arousal and incorporate both self-management and other-management strategies (Thompson, 1994). Although it may affect the discrete emotion that is experienced, most often emotion regulation affects the temporal and intensive aspects of emotion.Finally, emotion regulation is functional in terms of the individual’s objectives for a specific event or situation. Gross and

Thompson (2007) offer a complementary model that distinguishes, and provides an organising principle for, the regulatory processes that influence emotion generation at various points of the emotion generation process. According to the process model, adaptive regulation requires three crucial factors. Firstly, emotional awareness, which appears to increase the array of accessible regulation strategies, as well as the flexibility with which they are employed. Secondly, emotion-regulation goals, in other words, maintaining, augmenting or diminishing the extent or intensity of emotion experience and expression, or physiological arousal. Lastly, emotion-regulation strategies (see Figure 4), which involve situation selection, situation modification, attentional deployment, cognitive change and response modulation. In any given cycle of emotion-generation, an individual moves from left to right through the model as a function of time, but it is not uncommon to engage in behaviour that involves various strategies, as opposed to just a single strategy, during a cycle.

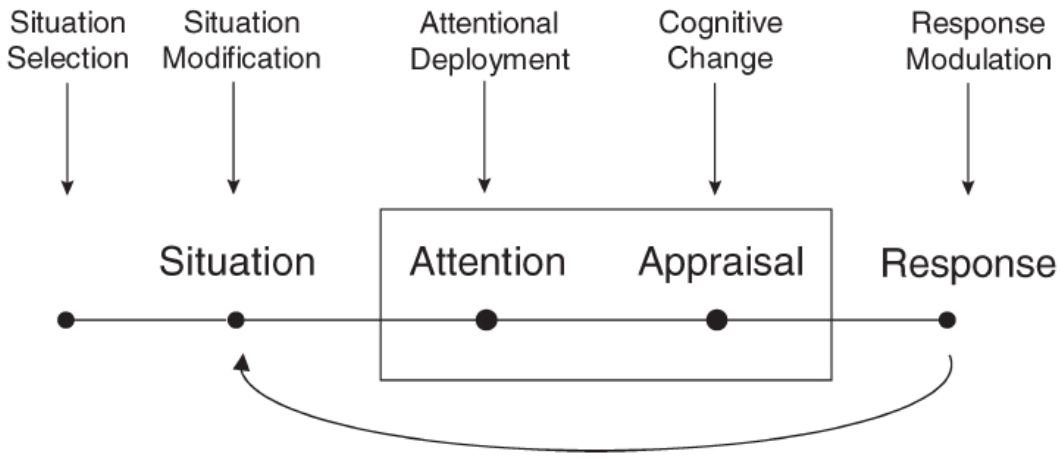

Figure 4. A Process Model of Emotion Regulation that emphasises five families of emotion regulation strategies. *Note*: From "Emotion Regulation: Conceptual Foundations," by J.J. Gross and R.A. Thompson, 2007, in *Handbook of Emotion Regulation* (p. 10). J.J. Gross (Ed.), New York, NY: Guilford Press. Reprinted with permission of Guilford Press.

Gross and Jazaieri (2014) consider the adaptive quality of emotion regulation to be dependent on the context, the individual and the specific emotion, but not all theorists agree.

There is considerable disagreement as to whether the term emotion regulation should refer only to adaptive or optimal functioning or whether it should also include a lack thereof (i.e., maladaptive emotion regulation). Restricting the term to only include optimal functioning overlooks emotion regulation in at-risk or clinical populations and contributes to the perception of positive emotions as "good" and negative emotions as "bad" (Cole et al., 2014). In fact, Kopp (1989) asserts that negative emotion and discomfort, in particular, is unavoidable and can be beneficial as it serves as the motivation for the infant to learn to modulate and tolerate negative emotion experiences in order to maintain physiological and psychological homeostasis. Similarly, Bridges, Denham, and Ganiban, (2004) maintain that all emotional regulation is adaptive in as far as it allows individuals to function within their environments. Over time, individuals develop stable styles of emotion regulation, which may later become maladaptive and, if not flexible enough to respond to changes in the environment, could lead to emotion dysregulation.

Emotion dysregulation are standard regulatory processes that operate in a dysfunctional manner and involve a cost that is evident in impairment or limitation of functioning (Cole, Martin, & Dennis, 2004). Emotion dysregulation includes emotion lability, blunted affect, high levels of emotional distress, inappropriate affect, the predominance of one emotion, and lack of emotional awareness. While it may fill a defensive or communicative function for the individual in a particular context, difficulty in regulating emotion increases the risk of externalising problem behaviour (i.e. aggressive and disruptive behaviour), problematic interpersonal relationships, and psychopathology later in life (Beauchaine, 2015a; Keenan, 2000).

In a longitudinal study of 440 children, both male and female, between the ages of two and seven, children with higher levels of emotion regulation evidence exhibited decreases in their subsequent externalising behaviour. This sample was, however,

overselected for externalising behaviour problems, and thus might not be representative of community samples (Blandon, Calkins, Grimm, Keane, & O'Brien, 2010). Another study of 56 two-year olds found that toddlers who are prone to anger or frustration and lack emotion regulation skills, whether their own or with the help of caregiver practices, were more likely to display conflict-oriented behaviours in early interactions with peers. Some of these behaviours, such as venting and aggression, were infrequent though, and were to be expected in a typical middle-class sample. It was also not clear whether these behaviours were representative of the children's typical behaviour or were limited to the testing environment, nor whether the distress/venting interaction was the result of extreme emotional reactivity or lack of emotion regulations strategies, or both. Data was generated using a small, middle-class sample in two brief laboratory sessions, so generalisability to other situations and populations is limited (Calkins, Gill, Johnson, & Smith 1999). Emotion regulation was also found to predict problematic behaviour, of which was influenced by differences in temperaments. A sample of 68 four-year olds was classified as either introverted or extroverted, based on observations of their interaction with peers. Ratings of emotion regulation and emotional and behavioural problems were also acquired from their mothers. Introverted preschoolers who were emotionally dysregulated, exhibited more internalising problems, whereas extroverted preschoolers who were emotionally dysregulated, had more externalising problems, such as disruptiveness (Rubin et al., 1995). However, comparisons were drawn between extreme groups of children. For instance, those who were highly emotionally reactive and also poor soothers, were contrasted with children who were emotional unreactive and also good soothers. Subsequently, no inferences could be made about how soothability and reactivity interact to produce maladptive outcomes.

Increasingly, data from older children suggests that emotion dysregulation is not only associated with the onset of mental health problems, but is also a risk factor for the

development of psychopathology. In a large, diverse sample of adolescents, emotion dysregulation (measured by three distinct processes of emotional understanding, dysregulated expression of sadness and anger, and ruminative responses to distress) predicted increases in anxiety symptoms, aggressive behavior, and eating disorders after controlling for baseline symptoms at two points over a period of seven months. Emotion regulation did not predict depressive symptoms. On the contrary, not one of the four forms of psychopathology predicted increases in emotion dysregulation after controlling for baseline emotion dysregulation (McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011). However, the findings of this study are limited by reliance on self-reported symptoms of psychopathology and emotion regulation. The study also only measured three aspects of emotion regulation, while other aspects of emotion regulation, such as management of fear, might also be relevant to risk of psychopathology (Mennin, Heimberg, Turk, & Fresco, 2005). Similarly, in a study of 314 third- and seventh-grade children (ages 8 to 12), children with a ruminative response style displayed increases in depressive symptoms over a period of 6 weeks. The association between rumination and increase in depressive symptoms was not moderated by intial levels of such symptoms, which suggests that rumination, an aspect of emotion dysregulation, predicts the onset of childhood depression (Abela, Brozina, & Haigh, 2002). A limitation of this study is that it also relied on self-reported measures of depressive symptoms. In addition, it only focussed on rumination, which is likely only one of many factors that play a role in the etiology of depression. While more research is necessary to establish the predictive role of emotion dysregulation, it seems likely that emotion dysregulation contributes to the development of mental health disorders and that, by implication, the development of emotion regulation processes and functioning may serve as a protective factor over the lifespan.

Several elements appear to shape the development of emotion regulation processes and functioning over the lifespan. These elements are biobehavioural processes, individual differences in temperament, developmental level and factors external to the individual that facilitate emotion regulation (e.g., social context, past social experience and learning, belief systems, situational factors, and culture; Calkins & Hill, 2007; Campos, Frankel, & Camras, 2004; Thompson, 2011; Thompson, Lewis, & Calkins, 2008).

The development of emotion regulation involves maturational changes in various related processes, including physiological arousal, neurological activation, attention processes, cognitive appraisal or attributional processes, and action tendencies. Underlying these processes are structures of nervous system organisation that have evolved to maintain homeostasis through the interaction between excitatory and inhibitory mechanisms (Shipman, Schneider, & Brown, 2004; Thompson, 1994). As human infants, like most mammals, are altricial, several of these structures are functionally immature at birth. The infant, therefore, depends on the caregiver's mature nervous system to compensate for its underdeveloped nervous system, creating a model of "symbiotic regulation" (Porges, 2011, p. 281). The caregiver supports the biobehavioural needs of the infant (e.g., food, warmth and protection) and the infant's behaviours trigger specific physiological processes (e.g., the release of oxytocin) in the caregiver to foster a bond, provide emotional comfort for, and support the health of, the caregiver (Porges, 2011). Despite the dominance of extrinsic emotion regulation in the first few months, even very young infants are equipped with an innate, yet limited, emotional repertoire to alleviate distress in the temporary absence of caregivers and protect them from overwhelming physiological arousal. Kopp (1989, p. 344) refers to them as "preadapted" or "biologically derived species-typical" programs. Examples of such self-soothing techniques include gaze aversion, head turning, eye closing, tactile stimulation, and non-nutritive sucking (Kopp, 1989; Thompson, 1994). There are, however, major constraints

to the immature infant's self-regulation strategies: Firstly, levels of arousal need to be comparatively low for the infant to manage discomfort on its own. Secondly, emotion regulation at this age is not planned or monitored, which means any regulation strategy that the infant can employ is recruited. Lastly, the infant is unable to address the cause of its distress, and therefore primarily relies on the adult to regulate its environment and experience, including emotion and state organisation (Cole et al., 1994; Kopp, 1989). Failures in this process of co-regulation of emotion can lead to enduring patterns of emotion dysregulation (Cole et al., 1994). Malfunctions in the co-regulation process may be the consequence of troubled and vulnerable relationships (e.g. dysfunctional parenting and environments) or caregiver and infant characteristics (e.g. parental psychopathology, temperament, discrepancy between infant's resources for emotion regulation and that which the situation demands, such as autism, visual impairment, and the like). In the first few years of life, the progressive maturation of the autonomic, cognitive and motor systems allows for the transition from other or extrinsic regulation to a higher degree of self or intrinsic regulation (Gross & Thompson, 2007; Porges, 2011). Most mammals, however, continue to rely on social interaction (i.e. trusting friendships and supportive, loving partnerships) for optimal state regulation and wellbeing. The infant-caregiver dyad provides the context for the socialisation of emotion regulation.

Attachment and Emotion Regulation

A considerable amount of the research on the development of emotion regulation skills and prosocial behaviour focuses on the construct of attachment (i.e., the quality of the affective bond between infant and caregiver), which is derived from traditional attachment theory, particularly the Bowlby/Ainsworth theory of attachment (Ainsworth, 1978; Bowlby, 1958, 1969). The underlying supposition of attachment theory is that humans are biologically disposed to form close emotional bonds to maintain proximity to the caregiver and so

increase the probability of survival (Bowlby, 1958; Cassidy, 1994; Pietromonaco & Barrett, 2000; Main, 1990; Saarni, 1999). During the first year, and based on repeated daily experiences with the caregiver, the infant develops of mental representations of "self" and "others", referred to as "working models". Working models help the individual understand and predict the environment, engage in behaviour that promotes survival (such as seeking and maintaining proximity to an attachment figure), and create a psychological sense of perceived safety (Bretherton, 1985). From this perspective, emotion organises the security or insecurity of the caregiver-infant relationship, which is then internalised as a working model and taken into subsequent relationships (Bretherton, 1985; Pietromonaco & Barrett, 2000). During early life, the development of adaptive emotion regulation patterns is dependent on adequate, responsive and reciprocal social interactions (Cole et al., 1994; Porges, 2011).

The emotional communication between the primary caregiver and the infant provides the framework for the child's understanding and organising of affective experience. The infant whose caregiver responds sensitively to a range of affective signals most of the time, without selective ignoring, is more likely to be securely attached and communicate his or her emotions openly, actively, and flexibly to the parent. The child, therefore, experiences negative affect (e.g. fear and anger) as tolerable and useful in alerting the caregiver during times of distress, and not as intolerable or threatening. Positive emotions experienced during the interaction with the parent function to communicate the infant's desire to maintain the relationship. The caregiver's sensitive responses enhance the child's sense of efficacy in regulating his or her own feeling states. These infants develop flexible emotion regulation in response to changes in the environment, without systematically distorting or hiding emotions. Certain attachment-related experiences, however, may contribute to the minimisation or heightening of emotion (Cassidy, 1994).

In her seminal “Strange Situation” study, Ainsworth (Ainsworth & Bell, 1970) illustrated the profound effects of attachment style on behaviour. During the study, researchers observed the behaviour of 12- to 18-month old toddlers who were introduced to laboratory playroom with their mothers, where they were later joined by a stranger. While the stranger played with the infant, the mother left briefly, and then returned. During a second separation, the infant was left completely alone, and then joined first by the stranger and then the mother. Upon reunion with their mothers, some children displayed ambivalence - while seeking contact, they angrily kicked or swiped at the mother. Another group had avoided the mother, although they had often searched for her during separation. Analyses of the data revealed that both these groups of children had less harmonious relationships with their mothers at home than those who sought proximity, contact or interaction upon reunion. Based on these observations, Ainsworth (Ainsworth & Bell, 1970) defined three attachment styles: secure attachment, ambivalent-insecure attachment, and avoidant-insecure attachment. Later, Main and Solomon (1986) expanded Ainsworth’s attachment classification by suggesting that when activation of the attachment system regularly results in rejection, the infant develops a strategy (i.e. avoidant-insecure attachment style) that minimises attention to the attachment relationship. Thus, the importance of the parent as a source of comfort, as well as his or her apparent need for the caregiver, is diminished. The infant achieves this by minimising negative emotions, such as distress, anger or sadness to maintain sufficient proximity to the attachment figure to ensure protection. In contrast, a minimally or inconsistently available caregiver results in an emotion regulation strategy that increases the infant’s bid for attention (i.e., insecure/ambivalent attachment style). The child amplifies the importance of the attachment relationship and exhibits extreme dependence on the caregiver. To this end, the infant heightens negative emotionality as a strategy to gain the parent’s attention. This heightening of negative emotionality may be chronic and exaggerated, as the infant realises

that allowing him- or herself to be soothed and calm in the presence of the attachment figure may jeopardise contact with the frequently unavailable parent (Cassidy, 1994). One such strategy is exaggerated fearfulness in response to comparatively benign stimuli, which increases the probability of obtaining the attention of the inconsistently available caregiver in the case of actual danger and increases the likelihood that the child's attachment needs will be met. Attention regulation may contribute to this process in that the child selectively attends to the threatening aspects of the environment (Main & Hesse, 1990). This kind of strategy may become dysfunctional if the negative emotionality is so ubiquitous that it jeopardises the continuation of the relationship or interferes with other developmental tasks (Cassidy, 1994). Consequently, the heightened negative emotionality (i.e., the chronic irritability or anger and frequent temper outbursts) that define DMDD may be interpreted as an emergent property of an insecure-ambivalent infant-caregiver dyad. This interpretation poses an alternative explanation for the attention bias to angry faces in children with severe mood dysregulation offered by Thomas et al. (2014). It is also conceivable that the internalisation of an insecure/ambivalent working model may contribute to the poor peer and social functioning seen in children with DMDD. These are questions that the current study seeks to investigate.

While the Ainsworth classification system (Ainsworth et al., 1978) provides some insight into developmental psychopathology, Porges (2003b) argues that this attachment schema only forms a small component of social behaviour and that traditional attachment theories fail to articulate the mechanisms that mediate social engagement between individuals. The polyvagal theory of emotion provides a plausible explanation of how physiological state restricts the range of affective and behavioural experience, and hence, determines contingent social behaviour. Although the theory has features that could be interpreted as structural (neurophysiological and neuroanatomical) and functional (adaptive

within changing environmental demands), Porges (2015, personal communication) maintains that the polyvagal theory informs both structural and functional theories of emotion. The next chapter provides an overview and critique of Porges' (Porges, 1995, 1997, 1998, 2001) polyvagal theory.

Chapter 4: The Polyvagal Theory

The polyvagal theory (Porges, 1995, 1997, 1998, 2001) relates the evolution of the mammalian autonomic nervous system to affective experiences and processes that are key components of social behaviour. The theory suggests that physiological state determines, and therefore limits, the experience and expression of emotion and social behaviour. Specifically, it emphasises the role of the bidirectional communication between the heart and the brain, via the vagus nerve, in affective and behavioural state regulation. While the systematic investigation of the body-brain connection in the expression of emotion forms the scientific basis of modern psychophysiology, the notion of a dynamic and reactive interface between physiological and psychological processes dates back to the 19th century. Porges' theory builds on the revolutionary work of scientists, such as Darwin, Bernard, James, and Jackson (Porges, 1995; Porges, 1997; Van der Kolk, 2014).

Darwin speculated that many facial expressions and emotions are evolutionary remnants of earlier survival activities, with the purpose of initiating a physiological and behavioural response that would serve survival (Valent, 1998). Rage and terror particularly, he suggested, foster the mobilisation behaviours necessary for "fight or flight". Although he could not identify the exact neurophysiological mechanisms associated with emotion, Darwin did acknowledge the dynamic feedback loop between specific structures of the brain and the peripheral organs, especially the heart, through the "pneumogastric" nerve (which was later renamed as the vagus nerve), to facilitate the autonomic activity linked to emotions (Porges, 1995). Similarly, Bernard viewed the heart as the source of afferent stimulation to the brain, capable of contributing or changing emotional state (Porges, 1995). Walter Cannon later extended Darwin's description of rage and terror to include sympathetic-adrenal excitation to prime the animal for fight or flight in the face of perceived threat or danger (Valent, 1998).

Autonomic Substrates of Emotion

Due to its capacity to mobilise, Cannon's assumption that the sympathetic nervous system is the primary physiological constituent of the autonomic nervous system involved in emotion was generally accepted by researchers who followed (e.g., Ax, 1953; Ekman, Levenson, & Friesen, 1983; Levenson, Ekman, & Friesen, 1990; Schachter, 1957; Schachter & Singer, 1962). The autonomic nervous system innervates smooth muscles and glands and maintains visceral processes, such as cardiovascular activity and internal thermoregulation (Brodal, 2010; Porges 2003). It is commonly described as a balance system consisting of two components, the sympathetic and parasympathetic nervous systems, which are structurally and functionally distinct and react to environmental stimuli in an antagonistic manner. Mainly, the sympathetic nervous system, together with the hypothalamic-pituitary-adrenal (HPA) axis, fosters increased metabolic output to deal with external challenges (such as dangerous or life-threatening situations). In contrast, the parasympathetic nervous system calms the visceral state and contributes primarily to vegetative and restorative processes, such as digestion and reproduction, and conservation of physical energy by slowing the heart rate. For example, to prepare the individual for the muscular reaction necessary to fight back or run away, the sympathetic nervous system rallies the energy reserves of the body: the pupils dilate; heart rate, blood pressure, and breathing rate increase; and blood vessels constrict. Blood is directed away from the intestines to make more oxygenated blood available to the skeletal muscles, heart, lungs and brain and other bodily functions, such as peristalsis, are inhibited. Even blood clotting function is altered: platelets change shape to become stickier to prevent excessive blood loss should injury occur during the process. The parasympathetic nervous system, in turn, quickly restores the normal state of the body once the danger passes: pupils are constricted; heart rate, blood pressure and breathing slow down; and normal bodily functions resume (Brodal, 2010; Porges, 2011).

This "sympathetic-centric" (Porges, 2011, p. 1) theory of the physiological covariates of emotion focuses on the balancing antagonism between the two efferent divisions of the autonomic nervous system on the visceral organs. This approach minimises the importance of visceral feedback to the brain and the influence of the vagus nerve, which is the key component of the parasympathetic nervous system, in the regulation of psychological and behavioural state (Porges, 1995). This arbitrary distinction, Porges argues, also disregards other important aspects, such as the interaction between the sympathetic and parasympathetic processes; the dynamic and adaptive nature of the autonomic nervous system; and the interface between the autonomic nervous system and the environment. More contemporary psychophysiologists have revisited Darwin and Bernard's theory on the role of the dynamic feedback between the heart and the brain in self-regulation at cognitive, emotional, social, and even health, levels (e.g., Beauchaine & Thayer, 2015; Diamond, Fagundes, & Butterworth, 2012; Eisenberg et al., 1995; Kemp & Quintana, 2013; Kok et al., 2013; McCraty & Shaffer, 2015; Porges, 1995; Thayer, Hansen, Saus-Rose, & Johnsen, 2009).

The Polyvagal Theory of Emotion

One such theory, the polyvagal theory (Porges, 1995, 1997, 1998, 2001), expanded the sympathetic-centric view of the autonomic nervous system in order to provide further insight into the complex reciprocal interaction between the autonomic nervous system and the social environment, and its implications for emotion and behaviour.

The polyvagal theory (Porges, 1995, 1997, 1998, 2001) was developed to provide a neurophysiological model for understanding complex psychophysiological processes, such as attention and emotion. The theory posits that the mammalian autonomic nervous system has evolved from that of more primitive vertebrates, such as reptiles, to support interactional social behaviour. Specifically, the theory is concerned with the phylogenetic modifications in the neural regulation of the heart by the vagus nerve and how these changes have evolved to

enable specific adaptive responses (Porges, 1995). The mammalian vagal system differs from that of earlier vertebrates in important ways: firstly, the vagus is not a single nerve as initially thought, but a number of pathways stemming from various areas of the brainstem. Its several branches carry out different roles in visceral function regulation and may have antagonistic outputs to the same organ. The vagus does not, however, only convey motor information from the brain to the organs (called efferent pathways); it also relays sensory information from the organs to the brainstem (called afferent pathways). In fact, about 80 percent of the vagus is comprised of afferent fibres. The vagus is lateralised, originating in right and left regions of the brainstem, and asymmetrical (i.e. the right and left sides execute different functions). The polyvagal theory originated from the identification of two neurophysiologically and neuroanatomically distinct branches of the vagus nerve that developed sequentially (Denver, 2004; Porges, 2011). Porges (2011) used the term "polyvagal" to emphasise the different functional capacities in visceral regulation and areas of origin in the brainstem of these two vagal pathways.

In mammals, the primary efferent vagal fibres originate from two distinct areas in the brainstem: the dorsal motor nucleus (DMNX) in the dorsomedial medulla and the nucleus ambiguus (NA) in the ventrolateral reticular formation (Porges, 2011). Mammalian studies show that these vagal nuclei may have separate central connections and perform independent functions. The fibres from the DMNX are unmyelinated and mostly project to structures below the diaphragm (such as the stomach and intestines), while the fibres from the NA are myelinated and mainly project to target organs above the diaphragm (such as the larynx, oesophagus, bronchi and heart). Myelin is composed mainly of fat (70-85% lipid) and forms a sheath around nerve fibres to insulate the nerves electrically and facilitate selective and rapid neural transmission (Porges, 2015). While vagal pathways from both the DMNX and the NA may influence the heart (i.e., vagal tone), in mammals the primary influences are

from the myelinated fibres originating in the NA (the right side). In reptiles, however, these nuclei are still connected. Porges (1995) argues this is the phylogenetic result of different environmental challenges and survival strategies. Reptiles are slow movers and passive feeders, and as such, their metabolic demands, and consequently cardiac output, are much lower than that of mammals. Mammals actively forage and hunt and adapt to their environment and thus have a metabolic demand four to five times that of reptiles. Due to these differing demands, mammals and reptiles use different vagal strategies to ensure survival (Barbas-Henry & Lohman, 1984; Else & Hulbert, 1981; Regal, 1978).

Reptiles use vagal efferents from the DMNX to the heart. Due to their reduced capacity to produce energy, vagal control of the heart is low during unchallenging situations, as this would further lower energy production. In response to novelty or threat in the environment, reptiles orient and immobilise - they "freeze" all gross motor activity or stay under water for extended periods of time. During these times, vagal influences to the heart via the DMNX are increased and heart rate is slower than during periods of breathing and other motor activities. Reptiles thus have low ambient vagal tone and temporary increases in vagal tone in response to external demands (Jacob & McDonald, 1976).

Mammals, in turn, use NA vagal efferents and these influences to the sinoatrial node of the heart (the heart's pacemaker, which is set faster than resting heart rate) are most profound during unchallenged situations to constrain the energy potential of their metabolic system. When faced with external threats or demands, including metabolically taxing states such as stress, exercise, attention and information processing, vagal inhibition to the heart is actively withdrawn (Friedman & Thayer, 1998; George et al., 1989; Kashdan & Rottenberg, 2010; Thayer, Friedman, & Borkovec, 1996) to increase metabolic output. This tonic slowing of heart's pacemaker earned the myelinated vagus its nickname of "vagal brake" (Porges, 2001). It should be noted that heart rate can be influenced by various factors, including

activation of the sympathetic nervous system. However, sympathetic nervous system influences on heart rate can take up to several seconds, whereas the vagal brake can be withdrawn almost instantly (Saul, 1990).

Depending on the context, the vagal brake promptly inhibits or disinhibits vagal influences to the heart to calm or mobilise the individual. In other words, by regulating the physiological state, the vagal brake allows the individual to quickly interact with or withdraw from the environment, while minimising the metabolic cost of the slower sympathetic nervous system (Appelhans & Lueken, 2006; Porges, 2007). Although this mobilisation response is immediate, it is limited. Should the challenge be prolonged or the intensity of the mobilisation be increased, the sympathetic nervous system is activated (Porges, 2011).

While both reptiles and mammals respond immediately to changes in their environment (called the orienting reflex), only mammals have the additional behavioural option to attend to these changes with sustained attention (to process information) or facial expression and vocalisation (to encourage communication), called neuroception.

Neuroception

Congruent with attachment theorists, such as Ainsworth (1978), Bowlby (1958) and Main (1990), Porges (2015) maintains that humans have a biobehavioural need for safety and social connectedness in their quest for survival. In order for mammals to survive, they must be able to discern whether individuals and situations are safe, dangerous or life-threatening, and communicate and engage with their social unit. The nervous system continually appraises environmental risk by processing information through the senses. However, it is not only the absence of threat or presence of physical safety that induces a feeling of safety. In order to feel safe and calm neural defence systems, the nervous system requires a unique set of cues that indicates that features of safety are present. The human nervous system is profoundly sensitive to subtle emotional shifts in others. Minute changes in the muscles of the face and

the angle of the head indicate if the other individual is relaxed, tense, suspicious, scared, angry, etc. and trigger appropriate internal state adjustments in the observer (Van der Kolk, 2014). This unconscious process, called neuroception (Porges, 2004), may occur involve the subcortical limbic structures of the brain (such as the amygdala), which then interprets and relays the signals to the cortex of the brain for higher level cognitive processing (Morris et al., 1999; Öhman, 2002). Neuroimaging techniques, such as positron-emission tomography (PET) and functional magnetic resonance imaging (fMRI) have helped to identify the structures involved in this process. Areas of the temporal cortex, fusiform gyrus, and superior temporal sulcus are involved in detecting social cues that indicate whether an individual is safe or a threat (Adolphs, 2010; Sprengelmeyer et al., 1998; Winston et al., 2002). The detection of the presence of safety or absence of risk and communication between these areas of the temporal cortex and the amygdala then trigger neurobiological processes that facilitate three hierarchical defence strategies. These survival strategies are supported by three phylogenetically organised subsystems.

Phylogenetic Subsystems

The polyvagal theory (Porges, 1995, 1997, 1998, 2001) suggests that evolution produced a human nervous system that consists of three distinct neural subsystems (see Table 2), each associated with one of the three major adaptive strategies: the dorsal vagal complex (DVC), the sympathetic nervous system, and the ventral vagal complex (VVC). The most primitive system, the DVC, is associated with immobilisation strategies (i.e., passive avoidance behaviours, including behavioural shutdown, fainting, and death feigning) and provides neural regulation via the DMNX. The sympathetic nervous system is linked to mobilisation strategies (i.e., active avoidance behaviours, such as fight-flight behaviours and tantrums). The VVC is behaviourally related to social engagement (i.e., visceral regulation, gaze, facial gestures, vocalisation, listening) and is associated with the mammalian

myelinated vagus, which provides neural regulation via the NA. In this hierarchy, the most recent subsystem (the VVC), associated with prosocial behaviour, is activated first. If this subsystem does not provide safety, the primitive subsystems are employed sequentially (Porges, 1995).

Table 2

Three Phylogenetic Stages of the Polyvagal Theory

Phylogenetic Stage	ANS Component	Behavioural Function	Lower Motor Neurons
III	Myelinated vagus	Social communication, self-soothing and calming, inhibit sympathetic-adrenal influences	Nucleus ambiguus
II	Sympathetic-adrenal	Mobilisation (active avoidance, inhibit dorsal vagal influences)	Spinal cord
I	Unmyelinated vagus	Immobilisation (death feigning, passive avoidance)	Dorsal motor nuclei of the vagus

Note: From "*The Polyvagal Theory: Neurophysiological Foundations of Emotions, Attachment, Communication, and Self-Regulation* (1st ed.)" by S.W Porges, 2011.

The development of this complex system serves to support an increasingly complex social life and enables the organism to react to the environment and others in an adaptive way. Essentially, it tells the individual whether she needs to protect herself, for example when being approached by a stranger, or whether it is safe to engage. When the environment is perceived as safe, the system is responsible for two important functions of the mammalian autonomic nervous system: firstly, the tonic regulation of visceromotor components to conserve bodily resources for the maintenance of visceral homeostasis, which furthers growth and restoration. This is achieved by increasing the influences of the NA vagal efferents to the heart, or applying the vagal break, to reduce heart rate, increase the depth of breathing, inhibit

the defensive mechanisms of the sympathetic nervous system, subdue the stress responses of the HPA axis, and reduce inflammation by modulating immune responses. This basically temporarily "shuts down" the brain's natural vigilance to facilitate physical and emotional intimacy with others, which Porges refers to as "immobilisation without fear" (Carter, 1998; Insel & Young, 2001; Porges, 1998). Secondly, the vagal system regulates the somatomotor components involved in social engagement (i.e., the striated muscles of the head and face that control gaze, facial expression, prosody, head gesture, listening). According to the polyvagal theory, the increased neural complexity is an evolutionary by-product that expands the organism's behavioural and affective repertoire (Porges, 1997) and provides an organising principle for human emotion and behaviour (Porges, 2011). From a polyvagal perspective, prosocial behaviour – engagement, attachment and formation of social bonds – depends on the vagal brake's regulation of the visceral state and can only occur when our neuroception accurately assesses the environmental cues as being safe.

The Social Engagement System

The polyvagal theory places social relationships central to our understanding of mental health, stating that safe connections to and reciprocal engagement with others are fundamental to optimal wellbeing. It suggests that humans seek to calm neural defences by detecting features of safety and that through evolutionary processes, social connectedness evolved as a fundamental biological necessity (Porges, 2015). This quest begins during infancy with the need for soothing and caregiving and continues throughout the lifespan with needs for social relationships to effectively co-regulate. All mammals, including humans, are social beings and hardwired to function as members of a tribe: they group together to reproduce, nurture infants, coordinate food acquisition activities, and defend against mutual enemies (Van der Kolk, 2014). The more effectively the VVC regulates arousal, the better attuned the physiology of each individual will be to that of other members of the tribe. To

effectively attune to and engage with others, a range of visceromotor and somatomotor functions are required (Porges, 2003a), such as a calm and relaxed visceral state (i.e., being pleasurably aroused), proper social orienting, the ability to maintain eye contact, attend to and interpret facial expressions and gestures, attend to and interpret vocalisations and language, assess the other person's emotions and intentions and initiate an appropriate verbal and non-verbal response, such as returning a smile, nodding in agreement, or frowning in empathy (Graziano & Derefinko, 2013). Collectively, these physiological and neural processes, and their related structures (i.e., the vagal brake and special visceral efferents), are labelled the social engagement system (SES; Porges, 2001). This system was proposed to understand deficits in spontaneous social behaviour: many psychiatric disorders involve difficulties in creating effective and satisfying relationships or regulating arousal, such as regularly becoming enraged, overexcited, disorganised or shut down, and usually a combination of both (Van der Kolk, 2014). The social engagement system thus offers a mechanism to explain the integrated neural feedback loop that both regulates social and emotional functioning, and, in turn, may be regulated by social behaviour (Porges, 2003b).

During embryological development, components of the vagus nerve and four adjoining cranial nerves (V, VII, IX, and XI) develop together to form the foundation for a social engagement system (Porges, 2001; 2003b). The system comprises of a control component, the corticobulbar pathways, which stem from the frontal cortex (i.e., upper motor neurons) that regulates the nuclei that originate in the medullary structures (i.e., lower motor neurons) to control behaviours that are involved in the detection, expression, and subjective experience of affect and emotions (see Figure 5).

These functions include regulation of the visceral organs (e.g., heart, bronchi, thymus via cranial nerve X), head-turning muscles (e.g., orientation and social gesture via cranial nerve XI), facial muscles (e.g., eyelid opening, emotional expression via cranial nerves VII

and V), middle ear muscles (e.g., extracting of human voice from background sounds via cranial nerve VII), pharyngeal and laryngeal muscles (e.g., vocalisation and intonation via cranial nerves IX and X), and mastication muscles (e.g., sucking, swallowing and salivation via cranial nerve V).

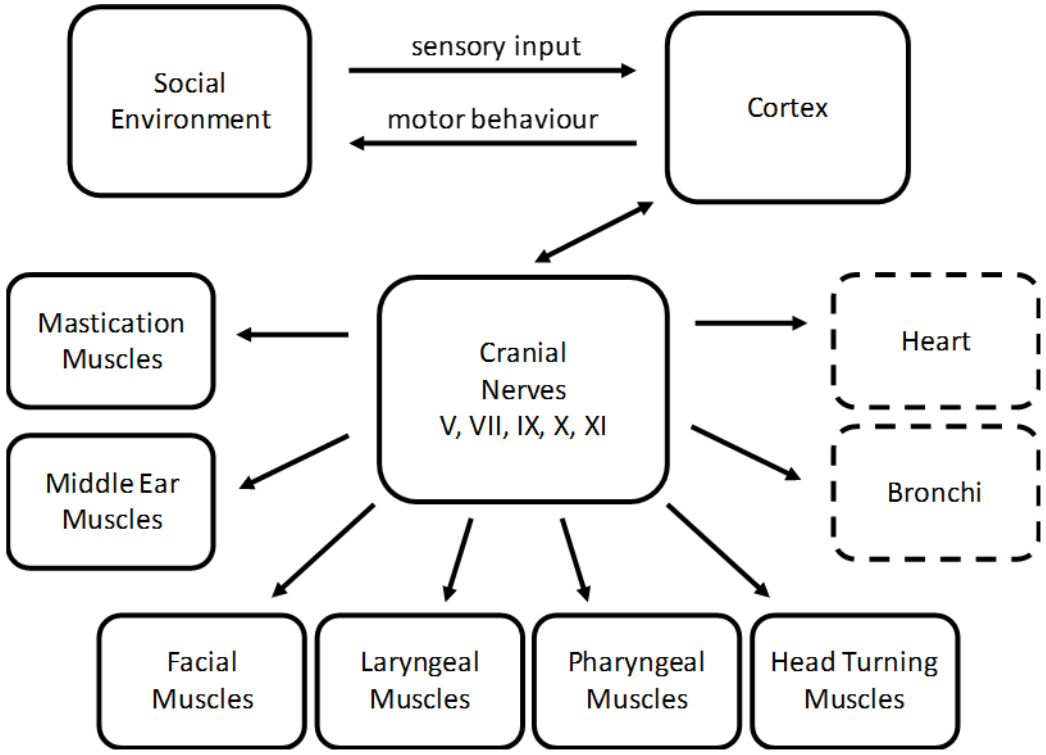

Figure 5. Schematic representation of the Social Engagement System. *Note:* From "*The Polyvagal Theory: Neurophysiological Foundations of Emotions, Attachment, Communication, and Self-Regulation* (1st ed.)" by S.W Porges, 2011.

Together, these muscles operate as a filter that limits social stimuli (e.g., eye contact, facial expression, tone of voice) and with the vagal brake determine engagement or disengagement with others and the environment (Porges, 1998). The regulation of the vagal brake either promotes calm, self-soothing states that support social engagement or support mobilisation (fight or flight). During states of mobilisation, the vagal brake and behaviour

elements of the social engagement system are not readily available. The polyvagal model proposes that a readily accessible and well-functioning social engagement system is imperative for physiological state regulation, as well as the initiation and maintenance of social engagement and also interprets dysregulated arousal and emotion (a common feature of psychiatric disorders) as a failure of the phylogenetically newer vagal system (Beauchaine et al., 2007).

Developmentally, the newer system (i.e., the myelinated vagal system) develops last, leaving it most susceptible to damage and most sensitive to environmental influences (Porges, 2011). Myelination of the vagal motor fibres in humans starts in the course of gestation at about 28 weeks and continues rapidly during the last trimester. Vagal fibres continue to myelinate throughout the first year postpartum and reaches a level similar to those of adults at around one year of age (Pereyra, Zhang, Schmidt, & Becker, 1992; Porges, 2015). One of the most important functions of the myelinated vagus is to down-regulate sympathetic arousal in order to foster calm biobehavioural states necessary for the establishment of social bonds (Porges & Furman, 2011).

Close proximity is critical for social interaction, which depends on the ability to reduce physical distance through voluntary behaviour (Porges, 2003b). If the establishment of social bonds was solely contingent on voluntary motor behaviour, the young infant, with immature neural regulation of the spinal motor pathways and thus limited ability to move away from or toward the caregiver, would be greatly disadvantaged. However, in mammals, not all muscles are regulated by corticospinal pathways. The corticobulbar pathways that control the striated muscles of the face and head are sufficiently developed at birth to express and receive social cues from the caregiver. The infant can thus effectively reduce or increase social proximity through social communication (i.e., facial expressions, eye gaze, vocalisation and head orientation).

As discussed in Chapter 3, the infant relies on the caregiver to regulate both biobehavioural and affective state. The polyvagal theory provides an unequivocal model of how physiological states relate to emotion regulation (both self- and other regulation) and social behaviour. Environmental cues that elicit feelings of safety (e.g., a mother's melodic vocalisations, which signal her calm state and her safe and supportive presence) have the potential to recruit the most recent of the three subsystems (i.e., the ventral vagal pathways) that facilitate the prosocial behaviours of the social engagement system (Porges, 2015). The cues of safety are detected by the infant's higher cortical structures, which inhibit the infant's defence systems and dampens sympathetic influences to the heart in order to foster a calm behavioural state. Regulation of the muscles of the face and head are also enhanced to enable reciprocal social interactions between the infant and the caregiver. These reciprocal interactions function as "neural exercises" (Porges, 2015, p.119) between infant and caregiver's social engagement systems, allowing for the use of social communication to co-regulate and build the infant's capacity to self-regulate and form relationships with others to co-regulate through the lifespan. Certain factors, such as deficits in the visual and auditory systems, or psychopathology of the caregiver, can result in a lack of opportunities for these reciprocal interactions. In terms of emotion regulation theory (discussed in Chapter 3), such failures in the process of co-regulation alter the emotional and social profile and development of the infant (Cole et al., 1994). Children who are in a physiological state of defence, such as children with DMDD, are at risk of misinterpreting cues of risk and safety, and are more likely to exhibit defensive behaviour (e.g., aggression and tantrums).

Various studies (e.g., Beauchaine, 2001; Calkins & Dedmon, 2000; Calkins, Graziano, & Keane, 2007; Calkins & Keane, 2004; Graziano, Keane, & Calkins, 2007; Leary & Katz, 2004; Mezzacappa et al., 1997; Musser et al., 2011; Pine et al., 1998; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996) support the neurobiological model of

social-emotional behaviour proposed by the polyvagal theory (Porges, 1995, 1997, 1998, 2001), specifically the premise that robust vagal tone is a "positive" index of emotional regulation and prosocial behaviour, while attenuated vagal tone seems to point to social and emotional lability, and in some findings, psychopathology (Graziano & Derefinko, 2013; Porges, 2007). Research on children with DMDD (discussed in Chapter 2) that indicate impairments in initial stages of attention, perturbed face-emotion labelling ability and difficulties in affective prosody labelling (e.g., Guyer et al., 2007; Hommer et al., 2014; Rich et al., 2007; Rich et al., 2008; Thomas et al., 2014), could be interpreted as deficits in the social engagement system. This formed the rationale for the current study, which at the time of writing, is the first study to investigate the role of vagal tone in DMDD.

Numerous studies suggest a link between dampened vagal response and externalising behaviour problems in children and adolescents, such as aggression, inattention and hyperactivity. In a sample of 24 infants (12 males and 12 females, ages seven to nine months), the infant's ability to regulate the vagal brake was a significant predictor of behavioural problems at the age of three (Porges et al., 1996). Another study found that two-year olds who displayed symptoms of aggression and/or disruption showed significantly and consistently lower RSA suppression during challenging tasks than did toddlers who displayed few such symptoms (Calkins & Dedmon, 2000). In an ongoing longitudinal study of five-year-old children with different patterns of behavioural problems, children at risk of externalising problems exhibited less vagal withdrawal than the control group (Calkins et al., 2007). Pre-schoolers who were able to regulate RSA showed less emotional negativity, fewer behavioural problems and better social skills than those without stable suppression of RSA (Calkins & Keane, 2004). Attention deficit/hyperactivity disorder (ADHD), which frequently coexists with DMDD, is associated with ineffective and inflexible parasympathetic responses (RSA) during emotion regulation in children aged 7 to 9 (Musser et al., 2011). Low vagal

tone has also been correlated with aggressive behaviour in male children and adolescents (e.g., Beauchaine, 2001; Mezzacappa et al., 1997; Pine et al., 1998) and higher levels of peer conflict in children in hostile-withdrawn co-parenting environments (Leary & Katz, 2004).

In contrast, in a study of 341 5½ -year-old children, vagal regulation was positively correlated with higher peer status, facilitated by better social skills in females and better social skills and fewer behavioural difficulties in males (Graziano et al., 2007). Vagal regulation was also correlated with emotion down-regulation ability during stressful parent-child interactions (Gottman & Katz, 2008); and greater emotional expressivity in preschool children (Cole et al., 1996). A meta-analysis (44 studies, n = 4996) of cardiac vagal control and children's adaptive functioning suggests a small positive effect size between vagal withdrawal and social problems in clinical/at-risk populations consisting mostly of children with high levels of aggression and/or disruptive behavioural disorders, such as ADHD (Graziano & Derefinko, 2013).

To summarise, vagal regulation in children is associated with fewer internalising and externalising problems and better social functioning. It is therefore conceivable that children with DMDD, who exhibit externalising behaviour and compromised social interactions, could have attenuated vagal regulation. However, literature in the area of social and emotional regulation in children has not produced consistent results, with many contradictory findings (Hastings et al., 2008). For example, in pre-school and primary school-aged children, both ambient RSA and reactive RSA were positively (Dietrich et al., 2007), negatively (Beauchaine et al., 2007) or non-significantly (Calkins et al., 2007) associated with externalising problems in children. Across these studies there is no significant overall correlation between cardiac vagal control and social functioning (Graziano & Derefinko, 2013). These incongruent findings have raised many questions about the validity of the polyvagal theory (Grossman & Taylor, 2007) and its ability to translate theory into practice

(Hastings et al., 2008). However, significant heterogeneity in the measurement and quantification of RSA, particularly RSA reactivity, as a marker of emotion dysregulation and psychopathology, contributes to these inconsistent findings (Beauchaine & Bell, 2020).

Various tasks conditions have been used to examine RSA reactivity as a biomarker of emotion dysregulation, including attention allocation tasks (e.g. Suess et al., 1994), problem-solving tasks (e.g., El-Sheikh, 2005), negative emotion elicitation tasks (e.g., Crowell et al., 2005), executive function tasks (Marcovitch et al., 2010), and positive mood inductions (e.g., Fortunato, Gatzke-Kopp, & Ram, 2013). However, inferences about physiological markers of emotion regulation or dysregulation are only valid insofar as these psychological constructs are elicited (National Advisory Mental Health Council Workgroup on Tasks and Measures for Research Domain Criteria, 2016). Therefore, inferences about RSA reactivity and emotion regulation/dysregulation should only be drawn from tasks that evoke strong negative emotional responses (Beachaine & Bell, 2020).

Studies also vary in terms of RSA quantification, with some using time domain assessments (such as standard deviation of R-R intervals or mean successive difference in R-R intervals), while others use frequency domain assessments (such as Fourier transform analysis and autoregressive spectral analysis; Graziano & Derefinko, 2013). While both methods offer advantages and disadvantages, contention continues as to how much respiration confounds RSA as a measure of cardiac vagal tone, particularly across development. Children breathe at a much faster rate than adults, which means that the application of inappropriate respiration frequency bands could lead to overestimation of RSA baselines and underestimation of RSA reactivity. These differences in RSA quantification may therefore contribute to measurement error in existing data and explain inconsistent or null findings (Beachaine & Bell, 2020).

Furthermore, several measurement issues need to be considered when collecting and interpreting RSA results, including baseline conditions while assessing resting RSA (which is crucial in the interpretation of RSA reactivity data), ECG sampling rates, ECG equipment used and placement of electrodes (Beachaine & Bell, 2020; Laborde, Mosley, & Thayer, 2017). A meta-analysis of studies on RSA reactivity in psychopathology found that adherence to standards published by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996) resulted in larger effect sizes (Beauchaine et al., 2019). Non-adherence to these standards and subsequent measurement error may therefore explain at least some of the inconsistent findings in existing literature.

Several stable and transient confounding variables affect RSA and need to be recorded, potentially controlled for, and considered when interpreting RSA data (see Limitations section; Laborde et al., 2017). Variables that may contribute to the inconsistent or null findings of studies in children include weight, height and hip ratio (given the variability in growth during the development phase), cardioactive medication, sleep routine, intensive physical exercise the day before training, meals and caffeine intake within two hours of recording ECG, and needing to use the bathroom.

Finally, differences in social context might also explain these inconsistencies (Hasting et al., 2008). In order to investigate this, Hastings et al. (2008) studied preschool-aged children’s neuroception and subsequent physiological responses in familiar social contexts with ambiguous threat presentation, such as interaction with unfamiliar peers. Data collection for this study was conducted during two sessions. For the first session, each family was visited in their home, where children's baseline RSA was recorded, and the parenting behaviours of the mothers and fathers were observed during parent–child interactions and assessed via self-report. Six to ten months later, families were assessed in a laboratory.

Children were assigned to groups of three unfamiliar same-age, same-sex playmates. Each child and mother arrived separately and were escorted to a small room, where the child was fitted with the cardiac monitor. Following a baseline RSA recording, the group of three children was brought together for free-play in a playroom. The children's social group RSA were measured during the free-play session. Mothers waited in an adjacent room and completed questionnaire measures of children's self-regulation, as well as externalising and internalising problems. Children who maintained higher RSA while adapting from a calm state with their mothers to a social interaction with other unfamiliar children were less likely to experience difficulties resulting from poor emotion regulation than those who exhibited more vagal suppression. Children whose mothers exerted more negative control exhibited poorer vagal regulation during the social challenge, suggesting that social context and parenting practices may affect children's physiological mechanisms for self-regulation. Attenuated parasympathetic regulation in response to social challenge also predicted more externalising problems and aggressive behaviour in boys than it did in girls, which substantiates gender models in developmental psychopathology (Zahn-Waxler, Crick, Shirtcliff, & Woods, 2015). These findings endorse the arguments that vagal regulation is strongly associated with disruptive behaviour problems, as seen in children with DMDD. However, the most salient finding from this study was that children's adjustment was associated with dynamic vagal regulation only under conditions of ambiguous social challenge, which suggests that differences in social context (i.e., perceived ambiguity, safety or threat) may account for contradicting research findings. The current study also required children to interact in a familiar setting (i.e., school) and in an unfamiliar social context (i.e., a strange adult researcher). It was hypothesised that children with DMDD would unconsciously (via neuroception) perceive threat in the unfamiliar or ambiguous social context and shift to a physiological state of mobilisation. Future studies on children with

DMDD could perhaps explore the extent to which ambiguous or unfamiliar social situations affect vagal regulation in the absence of additional emotionally evocative tasks. From the aforementioned study, however, it is clear that multiple variables, such as parenting style and socialisation, also contribute to children's dynamic vagal regulation.

In adults, higher baseline vagal tone predicts increased social connectedness and positive emotions (Kok & Fredrickson, 2010), while RSA regulation is negatively correlated with social anxiety and defensiveness (Movius & Allen, 2005). Similarly, an association exists between cardio vagal tone and emotion regulation, social interaction and coping in young adults (Geisler, Kubiak, Siewert, & Weber, 2013). In their first study, there was a direct correlation between RSA and problem-focused coping strategies (situation and response control, positive self-directives, and seeking of social support) and facets of social well-being. In their second study, RSA predicted reduced use of emotion-focused coping strategies (acceptance and avoidance) to regulate negative affective responses and increased employment of socially adaptive emotion-regulation strategies (such as seeking social support and making concessions for anger caused by others). Female adults with borderline personality disorder - a personality disorder characterised by problems in emotion regulation, behavioural states and relationships - also have difficulties activating the vagal brake in social situations (Austin, Riniolo & Porges, 2007). In response to film clips of variable emotional subject matter, the vagal mechanisms influenced RSA only in the control group, which supports the argument that emotion and behaviour regulation in BPD are linked to problems in the dynamic regulation of the vagal brake.

The polyvagal theory (1995) has been very influential and significantly furthered our understanding of the contribution of the autonomic nervous system to adaptive and maladaptive behaviour. However, it is primarily a phylogenetic and functional account of the vagal system with limited elaboration on developmental and systemic contributions. In terms

of development, it is mostly related to the integrity of the vagal system in determining the health outcomes of high-risk infants. Furthermore, its focus is mainly on parasympathetic functioning, which implicitly minimises the role of the sympathetic nervous system and potential interactions between the sympathetic and parasympathetic nervous systems (Beauchaine, 2001).

Bearing in mind that vagal regulation is only one component in a broader model of autonomic nervous system functioning that affects the ability to self-regulate (Beauchaine, 2001; Porges, 2007), it was assumed that the polyvagal theory would prove useful in the examination of the physiological underpinnings of, and environmental influences on, DMDD. To the author's knowledge, only one study explored the association between DMDD and RSA in children, which is currently under embargo (Ametti, 2019). This study indicated a strong, positive correlation between symptoms of dysregulation and non-episodic irritability. However, low levels of sympathetic responsiveness, and not levels of parasympathetic responsiveness (which is the focus of the current study) were associated with higher levels of irritability. The current study therefore contributes to the existing body of knowledge by examining vagal regulation in children with DMDD in a familiar social context (i.e., school) with ambiguous threat presentation (i.e., interaction with a strange researcher and emotional and frustrating stimuli). It proposes that children with DMDD have faulty neuroception, which misinterprets cues in the environment as threatening, and subsequently, have failure of the newer phylogenetic vagal subsystem to promote adaptive social functioning.

Research Questions/Hypotheses

For the purpose of the study, the following questions were addressed:

1. Do children with DMDD have lower respiratory sinus arrhythmia (RSA) amplitude than healthy controls?

2. Do children with DMDD show reduced speed and accuracy during emotional, frustrating, and emotion recognition tasks compared to control subjects?
3. Do children with DMDD report more unhappiness and arousal/excitement following trials that result in punishment and frustration than controls?
4. Do children with DMDD exhibit reduced acoustic modulation compared to healthy participants?
5. Do children with DMDD differ from controls in terms of self-reported attachment style?
6. Do parents of children with DMDD report lower levels of social and communication skills than controls?
7. Do parents report improvements in domains of social and communication behaviour in children with DMDD as a consequence of medication?

The primary research hypotheses are:

1. Children with DMDD will have significantly lower RSA amplitude than controls.

2. RSA amplitude would be significantly related to speed and accuracy of attention shifting and emotion recognition tasks – lower RSA amplitude is expected to be associated with more errors and reduced reaction time during emotional, frustrating and emotion recognition tasks in children with DMDD.

3. RSA amplitude would be significantly related to attachment style – lower amplitude RSA is anticipated to be related to anxious/ambivalent or avoidant attachment styles.

4. RSA amplitude would be significantly related to prosody – reduced RSA amplitude is predicted to be correlated with reduced acoustic modulation in children with DMDD.

Chapter 5: Method

The conceptualisation of the new diagnostic category, Disruptive Mood Dysregulation Disorder (DMDD), resulted from efforts to address the increasing rate of bipolar disorder among chronically and severely irritable youth who exhibit no symptoms of mania or hypomania (Dougherty et al., 2014; Lebeinluft et al., 2003). The purpose of this study was to explore the differences in vagal regulation and behavioural outcomes in children diagnosed with DMDD compared to non-case control children. Specifically, the study was designed to answer questions on variability in respiratory sinus arrhythmia at baseline, during and after emotionally arousing tasks, as well as differences in behavioural and emotional outcomes for each of these tasks. Further research questions addressed by the study were whether children with DMDD are distinguishable from healthy participants in terms of acoustic modulation, attachment style, and social and communication skills. Finally, the research sought to establish whether parents considered medication to result in improvements in social and communication behaviour in children with DMDD. This chapter is divided into several sections addressing the research design, measures, instrumentation, method and ethical considerations.

Research Design

The nature of the research questions and study aims required a quasi-experimental, two-group cross-sectional design. It extrapolated from Austin et al. (2007) study, which examined differences in adaptive shifts in autonomic state in individuals diagnosed with borderline personality disorder and control subjects while watching movie clips of variable emotional content. The study found that those with borderline personality disorder ended in a physical state that supported defensive behaviours, while those in the control group ended in a physical state that supported social engagement behaviours. Based on their conclusion that the findings are consistent with other research (Sahar, Shalev, & Porges, 2001; Umhau et al.,

2002) that demonstrates atypical vagal regulation of the heart in psychiatric disorders, and because borderline personality disorder shares the core features of DMDD (i.e., emotional and behavioural dysregulation), it was hypothesised in the current study that children with DMDD will exhibit a similar response as individuals with borderline personality disorder.

The current research design was also guided by studies on the effect of reward, punishment, and frustration on attention in paediatric bipolar disorder (Rich et al., 2005) and the neural mechanisms of frustration in children with severe mood dysregulation, which used attention orienting and shifting computer programmes to measure between-group differences in response to affective feedback and frustration (Rich et al., 2010b).

The nature of the study, i.e. the preclusion of randomisation due to DMDD diagnosis, limited the design to that of non-equivalent groups quasi-experimental research. This addressed ethical concerns regarding the identification of children who meet the criteria of DMDD, but who have not yet been diagnosed or treated for their symptoms. A quasi-experimental design also allows for practical studies in real-world settings, which minimises threats to ecological validity (White & Sabarwal, 2014). In addition, this alternative design catered for the anticipated small sample size, in which an even distribution of known and unknown confounding variables between the intervention and control groups is not adequately achievable (Harris et al., 2006). This non-equivalent two groups comparison design enabled the researcher to address the following research aims:

1. To determine whether there were any significant between-group differences in terms of respiratory sinus arrhythmia (RSA), or vagal tone, and heart period at baseline and in response to three different measures:
    a. The Affective Posner Cueing Task (Perez-Edgar & Fox, 2005)
    b. The Dynamic Affect Recognition Evaluation (DARE; Porges, Cohn, Bal, & Lamb, 2007)

    c. Prosody or acoustic modulation

2. To ascertain whether any significant between-group differences existed in behavioural (i.e., response time/latency and accuracy) and/or self-reported and observed affective (valence and arousal) data during the Affective Posner Cueing Task.
3. To determine whether there were significant between-group differences in the accuracy and latency in identifying emotions during the DARE task.
4. To establish whether there was significant variation in prosody between the two groups.
5. To determine any significant differences in self-reported attachment style between the groups and whether there was a significant correlation between attachment style and RSA.
6. To determine whether there was a significant correlation between RSA and/or heart period and behavioural outcomes of the Posner and DARE tasks.
7. For only the DMDD group, to discover whether a relationship existed between the number of parent-reported social behaviour and communication skills and RSA.

A quasi-experimental design does, however, have a number of limitations, one of which is a threat to internal validity due to the absence of randomisation and the lack of control over confounding variables. Conclusions about causality are therefore not possible, in contrast to those of true experimental designs (Campbell & Stanley, 2015; Harris et al., 2006). These limitations were controlled for by matching control participants as closely to DMDD participants as possible and by taking confounding variables into account during the statistical analyses.

Sample and Sampling Procedure

Purposive sampling was used to obtain the participants who met the criteria for DMDD and the typically developing children who served as matched controls. Due to the recent inclusion of this category in the *DSM-5*, and the niche population group it represents, a small sample size was anticipated. The final sample consisted of 30 children and adolescents between the ages of 7 and 13.

Participants were recruited through child and adolescent psychiatrists and psychologists in private practice, as well as at a government-based out-patient clinic at a psychiatric hospital in Gauteng. Information letters (Appendices A - D) containing details about the study, contact details of the researcher and letters of consent and assent were sent to doctors and psychologists who expressed willingness to assist with the identification of potential participants. Professionals were asked to briefly discuss the study and significance of thereof with the parents or guardians. Interested parents or guardians were asked for permission for the researcher to contact them telephonically. As the professionals had made the diagnosis of DMDD, there was no need for additional screening.

In total, 21 children who met the criteria for DMDD were identified and contacted. Of these, four declined participation and two were excluded due to epilepsy and developmental delay (see Inclusion/Exclusion Criteria). All but two (n = 13) of the remaining 15 had also been diagnosed with ADHD. Comorbid disorders reported by parents included reactive attachment disorder (n = 3), bipolar disorder (n = 3), anxiety (n =1), conduct disorder (n =1) and unspecified mood disorder (n =1).

Control subjects were age-, gender- and ethnicity-matched to the DMDD group and were recruited from public and private schools in the Johannesburg metropolitan area. Permission to recruit from these schools was obtained from the Gauteng Department of Education (see Appendix E) and, in the case of private schools, the principals and governing

bodies of the schools. Interested parties received an information letter and letters of consent similar to that of the experimental group.

Table 3 describes the demographic characteristics of the of DMDD and control groups on the demographic variables of gender, race, age, home language, medical aid, highest level of education of both parents, presence of ADHD and medication. As testing was conducted in English, home language data were grouped into English and non-English categories and years of exposure to English were compared in the statistical analyses. No significant difference in years of expose to English was found between the control (M = 8.9, SD = 2.2) and DMDD (M = 9.7, SD = 1.7) groups.

Table 3

Demographic Variables, by Group

Variable	Category	Group			
		Control		DMDD	
		n	%	*n*	%
		15		15	
Gender	Male	14	93	14	93
	Female	1	7	1	7
Race	Caucasian	7	47	7	47
	African	5	33	5	33
	Mixed	2	13	2	13
	Indian	1	7	1	7
Age	7	1	7	1	7
	8	2	13	2	13
	9	3	20	2	13
	10	3	20	4	27
	11	2	13	2	13
	12	2	13	3	20
	13	2	13	1	7
Home Language	English	11	73	10	67
	Other	4	27	5	33
Medication	Yes	3	20	15	100
	No	12	80	-	-

(continued)

(continued)

Variable	Category	Group			
		Control		DMDD	
		n	%	*n*	%
Medical Aid	Yes	11	73	8	53
	No	4	27	7	47
Parent 1 Highest Level of Education	Further	5	33	11	730
	Higher	9	60	2	130
	Other	1	7	2	13
Parent 2 Highest Level of Education	Further	4	27	9	60
	Higher	9	60	-	-
	Other	1	7	2	130
	Missing	1	7	4	270

Note: Further Education = grades 10, 11 and 12; National Qualifications Framework (NQF) levels 2 (Certificate), 3 (Certificate) and 4 (Diploma). Higher Education = NQF levels 5 (Certificate, Higher certificate and First diploma), 6 (Bachelor's degree, Professional first degree postgraduate, and General first degree), 7 (Postgraduate diploma, Honours degree, and Master's degree), and 8 (PhD). Source: The Department of Education in South Africa

Inclusion/Exclusion Criteria. English language proficiency was a requirement for all participants given that the procedure was carried out in English. As a proficiency measure, participants were required to be educated in English, have English Language as a subject in school, or have been exposed to the English language from Grade 1.

In addition, exclusion criteria for both groups included any form of compromised neurology (such as epilepsy or traumatic brain injury) or serious or debilitating illness (for example, meningitis) in the last three months that could potentially have confounding effects on test performance.

Parents of control subjects reported that their children did not meet the criteria of DMDD and did not have any first-degree relatives who had previously been diagnosed with psychiatric illnesses, including depression and anxiety.

Measures

A combination of nine measures were employed to obtain the data necessary to investigate the hypotheses of the study (see Table 4). It consisted of a demographic questionnaire, the Affective Posner Task (Perez-Edgar & Fox, 2005), the Self-Assessment Manikin (Bradley & Lang, 1994; Lang, 1980), the Observed Frustration Reactivity scale (Degnan, Calkins, Keane, & Hill-Soderlund, 2008), Respiratory Sinus Arrythmia, the Dynamic Affect Recognition Evaluation (DARE; Porges et al., 2007), prosodic features of vocalisations (Porges & Lewis, 2010), the Attachment Style Classification Questionnaire (Finzi, Har-Even, Weizman, Tyano, & Shnit, 1996; Finzi, Cohen, Sapir, & Weizman, 2000) and the Social Behaviour and Communication: Parent Questionnaire (adapted, with permission, from the Listening Project Parent Questionnaire; Bazhenova & Porges, 2000).

Table 4

Complete Assessment Battery

Construct	Assessment
Attention-emotion interaction	Affective Posner Task
Emotional arousal and valence	Self-Assessment Manikin
Frustration reactivity	Observed Frustration Reactivity
	Self-Assessment Manikin
Vagal tone	Respiratory Sinus Arrhythmia
Emotional reactivity	Respiratory Sinus Arrhythmia
Emotional regulation	Respiratory Sinus Arrhythmia
	The Social Behaviour and Communication: Parent Questionnaire
Facial emotion recognition	Dynamic Affect Recognition Evaluation
Prosodic features of vocalisation	Voice Recordings
Attachment style	The Attachment Style Classification Questionnaire
Gestural facial expression	The Social Behaviour and Communication: Parent Questionnaire
Language and social interaction	The Social Behaviour and Communication: Parent Questionnaire

(continued)

(continued)

Construct	Assessment
Listening and response to sound	The Social Behaviour and Communication: Parent Questionnaire
Emotional reciprocity	The Social Behaviour and Communication: Parent Questionnaire
Behaviour	The Social Behaviour and Communication: Parent Questionnaire

Demographic Questionnaire

A basic questionnaire to record demographic information, including age, gender, population group, home language, education, highest level of parents' education, parents' occupation, medical aid membership, English proficiency, learning disorders diagnosed and medication use in the last three months, was necessary to match DMDD participants to control subjects. It was also used for descriptive statistics in order to provide a clear picture of the sample composition (Appendix H).

Affective Posner Task

The Posner Cueing Test, also known as the Posner Paradigm, was designed and is widely used to investigate spatial attention and orienting to cueing (e.g., Hugdahl & Nordby, 1994; Posner, 1988; Posner & Cohen, 1984; Rich et al., 2005; Rich et al., 2007; Rich et al., 2010b). Perez-Edgar and Fox (2005) modified the original task to include positive and negative feedback as a means of examining attention-emotion interaction, known as the Affective Posner Task. Seven-year-olds performed this task and, compared to the original (affect-neutral) Posner Task, their performance in the affective version was characterised by significantly faster response times, increased errors and an increased validity effect (the difference in response times in response to the different cue types). A rigged version of the Perez-Edgar and Fox (2005) test has been used to elicit frustration in order to study the neural structures involved in frustration in chronically irritable children (Deveney et al., 2013) and

the impact of reward, punishment, and frustration on attention in paediatric bipolar disorder (Rich et al., 2005).

For the current study, the original version of the Affective Posner Task was obtained from Professor Perez-Edgar (2015, personal communication), and implemented in OpenSesame 2.9.7. This version was adapted to elicit frustration by telling participants that they were responding too slowly to collect the game reward, irrespective of their performance. OpenSeasame, project-managed and lead-developed by Sebastiaan Mathôt (Mathôt, Schreij, & Theeuwes, 2012) at the University of AIX-Marseille, is an open source graphical builder for creating psychological experiments, that enables the extension of such experiments using Python programming language. It was chosen as the experimental platform as it performed comparably well to the commercially available E-Prime in a comparison of various input devices (Freegard, 2012). In this scenario, OpenSesame's Python extension method was used to connect to a custom-built StimSync-based response box in order to record timestamps of responses read.

The entire task was made up of three games comprising various trials. Trials consisted of a white fixation cross, an icon indicating trial type (reward or no reward), three white squares, a blue block in one of the white squares (in the middle square for 20% of trials and equally distributed in the remaining trials) and a white target in either the left or right white square. Trials were either followed by accuracy feedback (i.e., "right" or "wrong") or accuracy and reward feedback (i.e. accuracy feedback and happy or sad emoticon). The objective was to push the button (blue for left and yellow for right) on the response box that corresponds to the location of the white target. The blue cue predicted the target location on 40% of trials (valid trials) and was in the opposite location on 40% of trials (invalid trials). The remaining 20% were in the middle box (neutral cue; see Figure 6).

Game 1 served as the baseline and provided accurate performance feedback (see Figure 7) and no reward or punishment (see Figure 8).

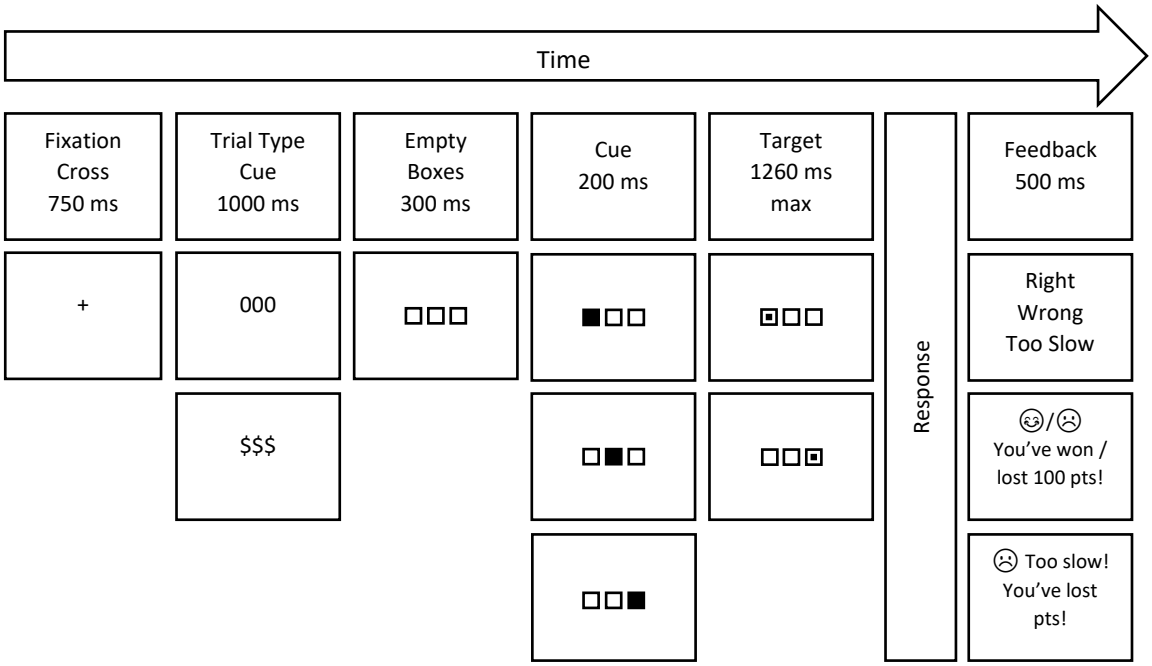

Figure 6. Schematic of the Affective Posner Paradigm computer stimuli, with examples of stimuli and associated timing of presentation

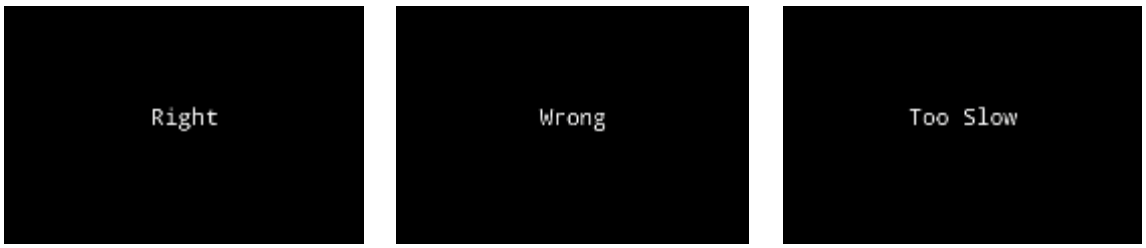

Figure 7. Accurate and neutral feedback during Game 1 of the Affective Posner Paradigm

Game 2 also provided accurate feedback and either awarded or subtracted game rewards (in the form of points), depending on performance, coupled with the relevant emoticon (see Figure 9 and Figure 10).

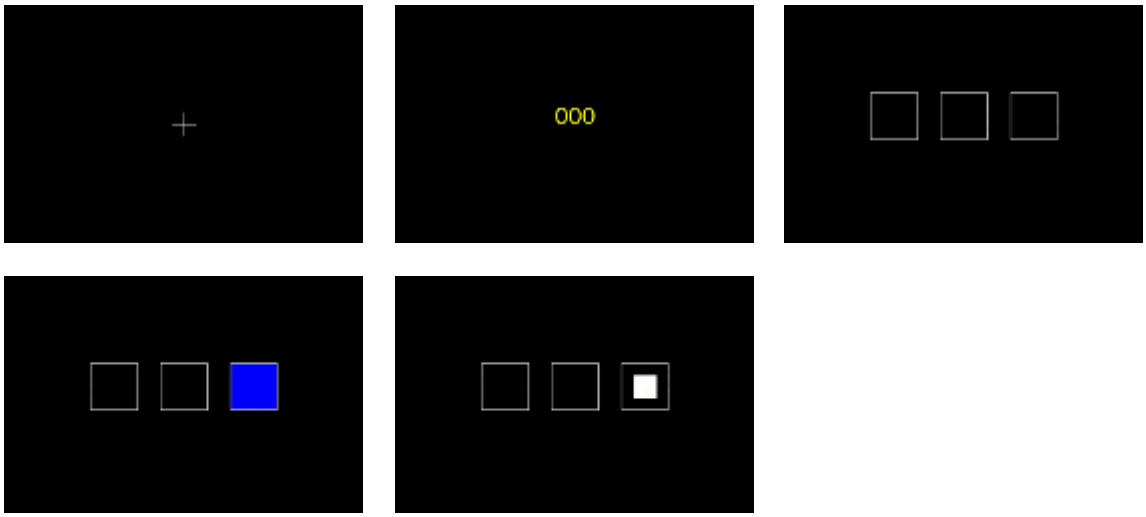

Figure 8. Screenshots of Game 1 sequence, with fixation cross, no-reward trial indicator, empty squares, cue block and target block.

Figure 9. Accurate and affective feedback during Game 2 of the Affective Posner Paradigm

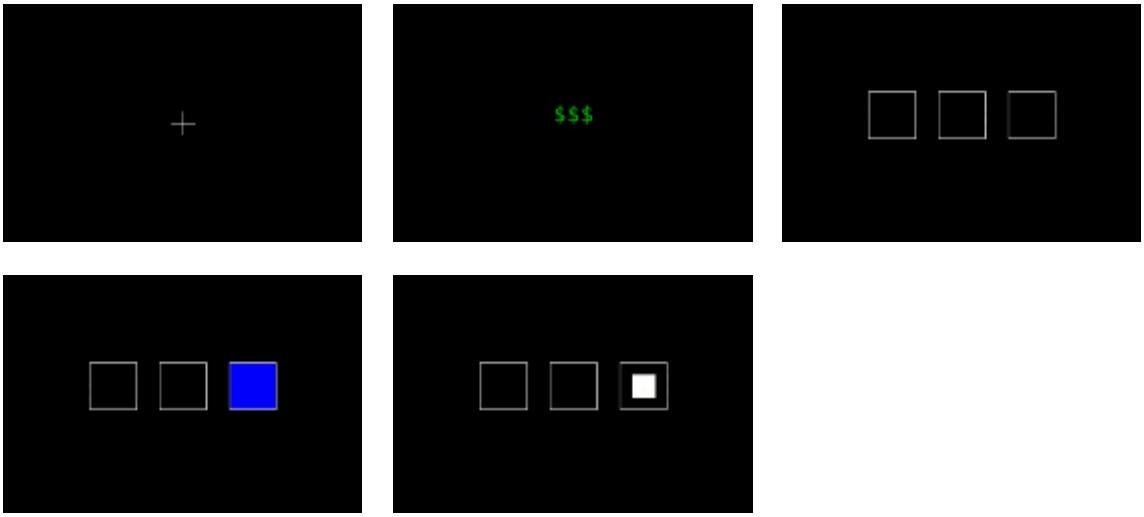

Figure 10. Screenshots of Game 2 sequence, with fixation cross, reward trial indicator, empty squares, cue block and target block.

The third game was the frustration condition, in which inaccurate, negative feedback (i.e., “too slow”) was randomly given on 60% of accurate trials and game rewards, regardless of participants’ reaction time. Frustration manipulation only occurred in the third game in an attempt to gradually increase arousal and prevent possible carry-over effect.

Behavioural data recorded included response accuracy (i.e. the ratio of responses that corresponded with the target location), calculated as a percentage score, and response time in milliseconds. For response accuracy, failure to respond was considered incorrect. Response time was calculated using only actual responses, therefore failure to respond was not considered.

In addition to the valid, invalid and neutral categorisation, trials were classified as post-positive and post-negative feedback to assess the impact of feedback on subsequent performance, and frustration and non-frustration to measure the effect of frustration on RSA.

Self-Assessment Manikin

The Self-Assessment Manikin (SAM; © Peter J. Lang, 1994), is a pen-and-paper pictorial, Likert-type scale that measures a person’s emotional experiences in response to stimuli on the dimensions of valence, arousal, and dominance (Bradley & Lang, 1994; Lang, 1980). Permission to use the SAM was obtained from The Centre for the Study of Emotion and Attention (CSEA) Media Core at the University of Florida. Bradley and Lang (1994) compared ratings of affective experience of university students obtained using the SAM with those of the Semantic Differential Scale and found that correlations were high both for reports of experienced pleasure and felt arousal. In the dominance dimension, the results suggested that SAM provides a better indication of the subjective experience to an affective stimulus. The SAM has been used in numerous studies to measure subjective experience in reaction to various kinds of stimuli, including images, sounds, advertisements and pain and

also in a variety of populations (e.g., children, anxiety patients, psychopaths and other clinical populations; Bradley & Lang, 1994; Gunn & Finn, 2015; Jacob et al., 2015; Reitz et al., 2015; Scott, Green, & Fairley, 2016; Van Leeuwen, Vink, Joëls, Kahn, & Vinkers, 2016; Zucker et al., 2017).

For the current study, SAM was the preferred instrument as its non-verbal design makes it an ideal tool in a cross-cultural environment, such as South Africa, with subjects of varying ages and levels of education. Only two dimensions were measured to assess subjective experience of affect and arousal (dependent variables) in response to reward, punishment and frustration (independent variables). It consisted of unlabelled drawings of human manikins on a 9-point scale, with extremes of happy/unhappy (i.e., valence; see Figure 11) and calm/aroused (i.e., arousal; see Figure 12).

Figure 11. 9-point valence scale of the Self-Assessment Manikin. Copyright 1994 by James Lang. Reprinted with permission from CSEA Media Core.

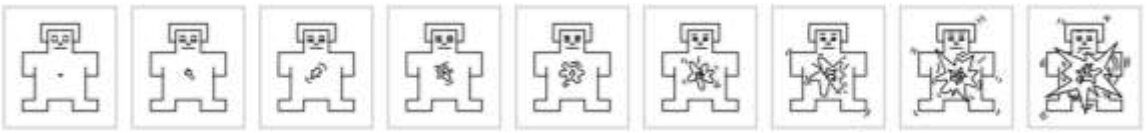

Figure 12. 9-point arousal scale of the Self-Assessment Manikin. Copyright 1994 by James Lang. Reprinted with permission from CSEA Media Core.

The SAM was integrated into the experiment software to minimise disruption and make it easier for the participants to respond by using the mouse to click on the relevant picture. Self-report affective data was collected using the SAM before the practice round and after each condition (i.e. baseline, reward/punishment and frustration).

Observed Frustration Reactivity

As children are not always able to accurately assess their own level of frustration or may want to present a more socially desirable image, the researcher conducted a qualitative observation of behaviours that may be indicative of frustration.

Reactivity was indexed using the method employed by Degnan et al. (2008; Appendix J). It includes measures of distress, including whining, pouting, fussing, crying, screaming, or throwing tantrums. This behaviour was coded according to a) global negative reactivity: rated for the entire frustration condition on a scale from 0 (no negative response) to 4 (game ended with the child in extreme distress); and b) global affect: rated for the entire frustration condition on a scale from −3 (highly negative affect) to 3 (highly positive affect).

Respiratory Sinus Arrhythmia (RSA) and Heart Period (HP)

The amplitude of respiratory sinus arrhythmia (RSA) – the phenomenon where heart rate increases during inhalation and decreases during exhalation - is considered a selective, valid, non-invasive and easily obtainable index of myelinated vagal influence on the sinoatrial node (Porges, 1995). It is well established in the literature, and is increasingly being used in psychophysiological studies, as an index of the parasympathetic nervous system, or cardiac vagal tone (Bernston et al., 1993; Chapleau & Sabharwal, 2011; Laborde et al., 2017; Malik et al., 1996; McCraty & Shaffer, 2015). Various studies have linked vagal tone (i.e. RSA amplitude) to emotion reactivity and regulation in children and adults (e.g. Appelhans & Luecken, 2006; Austin et al., 2007; Beauchaine et al., 2007; Calkins et al., 2007; Cole et al., 1996; El-Sheikh , 2001; Geisler et al., 2013; Gentzler, Santucci, Kovacs, & Fox, 2009; Gottman & Katz, 2002; Graziano & Derefinko, 2013; Graziano et al., 2007; Hastings et al., 2008; Jönsson & Sonnby-Borgström, 2003; Licht et al., 2009; Movius & Allen, 2005; Rottenberg, Salomon, Gross, Gotlib, 2005; Santucci et al., 2008; Watkins, Grossman, Krishnan, & Sherwood, 1998; Willemen, Goossens, Koot, & Schuengel, 2008).

Measures of heart period (HP; i.e., the inverse of heart rate) were included for comparison reasons. Heart rate is regulated by a number of influences, including sympathetic, parasympathetic, neuroendocrine and intrinsic factors (Doussard-Roosevelt, Montgomery, & Porges, 2003). An evaluation of the variables separately reflects overall autonomic functioning (HP) and specific myelinated vagal activity (RSA; i.e., parasympathetic contributions to the regulation of heart rate). Changes in HP that are not accompanied by changes in RSA reflect non-vagal regulation of heart rate. Examining the relationship between HP and RSA allows the detection of the portion of the overall heart rate that is due to myelinated vagal influences (a high correlation is an indication of adaptive vagal functioning, i.e., "calmer" or "healthier" individuals). As the objective of the current study was to assess myelinated vagal functioning in children with DMDD, it was necessary to include heart period as an outcome measure.

To calculate RSA, electrocardiograms (ECGs) were recorded at the start of each session (to obtain a stimulus-free, movement-free baseline, which is necessary to measure reactivity), as well as during and after the Affective Posner Task, the Dynamic Affect Recognition Evaluation, and the voice recordings. Inter-beat-intervals (IBI) were extracted from the ECG, which were then visually inspected for outlier points and manually edited if necessary. RSA amplitude was subsequently calculated using specialised computer software that uses the Porges (1985) method (see Instrumentation section for details).

The Dynamic Affect Recognition Evaluation

The Dynamic Affect Recognition Evaluation (DARE; Porges et al., 2007) was developed as a naturalistic tool for assessing facial emotion recognition. The DARE provides a standardised presentation of emotional expressions, and the stimuli were developed from the Cohn–Kanade Action Unit-Coded Facial Expression Database (Cohn, Zlochower, Lien, & Kanade, 1999). The database includes approximately 2000 image sequences from more

than 200 human subjects. The stimuli included uncompressed video files (i.e., series of still images) representing six basic emotions (anger, disgust, fear, happiness, sadness, and surprise). The images were morphed and the final videos used in this study included a face starting with a neutral expression and slowly transitioning into one of the six target emotions. Video length varied (ranging from 15–33 seconds), depending on the number of the frames in the original image sequence used to generate the video. Duration of video was independent of emotion category. The participant code, the emotion displayed, the emotion chosen by the participant, and the response latency (in seconds) were recorded for each video.

The DARE has identified differences in affect recognition latency and error in autistic children (Bal et al, 2010) and women with HIV (Heilman, Harden, Weber, Cohen, & Porges, 2013). It was included in the assessment battery to determine whether increased emotion recognition error and latency are features of DMDD, and if so, whether those differences correlate with vagal tone.

Prosodic Features of Vocalisations

Prosody is the conveyance of emotion in human voice through the modulation of intonation (i.e., pitch). In previous studies by the Porges laboratory (Kolacz, Lewis, & Porges, 2018; Porges et al., 2013; Porges & Lewis, 2010; Porter, Porges, & Marshall, 1988; Stewart et al., 2013), several prosodic variables have been related to autonomic state. These studies suggest that a faster heart rate (i.e., an autonomic state of "mobilisation") is associated with the dampening of the modulation of the acoustic features that are perceived as prosody (Stewart et al., 2013). Four variables in particular have been studied in this regard, namely modulation depth, bandwidth of fundamental frequency, shape separability 90, and spectral tilt.

Modulation depth is a measure of vocal instability, which is thought to reflect a deficit of vagal tone to smoothly control the muscles of the larynx and pharynx. Bandwidth of

fundamental frequency, or BW50, is an indicator of increased variability in pitch and a sign of prosody in voice. Higher bandwidth denotes greater prosody. Shape separability is derived from the joint amplitude and pitch modulation in the voice. Lower shape separability signifies voice quality that is more prosodic. Spectral tilt is an indicator of the energy distribution, or balance, between the low and the high frequencies of the voice (Kakouros & Räsänen, 2016; Kluender, Coady, & Kiefte, 2003).

Audio files of at least 20 seconds each were recorded at 44.1 kHz, using a Blue Snowball USB microphone (see Instrumentation), and saved in a .m4a file format.

Attachment Style

The Attachment Style Classification Questionnaire (ASCQ; Finzi et al., 1996; Finzi, et al., 2000) is an adapted version for children of the Hebrew version (Mikulincer & Segal, 1990) of Hazan and Shaver's (1987) questionnaire for the classification of attachment styles in adults. The questionnaire consists of 15 items, divided into three factors, which tapped Ainsworth's (1978) three attachment patterns, namely secure (e.g. "I usually believe that others who are close to me will not leave me"), anxious/ambivalent (e.g. "I'm sometimes afraid that no one really loves me"), and avoidant (e.g. "I find it uncomfortable and get annoyed when someone tries to get too close to me"). Results indicated a mean level of reliability (α = .69 – .81; Finzi et al., 2000). Test-retest stability after two weeks was in the high range (r = .87 – .95). Concurrent validity was assessed by means of Pearson's correlation with the children's scores in the Trait Anxiety Inventory, Child Depression Inventory, and Child Suicidal Potential Scales for Aggression. Distribution of the styles among the latency age children was similar to that found in previous studies of adults, infants and toddlers (Finzi et al., 2000). The ASCQ (Finzi et al., 1996; Finzi et al., 2000) was included in the assessment battery as it was a short and easy-to-understand questionnaire for latency-aged children to complete on their own. The instructions, which were read to

participants to make sure they understood, asked the participants to rate how true each of sentences was for them. The Likert-type scale was scored from 1 to 5, with 1 being “All wrong” and 5 being “Very right” (see Appendix K). The ten items tapping the avoidant and anxious styles were used to calculate two attachment scores by averaging the relevant items.

Social Behaviour and Communication

The Social Behaviour and Communication: Parent Questionnaire is an informal, structured questionnaire (i.e., psychometric properties were not established) that was adapted from pre- and post-intervention application to single application, with permission, from the Listening Project Parent Questionnaire (Bazhenova & Porges, 2000; Appendix L). The adapted questionnaire was approved by Prof. Porges and aimed at obtaining parents’ perceptions of their child’s developmental and behavioural problems in specific domains. These include hearing sensitivity, spontaneous speech, receptive speech, spontaneity, behavioural organisation, emotional control, affection, listening, eye contact, and relatedness (see Table 5). The questionnaire focussed on whether the child had/has had any difficulties in a specific behavioural area and whether there have been any changes since starting medication. For each behavioural category, parents were asked to document any changes and provide examples of new behaviours.

Developmental and behavioural problems in the five domains (i.e., Gestural and Facial Expression, Language and Social Interaction, Listening and Response to Sound, Emotional Regulation and Reciprocity, and Behaviour) were scored on a scale from -1 to 1, with 1 denoting “Problem indicated, improvement”, 0 meaning “Problem indicated, no/vague improvement” and 1 standing for “Problem indicated, deterioration”. The total number of problems in each domain was also calculated.

Table 5

Behavioural Domains and Explanations for the Structured Parent Questionnaire

	Definitions
Hearing sensitivity	Exaggerated negative responses (e.g., crying or placing hands over the ears) to common noises (e.g., vacuum cleaner, garbage disposal, baby crying, and air conditioning)
Spontaneous speech	Non-prompted use of words and sentences to communicate thoughts and ideas
Receptive speech	Ability to understand instructions and phrases
Spontaneity	Non-prompted behaviours initiated by the child
Behavioural organisation	Ability to occupy oneself (when left alone) in a productive and non-stereotypical way
Emotional control	Ability to calm quickly when upset, to respond to unexpected changes without getting upset, and to tolerate objections and contradictions of other people
Affection	Behaviours reflective of warm emotional state expressed by the child toward familiar people (e.g., hugging, kissing, and saying "I love you" to the parent)
Listening	Ability to focus on human speech without visual or contextual cues, to understand spoken words, and to follow verbal requests
Eye contact	Making and maintaining eye contact during social interactions
Relatedness	Non-prompted social behaviours that reflect understanding of a joint partnership in interactions and sharing the same goals during social interactions (e.g., looking at a partner, showing toys, sharing an idea or a thought, and directing emotions to the partner)

Note. Reprinted from Reducing auditory hypersensitivities in autistic spectrum disorder: preliminary findings evaluating the listening project protocol. Frontiers in Pediatrics, 2, 80., by Stephen Porges et al., 2014. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4117928/.

Instrumentation

The experimental setup consisted of a laptop, a custom-built response box and a heart rate monitor (see Figure 13).

Response Box

The response box is based on the StimSync Open Source hardware and software specification published by Dr Chris Rorden's Neuropsychology Lab (Rorden & Hanayik,

2014). Rorden is the director of the McCausland Center for Brain Imaging and a professor of Neuroimaging at the University of South Carolina.

For the current study, the response box used the StimSync basic configuration leveraging and Arduino Leonardo board with two switches connected to digital inputs pins (see Figure 14).

The StimSync, when emulating a USB keyboard, provides results that compare favourably to some commercial devices (2ms latency with a 0.17ms *SD*). In this instance, however, the microsecond timing mode was utilised, but timing was recorded in the experimental software and not using the hardware device clock, as this was more accurate.

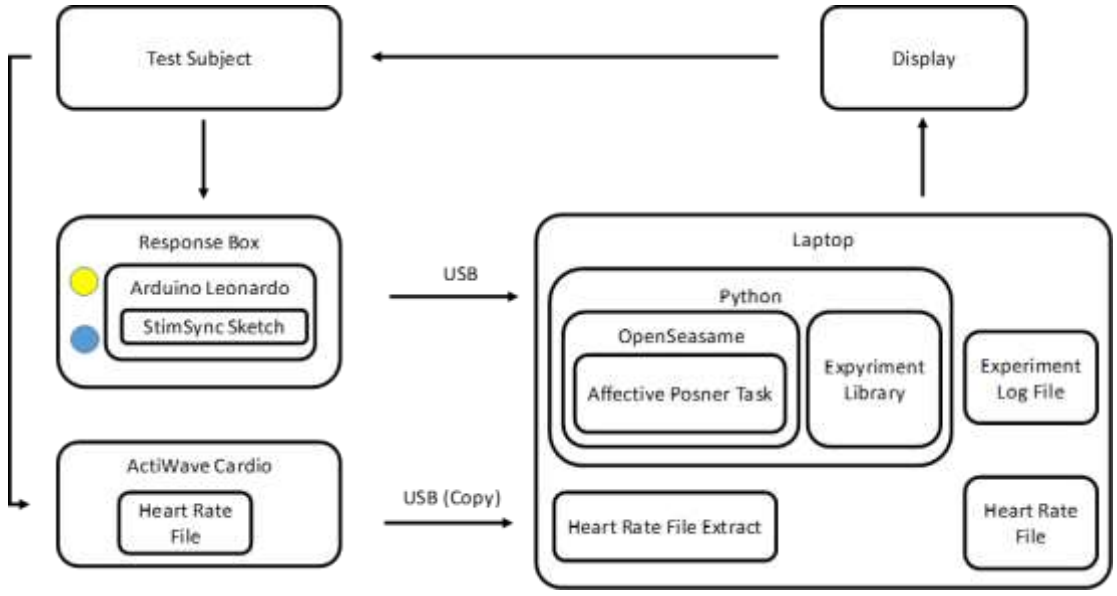

Figure 13. Schematic Representation of the Experimental Setup for the Affective Posner Cueing Task

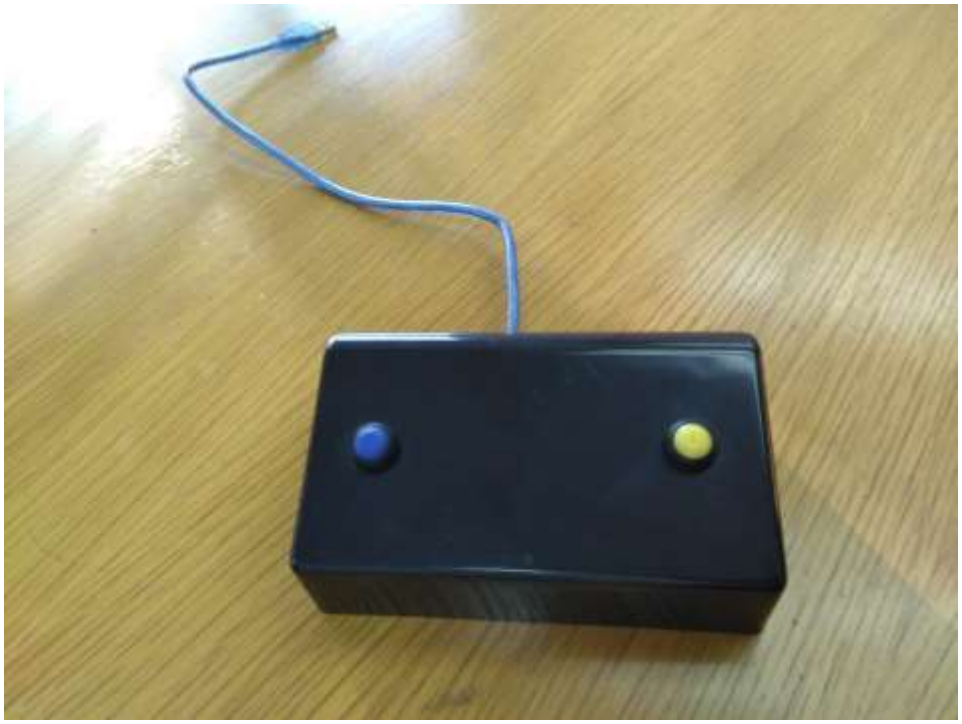

Figure 14. The Response Box used during the Experiment

Source code for the Arduino sketch was provided in the StimSync package and was compiled for the Arduino Leonardo platform. Additional advice in setting up the instrumentation was obtained from Gary Freegard at the University of Swansea, who has expertise in the use of Arduino-based response boxes and has written a paper comparing these to commercial response boxes (Freegard, 2012).

Laptop

The experiment was run on a laptop computer with Windows Vista, 1GB RAM and an Intel T2300@1.66GHz CPU.

Experimental Platform

The experimental platform used for implementation of the trials was OpenSeasame 2.9.7. OpenSeasame is a graphical builder for creating psychological experiments that enables the extension of such experiments using Python code. The version of Python used is 2.7.8. OpenSeasame is built on top of a basic backend library that enables basic input output

functions. For this task, Expyriment library was used for the backend, as it was designed for cognitive and neuroscientific experiments and enables high precision timing (Krause & Lindemann, 2014).

Actiwave Cardio

Heart rate and inter-beat variability measurements were recorded with a small, wireless single channel ECG waveform recorder called the Actiwave Cardio by CamNtech Ltd. The device has been tested and validated against various international standards and directives for risk management, safety and performance. A list of these can be found at: https://www.camntech.com/about-us/certifications (retrieved 20 May 2019). The device has also been approved for sale in South Africa and was obtained through the local distributor, LifeMax. Full training in the setup and use of the device was provided by LifeMax. The Actiwave has also been used in various research studies (Chou, Marca, Steptoe, & Brewin, 2014; Fenton-O'Creevy et al., 2012; Kusserow, Amft, Gubelmann, & Tröster, 2010; Kusserow et al., 2012; Mazilu et al., 2013) and has been shown to have an ECG recording similar to that of traditional Holter monitoring (Abell, 2012).

Blue Snowball Microphone

Audio files were recorded using a Blue Snowball omnidirectional/cardioid USB microphone. This microphone was one of the few microphones recommended by the Prosody Laboratory of the University of North Carolina (personal communication, 2017). It was included in the study design because it was cost-effective but still capable of producing clear and crisp recordings that were free of feedback and distortion. The microphone also had a plug-and-play design that was Windows Vista compatible and therefore required no additional drivers to record. The vocal recording setting of the microphone was used in this study.

Administration Procedure

Interested parents or guardians contacted, or were contacted by, the researcher and details of the study and procedure were explained (without disclosing details that would compromise the validity and reliability of the study). Information sheets and consent/assent forms, containing a statement that participants may receive misleading information (this was necessary to induce frustration during the third game of the Affective Posner Cueing task) during the assessment process, were emailed to those participants who requested more information. DMDD participants who agreed to participate were asked to delay medication until after the testing session (early morning sessions were arranged so that the delay was not too protracted) and all participants were requested not to apply cream or lotion to the chest area prior to the session for optimal adhesion of the electrodes. Participants were also asked not to consume any stimulants, such as caffeine or sugar, within two hours of being tested. As heart rate varies during the day, sessions were arranged for mornings, within a certain time range (between 08:00 am and 10:00 am), when it was convenient for participants and their parents/guardians, as well as for the participating schools.

The research was conducted during two sessions on separate days. The first session was approximately 45 minutes. Upon arrival, participants were reminded that participation is voluntary and that they could choose to withdraw at any time during the process. In order to allow the conductive gel on the electrodes to warm up before testing, adolescents were then given a drawing depicting where the electrodes and wireless unit should be placed, and then asked to place these on himself/herself in private (either in the restroom or the researcher left the room) and pull their shirt down once the unit was on. For younger children, the parent or guardian was asked to place the unit on the child in private. Once fitted with the unit, participants received a unique identification number to ensure

anonymity. Consent and assent forms, as well as the demographic questionnaire, were completed by the parent/guardian and/or collected.

Participants were subsequently seated in front of the laptop. If the child requested, the parent or guardian was allowed to sit in the room for the entire session. Otherwise, parents waited just outside the room, with the door remaining open. During a short information session, the procedure and object of the "game" were explained to participants. They were asked to sit as still as possible during the two-minute baseline ECG recordings (i.e., while a large hour glass appeared on the screen) and were guided through the SAM scales in detail. Participants were asked to, as honestly and accurately as possible, indicate how each game made them feel and were assured that there was no "right" or "wrong" answer.

The first session commenced with a 30-second practice session to make sure that participants understood what was expected of them (see Figure 15).

Figure 15. A screenshot of the Practice Round Instructions of the Affective Posner Task.

They were then asked to complete the first game, which was a longer version (2.7 minutes) of the practice session (see Figure 16).

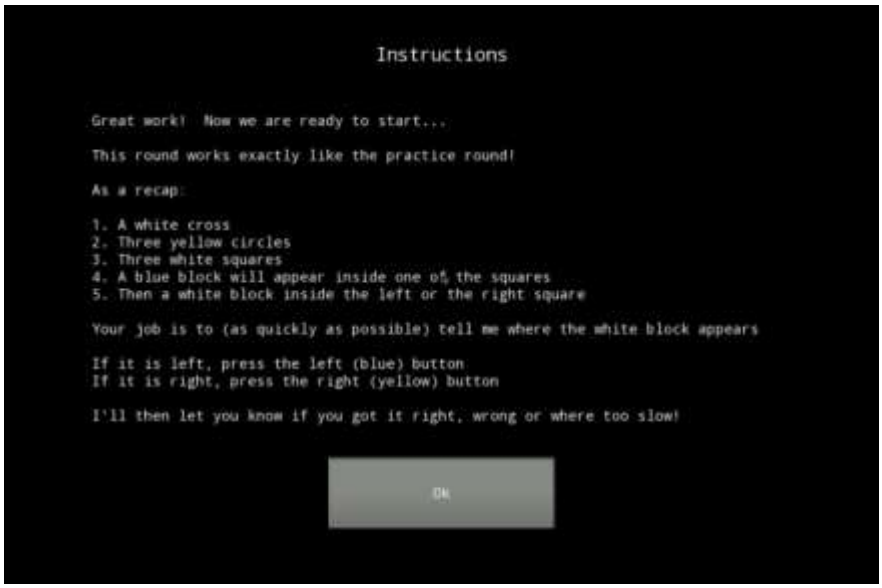

Figure 16. A Screenshot of the Instructions before Game 1 of the Affective Posner Task.

Before the second game (5.4 minutes), the concepts of non-money and money trials and reward and punishment were explained (see Figure 17).

Figure 17. A screenshot of the instructions prior to Game 2 of the Affective Posner Task to explain the difference between reward and no-reward trials.

Prior to the third game (5.4 minutes), participants were told that they had to perform accurately and quickly to gain rewards and that acceptable speed range will be calculated using a complex formula which factors in performance in previous trials (see Figure 18).

Figure 18. A screenshot of the instructions for Game 3 of the Affective Posner Task to inform children that their performance will be based on an average of their response times.

After the final SAM and baseline ECG recording, participants were debriefed (which included a full explanation of the study objectives) and thanked for their participation. A clinical psychologist was present and available for counselling, although this was not necessary for any of the participants.

The follow-up session was slightly shorter, varying between 20 and 30 minutes, depending on the individual's rate of response. During the second session, the DARE was explained to the participants. First, they were shown videos of the six target emotions. Thereafter, they were told that they would be shown the same photos and had to detect, via

the laptop keyboard, the emotion of the particular face presented as a practice round. Upon completion, they were informed that the experiment would commence as soon as possible.

Respiratory Sinus Arrhythmia (RSA)

RSA was quantified by recording each participant's electrocardiography (ECG), using two standard disposable electrodes and a small, wireless single channel ECG waveform recorder (see Instrumentation). The monitoring system sampled ECG at 1024 Hz with minimal artefact. During the first session, heart rate was recorded for two minutes prior to the start (as a baseline), and after each of the three games, of the Affective Posner Task. A similar protocol was followed in the second session for the DARE and prosody tasks.

Prosody

Participants were asked to talk about a positive experience for 20 seconds. They were given examples such as, "tell me something good that happened in school" or "tell me about your best friend" or "tell me about your last birthday party", etc. They were given a few minutes to think about what they wanted to say and were then recorded at 44.1 kHz, using the Blue Snowball USB microphone.

The participants were then asked to think about a negative experience for 20 seconds. Pre-empting the children speaking about traumatic experiences and becoming overly-distress, they were asked to speak about something along the lines of, "tell me about a time you got a bad grade" or "tell me about a time when something wasn't fair" or "tell me about a time when you argued with your friend", etc. Again, they were given a few minutes to think about it and were then recorded once they indicated that they were ready.

If the participant struggled to talk about a personal experience, they were asked to tell the researcher about a favourite part in a movie or a sad part in a movie. However, this strategy was only used as a last resort, as it is thought that prosody may be more "genuine" when speaking about personal experience.

Ethical Considerations

This study followed the ethical guidelines for research with human subjects outlined by the University of the Witwatersrand Codes for Research. Ethical clearance was obtained by the Medical Research Ethics Committee (Human Subjects) of the University of the Witwatersrand (protocol number: M150526, Appendix F). Approval for testing children attending public schools in Gauteng was obtained from the Gauteng Department of Education (reference numbers D2016/049, D2016/335A and D2017/319AA; see Appendix E).

The aims of the investigation were outlined to each of the participants and their legal guardians/parents. They were, however, informed that they may receive some misleading information when necessary, so as not to affect test results. It was necessary to withhold the rigged design of the frustration condition during the Affective Posner Cueing Task, however, all participants were debriefed immediately upon conclusion of the experiment. The participants were encouraged to approach the researcher if they have had any questions. In addition, the aims of the research were provided on paper for all participants to examine in their own time. All participants were required to give written assent, as they are legal minors (refer to Appendix C), and their parents or legal guardians were required to fill in a consent form (refer to Appendices A & B). In the instance that the parent gave consent for a child to participate in the research but the child did not assent, the child was not included in the study. Participants were reminded that participation is voluntary and that they would not be advantaged or disadvantaged in any way had they decided to continue with participation. It was emphasised that they could choose to withdraw at any time during the process without any fear of repercussion. Participants who withdrew during any part of the research were not included in the final sample.

To ensure confidentiality and anonymity, participants were only identifiable by a number code, which was used on all documentation and data. Although audio recordings are considered identifiable, the recordings were only associated with the participant code and no personal information was linked to results.

DMDD participants were screened and diagnosed by their attending psychiatrist/psychologist prior to the study, so no ethics were breached in this area.

As non-invasive, low risk instruments were used, no physical harm was anticipated in during the study. Although the experiment was designed to elicit frustration, which may have caused some psychological stress, care was taken not to exceed the minimal risk for paediatric research. This means that the frustration elicited by the tasks was not greater than that which participants would experience on a daily basis (Rich et al., 2005), for example by losing at a video game or taking a test at school. A psychologist or counsellor (from the school where possible, and when not, a psychologist in private practice volunteered her services) was present and available during all sessions for debriefing at the time of the experiment, although none of the participants expressed severe distress.

As data were anonymous, personalised feedback was not possible. Parents or legal guardians and psychiatrists/psychologists were provided with the option to receive a summary of the findings and general trends upon the completion of the research. Possible implications and potential treatment options based on other research in this area was included in this report and parents could decide whether or not to discuss this with their doctors. A summary of the findings and general trends was also sent to the sponsor of the heart rate monitor (Lifemax), principles of participating schools, the Gauteng Department of Education and the researchers at the University of North Carolina. Participants and their parents/guardians were informed the grouped and anonymous data may also be used in future publications or conference presentations.

Overview of Data Analysis

Prior to data analysis, the following raw data had to be prepared:

Respiratory Sinus Arrhythmia (RSA)

The ECG data obtained was converted to ASCII format with freeware called EDFbrowser (http://www.teuniz.net/edfbrowser/) and the inter-beat (RR) interval (IBI) series extracted with QRSTool (freeware written by David Towers and available at http://jallen.faculty.arizona.edu/qrstool_and_cmetx_software_calculating_metrics_cardiac_variability).

The IBI data were visually inspected for missed R-wave detections and artifacts, which were manually edited with CardioEdit software (Brain-Body Centre, University of Illinois at Chicago). Editing consisted of visually identifying outlier points followed by integer arithmetic (e.g., division of outliers caused by failure to detect an R-peak or summation of invalid detections). Less than 5% of each data file required editing. CardioBatch software (Brain-Body Center, University of Illinois at Chicago) was used to calculate RSA, using a method developed by Porges (1985). This method takes maturational shifts in the frequency of spontaneous breathing into consideration when quantifying the amplitude of RSA with age-specific parameters. First, beat-to-beat (R–R) intervals were timed to the nearest millisecond to yield a time series of sequential heart periods. Sequential heart periods were then resampled into 250 millisecond intervals to produce time-based data. A 51-point cubic moving polynomial (Porges and Bohrer, 1990) was used to detrend the time-based series to create a smoothed template. This template was subtracted from the original time-based series to produce a detrended residual series. Subsequently, a band-pass filter was applied to the detrended residual series to extract the variance in the heart period pattern at the frequency of respiration for children (0.12–1.0 Hz to account for the wide range of ages in the study sample). Finally, RSA amplitude was measured as the natural

logarithm of this variance (Bal et al., 2010; Riniolo & Porges, 1997). Values for RSA and heart period were quantified during sequential 30-second epochs for each condition (i.e., baseline, game 1, game 1 recovery, etc.). Averages for the 30-second epoch for each condition were used in data analyses (Heilman et al., 2013).

The Dynamic Affect Recognition Evaluation

For ease of data analyses and interpretation, data were grouped by the "correct emotion", the average latency (in milliseconds) for each emotion was calculated and the accuracy score for each emotion was added. Each subject would then have an average latency for each emotion category, together with a total number of correct responses for each emotion category.

Prosody

For each recording, a band-pass filter (0.2 – 6 kHz) was applied, followed by a reduction in sampling rate to 22.05 kHz, prior to analysis. The resulting audio files were analysed for spectral tilt and fundamental frequency variability using the SHRP Matlab function (Sun, 2002-2016).

Audio files were first separated into sequential 40ms windows with 30ms overlap and the resulting windows were decomposed using a Fast Fourier transformation. F0 was identified as the first peak above the noise floor within each window and the mean peak frequency across all windows was computed. Variability was operationalised as the bandwidth around the fundamental frequency that encompassed 50% and 100% of its peak energy (i.e., the frequency ranges from below to above the fundamental where all energy levels were > 50% of the peak energy). Spectral tilt was operationalised as the grand mean of the slopes of the linear regression of frequency upon amplitude of the FFT from 20 – 5000 Hz for each window.

In addition, audio was assessed using a modified version of the modulation power spectrum (MPS; Singh & Theunissen, 2003), a 2-dimensional Fast Fourier transformation that decomposes the time-varying acoustic signals of a spectrogram into a two-axis space of spectral (frequency) and temporal (time-related) modulation. These joint spectro-temporal representations of sounds have been found to characterise auditory neurons' higher-level processing of isolated natural sounds (Theunissen & Elie, 2014) and organisation of the auditory cortex includes features detectors that correspond to specific spectro-temporal patterns (Norman-Haignere, Kanwisher, & McDermott, 2015). The MPS was applied to 1.5-second sequential time slices with 50% overlap and averaged over the length of the audio file. The frequency resolution of the spectrogram was fixed at 32 Hz per increment, which is the suggested resolution for human speech (Singh & Theunissen, 2003). Each audio file was assessed for total modulation depth, operationalised using the square root of the ratio between power at origin (0, 0), reflecting unmodulated sound, to power in the rest of the spectrum (Singh & Theunissen, 2003). The MPS was also assessed for separability, the probability of any value along one axis not being predicted by its location on the other. In the case of the MPS, this means that frequency modulations are equally likely to occur at any value of temporal modulation. White noise meets the criteria for separability; the temporal and frequency modulations are independent. For speech, there is an interaction between the two distributions, such that less energy is seen in the jointly-high modulation regions, resulting in a distribution that is not perfectly separable.

Attachment Style

The ten items measuring anxious/ambivalent and avoidant attachment styles were averaged to obtain a score for each of these dimensions of attachment style.

All statistical analyses were conducted using SAS 9.3 for Windows. Descriptive statistics and exploration of normality were conducted. Descriptive data analysis was

conducted as follows: Categorical variables were summarised by frequency and percentage tabulation. Continuous variables were summarised by the mean, standard deviation, median and interquartile range.

Baseline demographic and clinical variables that were not used for matching, were compared between groups using the paired *t*-test for years of exposure to English, and McNemar's test for paired categorical data for the remaining variables.

The effect of group, test point (e.g. baseline, reward/no reward, frustration, etc.) and the group-test point interaction on each of the other outcomes (i.e., RSA, HP, accuracy, latency, etc.) was determined using a repeated measures mixed model with the outcome as the dependent variable, and group, test point and the group-test point interaction as independent variables. Group was treated as a repeated measured to capture the paired (matched case-control) nature of the data. Post-hoc comparisons were conducted using the Tukey Kramer adjustment for multiple comparisons.

Sample sizes were calculated using G*Power (Faul, Erdfelder, Lang, & Buchner, 2007). Sample size estimation depends on the main research question, in this case the comparison of the outcomes between cases and matched control groups. This necessitated the use of the paired samples *t*-test. Detection of a small, medium or large effect size (dz = 0.2, 0.5 and 0.8 respectively) with 80% power at the 5% significance level, required sample sizes of 199, 34 and 15, respectively. At least a medium effect size is ideal, should it exist; thus, a sample size of at least 34 would have been recommended for this study. The actual sample size of 30 used in this study was deemed sufficient to allow for the detection of medium to large effect sizes (dz = .53). However, a larger sample size would have added to the statistical power. Consequently, the small sample size is a significant limitation of the study.

This chapter outlined the research methodology employed for this study. The initial section provided a detailed description of the research aims and design. The main aim was to explore the differences in vagal regulation and behavioural outcomes in children with DMDD compared to typically developing control participants. Specifically, the study was designed to answer questions on variability in respiratory sinus arrhythmia at baseline, during and after certain test points that include reward and frustration conditions, as well as differences in behavioural and emotional outcomes for each of these tasks. The research also sought to explore whether children with DMDD are distinguishable from healthy participants in terms of acoustic modulation, attachment style, and social and communication skills. Finally, the research aimed to determine whether parents considered medication to result in improvements in social and communication behaviour in children with DMDD. Next, the research measures, instrumentation and procedure employed in the study were discussed. The chapter continued with a discussion of the ethical considerations of the study and concluded with an overview of the data analysis. The next chapter presents the results of the statistical analyses undertaken to investigate these research questions.

Chapter 6: Results

This chapter is organised around the research aims of the study. The first was to determine whether there is variability in the influence of the myelinated vagus to the heart in a paediatric population with disruptive mood dysregulation disorder (DMDD) compared to matched control cases at baseline and in response to reward, frustration and affective conditions by comparing respiratory sinus arrhythmia (RSA) and heart period (HP). The second component of the study was to investigate whether children with DMDD exhibit characteristics of a compromised social engagement system (SES) as conceptualised by the polyvagal theory (Porges, 2001, 2003). To address these objectives, between-group comparisons were undertaken on the accuracy and reaction time scores obtained during emotional, frustrating, and emotion recognition tasks. The groups were also compared in terms of emotional tone variables extracted from voice recordings to establish whether they vary in terms of expressive prosody. An examination of these results is preceded by a thorough exploration of the descriptive data for the DMDD and matched control groups.

Descriptive Data

This section details the descriptive results of the all the study variables. Normality tests were not performed, as these have little power to reject the null hypothesis in small samples (Öztuna, Elhan, & Tüccar, 2006). Non-normality of the residuals of the repeated measures mixed model was established by inspecting model diagnostics. Descriptive analyses for the categorical variables, namely the social behaviour and communication variables, entailed frequency and percentage tabulation.

Table 6 describes the levels of social behaviour and communication obtained from the Social Behaviour and Communication: Parent Questionnaire (Bazhenova & Porges, 2000) of both groups from their parents' perspective in terms of percentages.

Table 6

Social Communication and Behavioural Variables, by Group

Variable	Category	Group			
		Control		DMDD	
		n	%	*n*	%
		15		15	
Gestural and facial expression	No problem	15	100	7	47
	Problem indicated, deterioration				
	Problem indicated, no/vague improvement			5	33
	Problem indicated, improvement			3	20
Language and social interaction	No problem	15	100	2	13
	Problem indicated, deterioration			2	13
	Problem indicated, no/vague improvement			3	20
	Problem indicated, improvement			8	53
Listening and Processing Sound	No problem	15	100	5	33
	Problem indicated, deterioration			1	7
	Problem indicated, no/vague improvement			7	47
	Problem indicated, improvement			2	13
Emotional Regulation and Reciprocity	No problem	15	100		
	Problem indicated, deterioration			2	13
	Problem indicated, no/vague improvement			9	60
	Problem indicated, improvement			4	27
Behaviour	No problem	15	100	3	20
	Problem indicated, deterioration				
	Problem indicated, no/vague improvement			8	53
	Problem indicated, improvement			4	27

Note: No problem = age and developmentally appropriate behaviour/communication; Problem indicated, deterioration = behaviour/communication is not age and/or developmentally appropriate, and has deteriorated since the commencement of medication; Problem indicated, no/vague improvement = behaviour/communication is not age and/or developmentally appropriate, and has remained unchanged or mostly unchanged since the commencement of medication; Problem indicated, improvement = behaviour/communication is not age and/or developmentally appropriate, and has improved since the commencement of medication.

All of the children in the control group were considered to exhibit age- and developmentally appropriate social and communication behaviour by their parents. Parents of children in the DMDD group, however, reported problems in most areas. Fifty-three percent of parents indicated that their children experienced problems using body language and facial

expression in communication (e.g., using gestures to communicate something in a quiet movie theatre or religious service where talking is prohibited, making appropriate eye contact, using appropriate facial expressions while listening, turning to face towards another person when communicating, and tilting the head to indicate listening or interest). Of these, only 20% observed an improvement in this area following the commencement of medication.

Most of the children in the DMDD group (87%) experienced problems in terms of language and social interaction, including, a) not using verbal language appropriately to relate thoughts, feelings and events which have happened, are happening, or will happen; b) participating in a reciprocal conversation which continues beyond two exchanges (asking a question and answering it), switching topics and talking about something not on his or her own agenda, and talking without using scripts from movies or television; c) initiating social interactions for the sake of social pleasure; d) responding with reciprocity in peer relationships; and e) perceiving and adapting to convert social cues (i.e., reading social situations appropriately and adapting when the demands on social behaviour are changed or unstated. For example, does he or she know to be quiet in an audience setting or to use a quiet voice when inside). The majority of these parents (53%) noticed an improvement in their children's use of language and their social interaction after medication had been started.

Two-thirds of the children in the DMDD group also had difficulties with listening to and processing sound, including understanding of spoken words without visual or contextual cues and tolerating different sounds and noise without distress. In seven percent of these children, the parents indicated that the problems had deteriorated following the start of medication, while 47% noticed no or little improvement and 13% observed an improvement.

Expectedly, all of the children in the DMDD group had trouble with emotional regulation and reciprocity, including regulating emotions in an appropriate manner (e.g., reacting to unexpected changes in circumstances without fear, tantrums, or over-excitement),

experiencing unusual fears or fears that cannot be explained, and expressing and responding to emotions appropriately. Medication made no or little difference in 60% of the children. Parents reported a worsening since starting medication in emotional regulation and reciprocity in 13% of the children, while 27% showed an improvement.

Problematic behaviour was reported in 80% of the participants in the DMDD group, particularly engaging independently and effectively in play activities, flowing with unexpected changes in routine or environment, and engaging in preoccupations or unusual interests.

Continuous variables measured during the Affective Posner Cueing Task, namely response time, accuracy, RSA and HP, were summarised by the means and standard deviations. As almost all of the continuous variables were not normally distributed, medians and interquartile range are also reported. Table 7 reports the means, the standard deviations, the medians and interquartile ranges for response time measured during the Affective Posner Cueing Task.

The participants in the DMDD group were slower to respond to the Affective Posner stimuli than those in the control group during the practice round. Both groups improved their response times from the practice round to game 1 and were slower once affective feedback and rewards were introduced in game 2. Participants in both groups reduced their response time further during the frustration condition, and both groups took longer to respond to neutral cues compared to valid and invalid cues. The groups responded differently to valid and invalid cues, however. Participants in the DMDD group were slower to respond to invalid cues than to valid ones, whereas participants in the control group showed the opposite pattern and responded faster to invalid cues than to valid cues.

Table 7

Descriptive Statistics for Affective Posner Cueing Task Response Time (in milliseconds), by Group

	Control (*n* = 15)				DMDD (*n* = 15)			
	M	*SD*	*Mdn*	*IQR*	*M*	*SD*	*Mdn*	*IQR*
Practice	446.9	75.5	427.4	399.2 - 492.7	509.6	123.3	523.7	451.4 - 613.7
Game 1 (Baseline)	389.7	78.4	388.4	325.1 - 432	465.7	130.6	448	348.7 - 581.1
Game 2 (Affective Feedback/Reward)	393	69.2	391.2	355.6 - 443.3	484.1	85.3	476.5	434.7 - 561
Game 3 (Affective Feedback/Reward)	249.5	68.9	225.3	197.2 - 290.5	335.1	110.9	238.4	256.1 - 450.3
Invalid Cues	330.9	68.6	319	282.6 - 353.1	442.7	115.5	397.5	376.8 - 522.7
Valid Cues	327.6	63.5	329.3	275.7 - 359.6	399.4	100.8	368.5	329.5 - 499
Neutral Cues	361.1	76.9	344.8	300.7 - 400.8	463.2	85.3	425.1	407.6 - 517.9
Game 1 Invalid Cues	395.3	93	374	330.2 - 463.1	480.6	135.9	498.3	378.1 - 576.6
Game 1 Valid Cues	380.3	76	370.7	314.2 - 416	445.2	140.9	421.9	346.7 - 558
Game 1 Neutral Cues	412.7	95.1	404.2	322 - 487	512.3	120.2	507.2	435.8 - 624.8
Game 2 Invalid Cues	405	78	394.1	347.2 - 459.2	530.1	101.3	512.6	457.1 - 635.1
Game 2 Valid Cues	385.4	67.9	396.6	341.8 - 422.7	461.5	93.2	464.8	392.7 - 535.9
Game 2 Neutral Cues	404.1	79.9	393.6	351.5 - 426.2	505.8	73.9	522.6	463.8 - 563.9
Game 3 Invalid Cues	224.5	74.1	182.8	176.2 - 278.8	336.4	139.8	273.2	219.9 - 440.1
Game 3 Valid Cues	243.6	67.4	227	197.8 - 280.5	314.3	109.1	312.4	230.5 - 427.4
Game 3 Neutral Cues	292.3	80.1	273.4	224.8 - 344.9	396.1	108.4	336.4	311.9 - 484.8
Post-positive Feedback	362.9	67.1	362.1	307.9 - 399.1	446.1	94.3	412.7	384.3 - 544.6
Post-negative Feedback	315	78.3	299.1	250.5 - 333.7	413.6	124	407.7	342.7 - 492.6
Post-frustration Feedback	240.4	69.1	219.8	197.3 - 261.6	330.1	121.8	326	235.4 - 446.1
Reward	314.1	61	313	265.6 - 326.6	401.4	94.4	395.4	331.3 - 481.4
No Reward	328.5	71.6	312.2	280-384	417.8	99.4	373.3	350.6-493.9
Game 2 Reward	387	69.8	380.6	348.7 - 443.4	476.1	85	462.9	428.2 - 536.8
Game 2 No Reward	399.1	70.8	400.9	362.2 - 443.7	492	88	490.1	440.1 - 565.3
Game 3 Reward	241.2	63.1	221.9	195.1 - 267.9	80.9	13.9	78	61.5 - 114
Game 3 No Reward	257.9	78.6	228.3	201 - 306.7	343.6	116.2	284.7	260.1 - 422.5

When rewards were introduced, the control group was only slightly slower to respond compared to no-reward trials, while the DMDD group took longer to respond to reward trials than no-reward trials.

The extent to which these differences were statistically significant is discussed under the inferential statistics section. Table 8 reports the means, the standard deviations, the medians and interquartile ranges for accuracy of responses on the Affective Posner Cueing Task.

Table 8

Descriptive Statistics for Affective Posner Cueing Task Accuracy (in percentage), by Group

	Control (*n* = 15)				DMDD (*n* = 15)			
	M	*SD*	*Mdn*	*IQR*	*M*	*SD*	*Mdn*	*IQR*
Practice	0.9	0	0.9	0.9 - 1	0.9	0.1	0.9	0.8 - 1
Game 1 (Baseline)	1	0	1	0.9 - 1	0.9	0.1	0.9	0.9 - 1
Game 2 (Affective Feedback/Reward)	1	0	1	1 - 1	1	0	1	0.9 - 1
Game 3 (Affective Feedback/Frustration)	0.8	0.1	0.8	0.8 - 0.8	0.8	0.1	0.8	0.8 - 0.9
Invalid Cues	0.7	0.1	0.7	0.5 - 0.8	0.7	0.2	0.7	0.6 - 0.8
Valid Cues	1	0	1	1 - 1	1	0	1	1 - 1
Neutral Cues	0.9	0	0.9	0.9 - 1	0.9	0.1	1	0.9 - 1
Game 1 Invalid Cues	0.9	0.1	0.9	0.8 - 1	0.8	0.2	0.8	0.6 - 0.9
Game 1 Valid Cues	1	0	1	1 - 1	1	0.1	1	1 - 1

(continued)

(continued)

	Control (*n* = 15)				DMDD (*n* = 15)			
	M	*SD*	*Mdn*	*IQR*	*M*	*SD*	*Mdn*	*IQR*
Game 1 Neutral Cues	1	0	1	1 - 1	1	0	1	0.9 - 1
Game 2 Invalid Cues	0.9	0.1	0.9	0.9 - 1	0.9	0.1	0.9	0.9 - 1
Game 2 Valid Cues	1	0	1	1 - 1	1	0	1	1 - 1
Game 2 Neutral Cues	1	0	1	1 - 1	1	0.1	1	0.9 - 1
Game 3 Invalid Cues	0.4	0.2	0.3	0.2 - 0.5	0.5	0.2	0.6	0.3 - 0.6
Game 3 Valid Cues	0.9	0	1	0.9 - 1	0.9	0.1	0.9	0.9 - 1
Game 3 Neutral Cues	0.8	0.1	0.8	0.8 - 0.9	0.9	0.1	1	0.8 - 1
Post-positive Feedback	0.9	0.1	0.9	0.9 - 1	0.9	0	0.9	0.9 - 1
Post-negative Feedback	0.9	0.1	0.9	0.8 - 1	0.9	0.1	0.8	0.7 - 1
Post-frustration Feedback	0.8	0.1	0.8	0.7 - 0.8	0.8	0.1	0.8	0.8 - 0.9
Reward	0.9	0	0.9	0.9 - 0.9	0.9	0.1	0.9	0.9 - 1
No Reward	0.9	0	0.9	0.9 - 0.9	0.9	0.1	0.9	0.9 - 0.9
Game 2 Reward	1	0	1	1 - 1	1	0.1	1	0.9 - 1
Game 2 No Reward	1	0	1	0.9 - 1	1	0	1	0.9 - 1
Game 3 Reward	0.8	0.1	0.8	0.7 - 0.8	0.8	0.1	0.8	0.7 - 0.8
Game 3 No Reward	0.8	0.1	0.8	0.8 - 0.8	0.8	0.1	0.8	0.7 - 0.9

Across all three games, cue type, reward condition and feedback type the average accuracy was very similar for both groups. The DMDD group was slightly less accurate following negative feedback compared to positive feedback, while the control group's

median accuracy remained the same for both feedback types. Both groups made more errors following frustrative feedback compared to positive feedback.

The extent to which these differences were statistically significant is considered under the inferential statistics section. Table 9 reports the means, the standard deviations, the medians and interquartile ranges for respiratory sinus arrhythmia measured during the Affective Posner Cueing Task.

Table 9

Descriptive Statistic for Affective Posner Cueing Task Respiratory Sinus Arrhythmia (RSA, in msec^2), by Group

	Control (*n* = 15)				DMDD (*n* = 15)			
	M	*SD*	*Mdn*	*IQR*	*M*	*SD*	*Mdn*	*IQR*
Pre-task Baseline	8	0.9	8	7.1 - 9.2	7.4	1.3	7.5	6.6 - 8.2
Practice	8.1	1.2	8	7.4 - 9.1	7.2	1.2	7.4	6.8 - 8
Game 1	7.8	1.1	7.6	6.9 - 8.7	7	1.1	7.2	6.8 - 7.6
Game 1 Recovery	7.9	1	7.8	7 - 8.7	7.2	1	7.2	6.7 - 8.2
Game 2	7.7	1.1	7.6	7 - 8.7	7.2	1	7.3	7.1 - 7.7
Game 2 Recovery	8	0.9	8	7.3 - 8.7	7.7	1.6	7.2	6.6 - 8.8
Game 3	7.8	1	7.8	7.2 - 8.6	7.3	1	7.4	6.7 - 7.9
Game 3 Recovery	8	0.9	8.1	7.1 - 8.7	7.4	1.2	7.3	6.7 - 8.9

The median respiratory sinus arrhythmia did not vary much between the groups at baseline or at any point during the Affective Posner Cueing Task.

The extent to which these differences were statistically significant is discussed under the inferential statistics section. Table 10 reports the means, the standard deviations, the medians and interquartile ranges for heart period during the Affective Posner Cueing Task.

Table 10

Descriptive Statistics for Affective Posner Cueing Task Heart Period (HP, in milliseconds), by Group

	Control (*n* = 15)				DMDD (*n* = 15)			
	M	*SD*	*Mdn*	*IQR*	*M*	*SD*	*Mdn*	*IQR*
Pre-task Baseline	759.7	82.6	768.3	687.5 - 807.4	671.7	86.7	653.8	618.8 - 676.6
Practice	761.4	74.6	763.6	714.1 - 806.4	681.8	91.9	662.4	617.9 - 714.9
Game 1	738.5	76.1	747.1	692.9 - 779.3	666.9	83.5	644.3	621.9 - 717.1
Game 1 Recovery	748.9	81.4	733.9	701.1 - 786.1	671.8	79.3	634.8	625.1 - 703.6
Game 2	732.2	71.6	736.8	691.6 - 797.8	666.9	83.5	644.3	621.9 - 717.1
Game 2 Recovery	746.7	77.9	741.5	693.9 - 804.5	684.4	93	640.2	624.6 - 782.2
Game 3	739.3	75.2	733.7	708.2 - 780.8	679.9	83.7	647.1	633.1 - 722
Game 3 Recovery	742.2	71.9	746	698.3 - 802.2	668.4	70.3	646.9	634.8 - 688.9

Average heart period was shorter for the DMDD group compared to that of the control group at baseline and each test point of the Affective Posner Cueing Task. The extent to which these observed differences between groups in response time, accuracy, RSA and heart period during the Affective Posner were statistically significant is reported under the inferential statistics section.

Table 11 encapsulates the descriptive statistics for the continuous arousal and valence variables measured using the Self-Assessment Manikin (© Peter J. Lang, 1994) during the Affective Posner Cueing Task, which participants completed prior to the practice round (baseline) and again at the conclusion of each game.

The participants in the DMDD group reported more arousal at baseline than those in the control group. Self-reported arousal was similar for both groups during the first two games. Both groups reported highest arousal after game 2, which was the reward/no-reward condition, and the least following game 3, the frustration condition.

Table 11

Descriptive Statistics for Self-Assessment Manikin, by Group

	Control (*n* = 15)				DMDD (*n* = 15)			
	M	*SD*	*Mdn*	*IQR*	*M*	*SD*	*Mdn*	*IQR*
Arousal								
Pre-Task Baseline	5.3	2.1	5	4 - 7	7.1	2.2	8	5 - 9
Game 1	7.1	1.5	7	6 - 9	6.2	2.5	7	5 - 9
Game 2	7.1	2.2	8	5 - 9	6.2	3.3	8	2 - 9
Game 3	6.9	2.1	7	5 - 9	5.4	3.4	6	1 - 9
Valence								
Pre-Task Baseline	6.8	1.7	7	5 - 8	7.1	1.9	7	6 - 9
Game 1	6.8	2.2	7	6 - 9	7.7	1.7	9	6 - 9
Game 2	7.7	1.6	9	6 - 9	6.3	3.5	8	1 - 9
Game 3	5.6	2.6	5	4 - 9	5.3	3.1	5	2 - 9

Note: Arousal and valence were measure on a 9-point Likert type scale, with 1 being “least” and 9 being “most”.

During the third game, participants in the DMDD group reported slightly less arousal than participants in the control group. Both groups conveyed comparable levels of valence at baseline and after game 3. The participants in the DMDD group reported feeling happier during game 1 (neither reward nor frustration) than game 2, while the inverse was reported by participants in the control group.

The statistical significance, or lack thereof, of these findings is reported under the inferential statistics section. Table 12 summarises the descriptive statistics for the continuous variables measured using the Observed Frustration Reactivity scale (Degnan et al., 2008). These ratings were made by the researcher during the completion of the frustration condition (game 3).

Table 12

Descriptive Statistics for the Observed Frustration Reactivity, by Group

	Control (*n* = 15)				DMDD (*n* = 15)			
	M	*SD*	*Mdn*	*IQR*	*M*	*SD*	*Mdn*	*IQR*
Negative reactivity								
Frowning	0.9	0.8	1	0 - 1	1.5	0.8	2	1 - 2
Sighing	1.5	0.9	1	1 - 2	1.5	1.2	2	0 - 2
Pouting	0.9	1	1	0 - 2	1.2	0.9	1	0 - 2
Whining	0.8	1.3	0	0 - 2	1.3	1	1	0 - 2
Fussing	1.7	1.2	1	1 - 3	1.9	1.1	2	1 - 3
Crying	0	0	0	0 - 0	0	0	0	0 - 0
Swearing	0	0	0	0 - 0	0.2	0.8	0	0 - 0
Shouting	0.1	0.5	0	0 - 0	0	0	0	0 - 0
Tantrumming	0	0	0	0 - 0	0	0	0	0 - 0
Global Distress	5.9	3.8	5	3 - 9	7.5	3.4	7	6 - 10
Global Affect	0.1	1.9	0	-1 - 2	-1	0.9	-1	-2 - 0

Note: Frustration behaviour was coded according to a) global negative reactivity or distress: rated for each behaviour during the entire frustration condition on a scale from 0 (no negative response) to 4 (extreme distress) and added for a global score; and b) global affect: rated for the entire frustration condition on a scale from −3 (highly negative affect) to 3 (highly positive affect).

Participants in the control group exhibited fewer negative reactivity behaviours (i.e., distress) during the frustration condition (game 3) than participants in the DMDD group, although the only shouting that occurred was in the control group. In the DMDD group, swearing was the most negative reactivity behaviour observed, but this was only one participant.

None of the participants in either group had tantrums. Observed global distress was higher in the DMDD group than in the control group, while observed global affect was higher in the control group than in the DMDD group. The degree to which these differences are statistically significant are explored later in the inferential statistics section.

The continuous variables measured during the Dynamic Affect Recognition Evaluation (DARE) are summarised in the Table 13. These refer to the physiological

measurements of RSA and HP in milliseconds during the recognition of various emotional expressions on the DARE.

Table 13

Descriptive Statistics for the Dynamic Affect Recognition Physiological Measurements (in milliseconds), by Group

	Control (*n* = 15)				DMDD (*n* = 15)			
	M	*SD*	*Mdn*	*IQR*	*M*	*SD*	*Mdn*	*IQR*
RSA								
Baseline	8.1	1.2	8.3	6.9 - 9.2	7.4	1.1	7.3	6.5 - 8.3
Video	7.9	1.4	7.9	6.7 - 8.9	7.5	1	7.6	6.6 - 8.3
Recovery	7.9	1.5	8.3	6.9 - 9.3	7.3	1.2	7.2	6.5 - 8.3
HP								
Baseline	787.7	136.8	749.6	711.2 - 884.6	751.1	129.3	724.4	665.8 - 817.1
Video	760.2	117.4	730.5	655.5 - 844.5	738.6	119.2	717.4	668.2 - 793.6
Recovery	757.6	113.8	739.4	656.8 - 818	750.8	132.8	735.6	663.3 - 790.1

Respiratory sinus arrhythmia was similar for both groups at baseline, during the DARE task and at recovery thereafter. During the task, however, the RSA of the control group decreased from the baseline by 0.4 milliseconds, while that of the DMDD group increased by 0.3 milliseconds. Heart period was longer in the control group at before and during the DARE task than in the DMDD group (i.e. indicating a faster heart rate in the latter), but more similar during recovery. The continuous performance variables measured during the DARE are summarised in Table 14.

Table 14

Descriptive Statistics for the Dynamic Affect Recognition Performance, by Group

	Control (*n* = 15)				DMDD (*n* = 15)			
	M	*SD*	*Mdn*	*IQR*	*M*	*SD*	*Mdn*	*IQR*
Accuracy (%)								
Anger	0.7	0.2	0.7	0.6 - 0.9	0.6	0.2	0.7	0.4 - 0.9
Disgust	0.7	0.3	0.7	0.4 - 0.9	0.5	0.4	0.6	0.1 - 0.9
Fear	0.7	0.3	0.9	0.6 - 0.9	0.5	0.3	0.4	0.1 - 0.7
Happiness	1	0.1	1	0.9 - 1	0.9	0.2	1	0.9 - 1
Sadness	0.9	0.2	1	0.9 - 1	0.9	0.2	0.9	0.7 - 1
Surprise	1	0.1	1	1 - 1	0.9	0.1	0.9	0.9 - 1
Response Time (msec)								
Anger	13.7	3.3	14	11.1 - 15.3	15.9	3.9	16.4	12.3 - 19.8
Disgust	10.8	2.5	9.7	9.4 - 12	11	2.6	9.8	8.7 - 13.9
Fear	13.4	2.9	13	12 - 14.3	13.6	2.5	12.9	11.8 - 16.3
Happiness	9.9	2.4	9.2	8.4 - 10.5	10.4	2.5	10.2	8.2 - 12.8
Sadness	11.2	3.3	10.5	8.8 - 12.8	12.1	3.5	12.5	9.1 - 14.5
Surprise	10	2.2	9.3	8.3 - 12.2	11	2.4	10.6	9.2 - 11.7

The groups were comparable in their accuracy scores for all of the target emotions, except fear, on which the DMDD group scored much lower. The identification of the control group was a little faster, except on fear and disgust, for which the response time of the latter was similar to that of the DMDD group. The statistical power of these results will be discussed in the section on inferential statistics.

The descriptive statistics of the sample's physiological measurements (i.e., RSA and HP, in milliseconds) during the completion of the prosody task are contained in Table 15.

Respiratory sinus arrhythmia was slightly lower in the DMDD group at baseline and at recovery, as well as both the positive and negative prosody tasks. Heart period was similar for both groups during baseline, but differed at each prosody test point and at recovery. The control group's shortest heart period was during the positive task, while the DMDD group's heart period was shortest during the negative task. At recovery, the control group's heart period was longer than, and further away from, their baseline.

Table 15

Descriptive Statistics of Participants' Physiological Measurements during the Prosody Task, by Group

	Control (*n* = 15)				DMDD (*n* = 15)			
	M	*SD*	*Mdn*	*IQR*	*M*	*SD*	*Mdn*	*IQR*
RSA ($msec^2$)								
Baseline	8	1.3	8.3	7.1 - 8.8	7.6	1.2	7.9	7 - 8.6
Negative	8	1.1	8.3	7.4 - 8.7	7.4	1.1	7.7	6.8 - 8.1
Positive	8	1.2	8.2	7.1 - 9	7.5	1.1	7.6	7 - 8
Recovery	8	1.2	8.4	7.4 - 9	7.7	1	7.7	7.1 - 8.2
HP (msec)								
Baseline	765.5	122.6	738.7	666 - 820.4	773.9	134.2	729.7	699.9 - 830.4
Negative	754	93.8	748.8	670.4 - 797.2	749.2	127	707.4	673.5 - 766.6
Positive	745	112.1	717	646.8 - 824.2	750.5	116.9	721.8	671.4 - 776.3
Recovery	759.2	91.2	758.8	669.1 - 801.7	769.8	127	739.9	686.9 - 804.4

The DMDD group's heart period was also longer than their baseline, but only by 10.2 milliseconds. The descriptive statistics of the sample's prosodic measurements (i.e., modulation depth, bandwidth of fundamental frequency and spectral tilt) during the completion of the prosody task are contained in Table 16.

Table 16

Descriptive Statistics of Participants' Acoustic Features during the Prosody Task, by Group

	Control (*n* = 15)				DMDD (*n* = 15)			
	M	*SD*	*Mdn*	*IQR*	*M*	*SD*	*Mdn*	*IQR*
Modulation Depth								
Negative	0.8	0.1	0.8	0.7 - 0.8	0.8	0.1	0.8	0.8 - 0.9
Positive	0.8	0.1	0.7	0.7 - 0.9	0.9	0.2	0.9	0.7 - 0.9

(continued)

(continued)

	Control (n = 15)				DMDD (n = 15)			
	M	*SD*	*Mdn*	*IQR*	*M*	*SD*	*Mdn*	*IQR*
Bandwidth of fundamental frequency (BW50)								
Negative	33.1	3.3	31.8	30.9 - 35.8	34.3	3.2	34	32.9 - 36.1
Positive	33.4	2.6	33.1	31.5 - 35.2	33.8	3.9	32	31.3 – 37.2
Spectral Tilt								
Negative	-.00493	.00074	-.00497	-.00536 - -.00443	-.00471	.00078	-.00450	-.00510 - -.00431
Positive	-.00512	.00082	-.00532	-.00557 - -.00442	-.00466	.00096	-.00447	-.00503 - -. 00403

The median values of the prosodic variables, which measure the modulation of intonation of the voice, of the two groups seemed similar, however whether these differences were statistically significant, is discussed in the next section.

Inferential Data

In order to establish whether significant differences between the groups exist, they were compared on all variables, except those used for matching (i.e., age, gender and ethnicity), using the paired *t*-test, and McNemar's test for paired categorical data.

The effect of group membership (DMDD or control), test point (i.e. Affection Posner Task, SAM, Observed Frustration Reactivity, DARE, and prosody task) and group-test point interactions on the various outcomes of the measures was determined using a repeated measures mixed effects model with the outcome (i.e. RSA, HP, response time, accuracy, valence, arousal, attachment scores, and prosodic features) as the dependent variables, and group, test point and the group-test point interaction as independent variables. Group was treated as a repeated measured to capture the paired (matched case-control) nature of the data. To minimise Type I errors, post hoc comparisons were conducted using the Tukey-Kramer adjustment for multiple comparisons. This method was also preferred, as it does not assume

equal sample sizes given that the samples in this study were not equal for all the tasks (two children declined to participate in the prosody task) and some of the independent variables had various levels (such as game type, cue type, trial type, and feedback during the Affective Posner Cueing Task).

Demographic Details

The two groups were compared on those demographic variables that were not used for matching. These included English language proficiency, home language, medical aid and parent levels of education. As English language proficiency was a requirement for participation, the groups were compared in terms of the number of years exposure to English. A paired t-test revealed no significant difference between the groups on this variable (*adj* M_{diff} = -0.6; *adj p* = .085), 95% CIs [-1.3, 0.1]. Exact McNemar's tests showed no statistically significant between-group differences on the demographic variables of home language ($p = 0.6$), medical aid ($p = 0.3$), or parental level of education for both parents ($p = 0.2$ for the first parent and $p = 0.1$ for the second parent), indicating that the DMDD and control groups were comparable on all of these variables.

Respiratory Sinus Arrhythmia and Heart Period

The first research aim was to determine whether significant between-group differences exist in terms of respiratory sinus arrhythmia (RSA), or vagal tone, and heart period at baseline and in response to the Affective Posner Cueing Task (Perez-Edgar & Fox, 2005), the Dynamic Affect Recognition Evaluation (DARE; Porges et al., 2007) and prosody tasks. It was hypothesised that participants diagnosed with DMDD would have significantly lower RSA amplitude and shorter heart period than participants in the control group.

***Affective Posner Cueing Task*.** The effects of group ($F[1,15] = 4.19$, $p = .058$), game ($F[7,23] = 1.77$, $p = .141$) and group x game interaction ($F[7,23] = 1.11$, $p = .389$) on RSA

were non-significant. This indicates that no significant difference was evident between groups in vagal regulation of the heart.

However, the effects of group (F[1,15] = 8.6, p = .011) and game (F[7,22] = 3.53, p = .011) on heart period were significant. At baseline and during all three games of the Affective Posner Cueing Task, heart period was significantly higher for the control group (*adj M* = 746; *adj p* < .001), 95% CIs [706, 786] compared to the DMDD group (*adj M* = 675; *adj p* < .001), 95% CIs [635, 715]. This means that participants in the DMDD group had a faster heart rate than those in the control group at every measurement point. Although this was expected, shorter heart period in the absence of significantly lower RSA amplitude does not necessarily implicate the vagus nerve in the variability of between heart beats. The group x game interaction was not significant (F[7,22] = 0.67, p = .697).

Post hoc tests showed that heart period for the practice game was significantly higher than that for game 1 (baseline) across both groups (*adj* M_{diff} = -18.89, *adj p* = .003), 95% CIs [-32.79, 80.06]. This was, however, the game that showed a significant difference, indicating that, once the experiment formally commenced, the game condition (i.e., baseline, contingencies, and frustration) did not significantly affect participants' heart period.

Dynamic Affect Recognition Evaluation (DARE). The effect of group on RSA during the DARE was not significant (F[1,15] = 1.33, p = .267). This finding, which was not anticipated, suggests that groups responded similarly during the DARE task in terms of vagal regulation of the heart. Group membership did not significantly affect recovery RSA either (F[1,14] = 0.86, p = .369).

Heart period during the DARE did not vary significantly between the two groups (F[1,16] = 0.68, p = .423). The mean recovery heart period after the completion of the DARE was significantly lower (F[1,1] = 4.97; p = .043) for the control group (*adj M* = 741; p < .001), 95% CIs [721, 761] compared to the DMDD group (*adj M* = 767; p < .001), 95% CIs

[747, 787], controlling for heart period at baseline. Again, in the absence of reduced RSA amplitude, this finding does not exclude sympathetic, hormonal, or homeostatic influences on heart rate variability. There were no other significant between-group differences in heart period.

Prosody. No between-group differences in RSA or heart period were found during the positive and negative prosody tasks or on the subsequent recovery period, which did not support the hypothesis that participants diagnosed with DMDD would have lower vagal tone and more instability in prosody than participants in the control group. The results of these analyses are summarised in Table 17.

Table 17

Post hoc Results of RSA and Heart Period during the Prosody Task

Measure	Test Point	*Adj* M_{diff}	*p*	*Adj p*	Lower	Upper	Adj Lower	Adj Upper
RSA	Positive	0.04	.797	.797	-0.30	0.38	-0.30	0.38
	Negative	0.22	.073	.073	-0.023	0.4552	-0.02	0.46
	Recovery	0.09	.604	.604	-0.26	0.43	-0.26	0.43
HP	Positive	1.80	.880	.880	-22.62	26.22	-22.62	26.22
	Negative	21.37	.151	.151	-9,10	51.83	-9.09	51.83
	Recovery	1.24	.881	.881	-16.55	19.03	-16.55	19.03

Note. Tukey-Kramer adjustment used. $\alpha = .05$.

In summary, the groups did not differ significantly in RSA or heart period at baseline or in response to emotional, frustrating or emotion recognition tasks.

Posner Behavioural and Affective Data

The second research goal was to establish whether any significant between-group differences existed in behavioural (i.e., response time/latency and accuracy) and/or self-reported and observed affective (valence and arousal) data during the Affective Posner Cueing Task.

Posner Effect. Previous studies (Hugdahl & Nordby, 1994; Posner, 1988; Posner & Cohen, 1984), have found faster responses in healthy children and adults to valid versus invalid cues, as invalid trials require participants to shift their attention from the location of the cue to that of the target. To explore this "Posner effect" (i.e., the response time costs and benefits of cued versus non-cued targets), the response times, in milliseconds, for the various cue types (invalid, valid, and neutral) were compared across the two groups.

The effect of cue type on response time was significant (F[2,27] = 8.65, p = .001) during game 1 (baseline). Post hoc analyses showed that average response time for valid trials was significantly faster than for neutral trials, across both groups, and when controlling for the practice test (results are summarised in Table 18). At this test point, response time differences between valid versus invalid cues and neutral versus invalid cues were non-significant.

Table 18

Post hoc Results of Cue Type Effect on Response Time during Posner Baseline (Game 1)

Cue Type 1	Cue Type 2	*Adj* M_{diff}	*p*	*Adj p*	Lower	Upper	Adj Lower	Adj Upper
Valid	Neutral	49.79	< .001	.001	24.79	74.80	19.53	80.06
Valid	Invalid	25.25	.023	.059	3.71	46.79	-0.83	51.32
Neutral	Invalid	-24.54	.075	.174	-51.75	2.67	-57.48	8.39

Note. Tukey-Kramer adjustment used. α = .05.

At this test point, the effects of group (F[1,16] = 1.96, p = .181) and group x cue type interaction on response time (F[2,27] = 1.12, p = .342) were not significant.

During game 2 (reward and emotive feedback condition), the effects of group (F[1,12] = 18.64, p = .001) and cue type (F[2,27] = 9.75, p < .001) were significant. Post hoc tests showed that the least mean squares response time for valid cues was significantly lower than that for neutral (*adj* M_{diff} = 31.47, *adj p* = .011), 95% CIs [6.51, 56.44], and invalid cues

(*adj* M_{diff} = 44.09, *adj p* < .001), 95% CIs [19.73, 68.44], for both groups. The DMDD group had a significantly longer response time than the control group, regardless of cue type (*adj* M_{diff} = -81.07, *adj p* = .001), 95% CIs [-121.99, -40,15]. The effect of group x cue type interaction was not significant (*F*[2,27] = 3.36, *p* = .050) during game 2.

The group x cue type interaction was significant for game 3 (frustration condition), *F*(2,27) = 4.74, *p* = .017. This means cue type affected the response time of participants in a certain group. The mean response time was significantly longer for neutral cues versus valid and invalid cues for both the control and DMDD groups. The mean response time for the DMDD group for neutral cues was significantly higher than that of the control group for both valid and invalid cues (Figure 19).

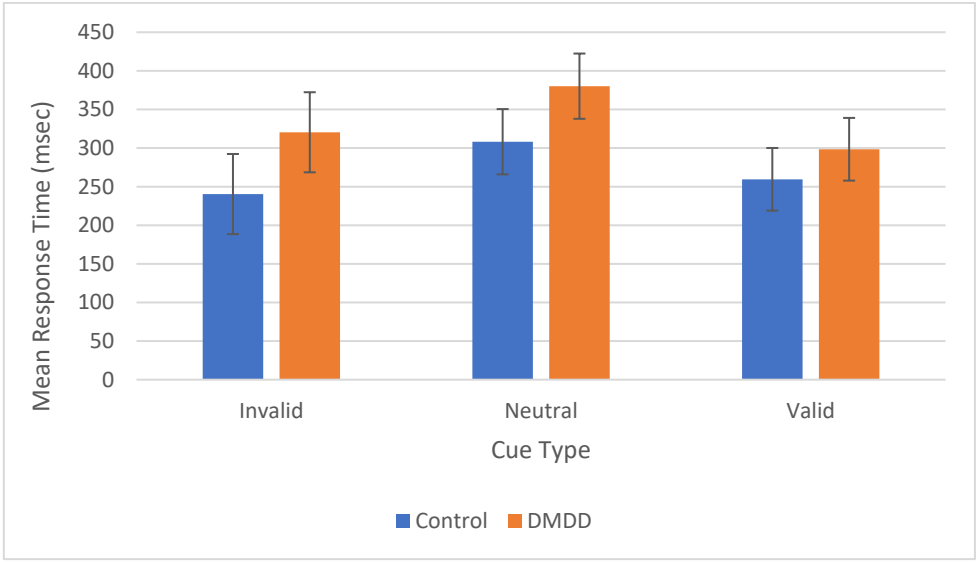

Figure 19. Group x cue type interaction during game 3, by group

The results of the post hoc analyses for the group x cue interaction during the frustration condition of the Affective Posner Cueing Task are summarised in Table 19.

In summary, these results indicate that response time was faster for valid trials than for neutral or invalid trials, regardless of the condition (i.e., baseline, emotive, or frustration) and group status, confirming the Posner effect in both groups. They also suggest, as

anticipated, that participants with DMDD took longer to shift their attention from the location of the cue to that of the target than participants in the control group during games 2 and 3.

Table 19

Post hoc Results of Group x Cue Type Interaction during the Affective Posner Cueing Task (Frustration Condition)

Group 1	Cue Type 1	Group 2	Cue Type 2	*Adj* M_{diff}	*p*	*Adj p*	Lower CI	Upper CI	Adj. Lower CI	Adj. Upper CI
Control	Invalid	Control	Neutral	-67.79	< .001	.008	-103.97	-31.61	-121.91	-13.68
Control	Invalid	Control	Valid	-19.07	.108	.569	-42.60	4.47	-54.27	16.14
Control	Invalid	DMDD	Invalid	-80.00	.036	.270	-154.36	-5.64	-191.38	31.378
Control	Invalid	DMDD	Neutral	-139.72	< .001	.003	-207.1	-72.35	-241.55	-37.90
Control	Invalid	DMDD	Valid	-57.99	.086	.500	-124.74	8.76	-158.3	42.32
Control	Neutral	Control	Valid	48.73	< .001	.011	21.91	75.55	8.61	88.84
Control	Neutral	DMDD	Invalid	-12.21	.712	.999	-79.58	55.17	-114.03	89.61
Control	Neutral	DMDD	Neutral	-71.93	.023	.187	-132.95	-10.91	-163.2	19.34
Control	Neutral	DMDD	Valid	9.80	.740	.999	-49.65	69.25	-79.78	99.38
Control	Valid	DMDD	Invalid	-60.93	.072	.446	-127.68	5.81	-161.24	39.37
Control	Valid	DMDD	Neutral	-120.66	< .001	.004	-180.11	-61.21	-210.24	-31.08
Control	Valid	DMDD	Valid	-38.92	.186	.751	-97.68	19.83	-126.78	48.93
DMDD	Invalid	DMDD	Neutral	-59.72	.002	.024	-95.90	-23.54	-113.84	-5.61
DMDD	Invalid	DMDD	Valid	22.01	.066	.415	-1.53	45.55	-13.19	57.21
DMDD	Neutral	DMDD	Valid	81.73	< .001	< .001	54.91	108.55	41.62	121.85

Note. Tukey-Kramer adjustment used; α = .05; CI = Confidence Intervals

Response Time and Game Condition. To examine the prediction that responsivity would be impaired in participants in the DMDD group compared to that of participants in the control group based on game condition, response times were compared at baseline (game 1), when contingencies were introduced (game 2) and when frustration was introduced (game 3). Overall, the effects of both group (F[1,18] = 13.62, p = .002) and game (F[2,27] = 134.12, p < .001) were significant. However, the group x game interaction was not significant (F[2,27] = 0.2, p = .820). Post hoc analyses showed that, as anticipated, the response time was significantly lower for the control group compared to the DMDD group

across all games (*adj* M_{diff} = -84.18, *adj p* = .002), 95% CIs [-132.03, -36.33]. A within-group comparison showed that the difference in response latency between game 1 and game 2 was not significant, but the differences in response times between game 1 versus game 3 and game 2 versus game 3 were significant (see Table 20).

Table 20

Post hoc Results of Response Times during Affective Posner Cueing Task game, across Both Groups

Game A	Game B	*Adj* M_{diff}	*p*	*Adj p*	Lower CI	Upper CI	Adj Lower CI	Adj Upper CI
Game 1	Game 2	-10.84	.374	.643	-35.44	13.75	-40.61	18.93
Game 1	Game 3	135.4	< .001	< .001	109.07	161.74	103.52	167.28
Game 2	Game 3	146.25	< .001	< .001	127.81	164.68	123.94	168.55

Note. Tukey-Kramer adjustment used; α = .05; CI = Confidence Intervals

To investigate the expectation that participants diagnosed with DMDD would be slower than control participants to respond to emotional and frustrating trials, compared to baseline trials, the effects of group, feedback, and group-feedback interaction on response time were determined. Main effects for both group (F[1,83] = 4.42, p = .039) and feedback type (F[2,23] = 83.77, p < .001) were found. No main effect for the group x feedback type interaction was evident (F[2,23] = 0.43, p = .660).

A between-group comparison revealed, as anticipated, that response time was significantly lower for the control group than the DMDD group across all feedback types, when controlling for the practice response time (*adj* M_{diff} = -59.55, *adj p* = .039), 95% CIs [-115.89, -3.22]. Within-group comparisons found that participants took longer to respond to positive feedback (*adj M* = 405; p < .001), 95% CIs [364.48, 444.58], than to negative feedback (*adj M* = 364; p < .001), 95% CIs [312.41, 416.17]. Frustrative feedback prompted

the fastest response time in both groups (*adj M* = 285; *p* < .001), 95% CIs [231.16, 339.33] (see Figure 20).

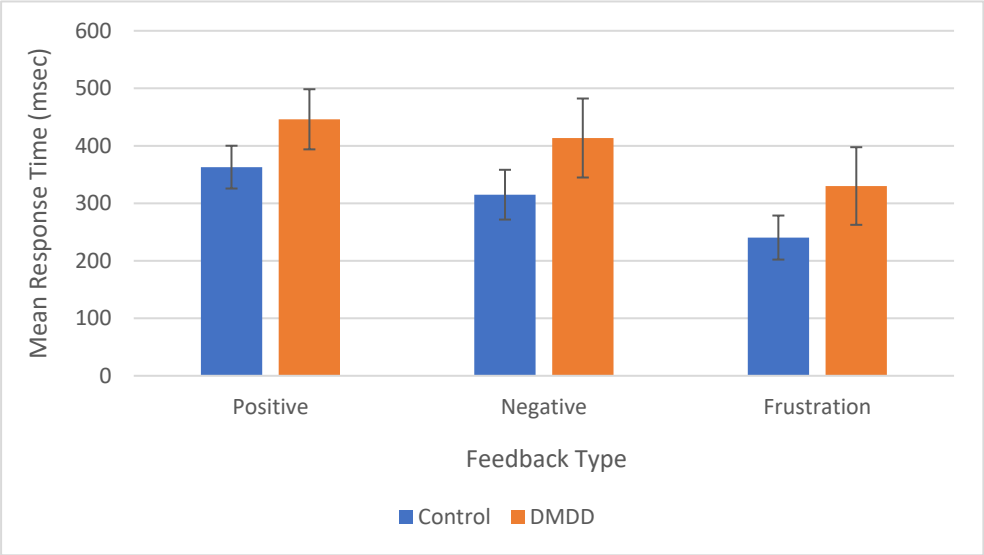

Figure 20. Effect of feedback type on average response time.

The results of the post hoc analyses for the effect of feedback on response time during the Affective Posner Cueing Task are given in Table 21.

Table 21

Post hoc Results of the Effect of Feedback on Response Time during the Affective Posner Cueing Task, across both Groups

Feedback Type 1	Feedback Type 2	*Adj* M_{diff}	*p*	*Adj p*	Lower CI	Upper CI	Adj Lower CI	Adj Upper CI
Negative	Positive	-40.24	< .001	< .001	-57.23	-23.25	-60.96	-19.51
Frustration	Negative	-79.04	< .001	< .001	-101.37	-56.72	-106.35	-51.74
Frustration	Positive	-19.28	< .001	< .001	-138.28	-100.28	-142.19	-96.37

Note. Tukey-Kramer adjustment used; α = .05; CI = Confidence Intervals

It was also hypothesised that participants in the control group would be more affected by the introduction of contingencies than participants in the DMDD group. Results showed

that both group (F[1,15] = 15.56, p = .001) and trial type (i.e., reward or no-reward; F[1,28] = 8.07, p = .008) had a significant effect on response time during game 2. The response time was significantly lower for the control group (*adj M* = 403; p < .001), 95% CIs [366, 440] compared to the DMDD group (*adj M* = 474; p < .001), 95% CIs [437, 511] across all trial types, when controlling for the practice response time scores. The main effect for contigencies indicated that both groups of participants responded faster during reward trials than non-reward trials (*adj* M_{diff} = 13.97, p = .008), 95% CIs [3.89, 24.05]. No significant effect was found for the group x trial type interaction (F[1,28] = 0.15, p = .701).

While no group (F[1,27] = 3.28, p = .081) or group x trial type effects (F [1,28] = 0, p = .988) were apparent on response time during game 3, the effect of trial type was significant. Again, response time was significantly lower for reward trials (*adj M* = 284; p < .001), 95% CIs [256, 312] compared to no-reward trials (*adj M* = 301; p = .049), 95% CIs [270, 332].

It was anticipated that emotive and frustrative feedback would have less of an impact on the response times of participants in the DMDD group than those in the control group. While the former group's response latency did not improve as much as that of the control group, these results were not statistically significant (see Table 22).

Table 22

Comparisons between Groups on Response Time Improvement following Emotive and Frustrative Feedback

Feedback Type	F(1,28)	p
Positive	0.58	.456
Negative	1.48	.234
Frustration	0.59	.449

Note. α = .05

In brief, partipants diagnosed with DMDD had, as anticipated, a longer response latency than participants in the control group during the Affective Posner Cueing Task across all three conditions. The results did not support the hypothesis that emotive and frustrative feedback would produce a significantly smaller reduction in response time in the DMDD group than in the control group.

Accuracy. Similar to the exploration of both groups' response times during the Affective Posner Cueing Task, analyses were conducted for accuracy, with percent correct as the outcome measure. A high-level analysis indicated that game condition (i.e., baseline, feedback and reward, and frustration) had a significant effect on accuracy ($F[2,27] = 57.56$, $p < .001$). Group ($F[1,24] = 0.04$, $p = .840$) and the group x game interaction ($F[1,24] = 0.04$, $p = .840$) had no significant effect on global accuracy. Post hoc tests showed that the participants were most accurate during game 2 (emotive feedback and reward) and least accurate during game 3 (frustration condition). This supports the hypothesis that frustration would compromise accuracy in both groups, however it was anticipated that the participants in the DMDD group would be significantly less accurate than the participants in the control group. This was not the case. The results are illustrated in Figure 21.

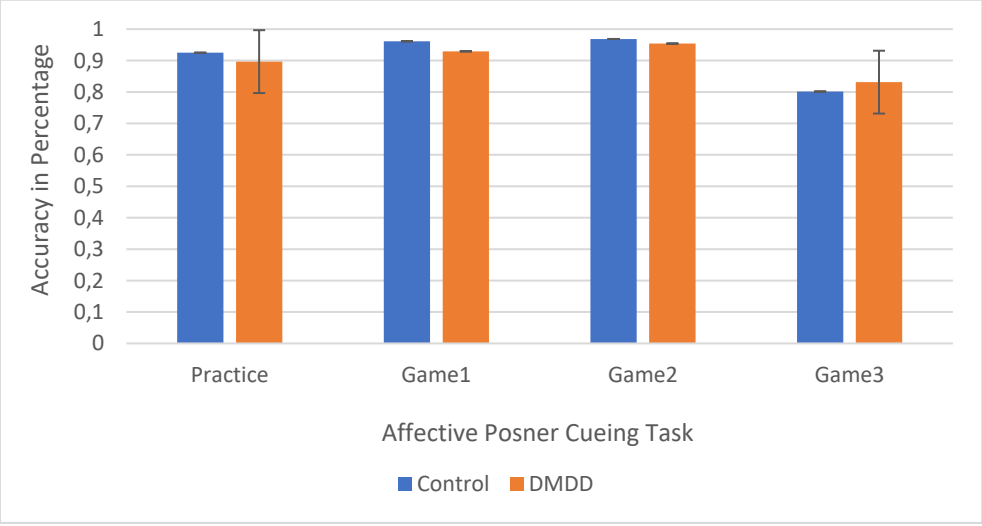

Figure 21. Mean accuracy as percent correct for both groups across all three Affective Posner Cueing Task games.

In order to evaluate whether the groups differed significantly in accuracy as a function of cue type, the accuracy outcomes of valid, invalid and neutral cues of all three Affective Posner Cueing Task games were compared across groups. Table 23 contains the least squares means accuracy percentage when groups were compared at each test point of this task.

Table 23

Least Means Squares Accuracy as a Function of Cue Type during Affective Posner Cueing Task

Cue type	Accuracy %		*p*	Lower CI	Upper CI
	Control	DMDD			
Game 1 (baseline)					
Invalid	0.85 ± 0.14	0.78 ± 0.22	< .001	0.78	0.87
Neutral	0.98 ± 0.04	0.97 ± 0.04	< .001	0.96	1.00
Valid	0.99 ± 0.02	0.97 ± 0.04	< .001	0.98	1.00
Game 2 (feedback & reward)					
Invalid	0.89 ± 0.1	0.86 ± 0.13	< .001	0.85	0.93
Neutral	0.99 ± 0.02	0.96 ± 0.05	< .001	0.97	0.99
Valid	0.99 ± 0.02	0.98 ± 0,04	< .001	0.98	0.99
Game 3 (frustration)					
Invalid	0.37 ± 0.24	0.48 ± 0.24	< .001	0.33	0.52
Neutral	0.82 ± 0.24	0.88 ± 0.13	< .001	0.80	0.90
Valid	0.94 ± 0.05	0.93 ± 0.06	< .001	0.92	0.95

Note: Values are adjusted means ± SD; α = .05; CI = Confidence Intervals

It was anticipated that participants in the DMDD group would be significantly less accurate in response to invalid and neutral cues than participants in the control group, however the post hoc results did not support this prediction.

During game 1 (baseline), neither group (F[1,32] = 1.68, p = .205) nor the group x cue type interaction (F[2,34] = 1.09, p = .349) had a significant effect on accuracy, although a main effect was found for cue type (F[2,34] = 27.76, p < .001). Both groups were significantly more accurate in response to neutral and valid cues than to invalid cues. The post hoc results are summarised in Table 24.

Table 24

Post hoc Results of Cue Type Effect on Accuracy during Affective Posner Cueing Task Baseline (Game 1)

Cue Type 1	Cue Type 2	*Adj* M_{diff}	*p*	*Adj p*	Lower CI	Upper CI	Adj Lower CI	Adj Upper CI
Valid	Neutral	-0.01	.308	.559	-0.02	0.01	-0.03	0.01
Valid	Invalid	-0.16	< .001	< .001	-0.21	-0.12	-0.22	-0.11
Neutral	Invalid	-0.15	< .001	< .001	-0.20	-0.11	-0.21	-0.10

Note. Tukey-Kramer adjustment used; α = .05; CI = Confidence Intervals

Similar results were found when affective feedback and rewards were introduced during game 2. No significant effect emerged for group (F[1,24] = 0.26, p = .618) or group x cue type interaction (F[2,26] = 2.15, p = .137). Again, cue type had a significant effect on accuracy (F[2,26] = 13.32, p = .001) for both groups, with significantly faster responses to valid and neutral cues than to invalid cues. The post hoc results follows in Table 25.

Game 3 (frustration) yielded comparable results. Cue type significantly affected accuracy (F[2,27] = 97.9, p < .001) across both groups, with post hoc analyses revealing an additional significant effect for valid versus neutral cues (see Table 26).

Table 25

Post hoc Results of Cue Type Effect on Accuracy during Affective Posner Cueing Task (Game 2)

Cue Type 1	Cue Type 2	*Adj* M_{diff}	*p*	*Adj p*	Lower CI	Upper CI	Adj Lower CI	Adj Upper CI
Valid	Neutral	-0.01	.353	.617	-0.02	0.01	-0.02	0.01
Valid	Invalid	-0.10	< .001	< .001	-0.14	-0.06	-0.15	-0.05
Neutral	Invalid	-0.09	< .001	< .001	-0.13	-0.06	-0.14	-0.05

Note. Tukey-Kramer adjustment used; α = .05; CI = Confidence Intervals

Table 26

Post hoc Results of Cue Type Effect on Accuracy during Affective Posner Cueing Task (Game 3)

Cue Type 1	Cue Type 2	*Adj* M_{diff}	*p*	*Adj p*	Lower CI	Upper CI	Adj Lower CI	Adj Upper CI
Valid	Neutral	-0.08	< .001	.002	-0.13	-0.04	-0.14	-0.03
Valid	Invalid	-0.51	< .001	< .001	-0.6	-0.42	-0.62	-0.40
Neutral	Invalid	-0.43	< .001	< .001	-0.13	-0.04	-0.14	-0.03

Note. Tukey-Kramer adjustment used; α = .05; CI = Confidence Intervals

Participants were most accurate in response to valid cues and least accurate in response to invalid cues. They were slightly less accurate in response to neutral cues than valid cues, but much more accurate compared to invalid cues. Effects for group (F[2,27] = 1.04, p = .317) and group x cue type interaction (F[2,27] = 1.27, p = .298) were not significant.

These results mean that, during all three games, both groups' mean accuracy for invalid cues was significantly lower than that for neutral and valid cues. When frustration

manipulation was introduced in game 3, participants in both groups were least accurate for invalid cues and most accurate for valid cues.

Further assumptions were that accuracy would vary in response to feedback type (i.e. positive, negative, and frustration), and that there would be a significant between-group difference in post-feedback accuracy scores, with the DMDD group being less accurate than the control group following emotive and frustrative feedback.

Controlling for baseline accuracy scores, a significant effect was found for feedback type ($F[2,26] = 29.89$, $p < .001$), but not for group ($F[1,27] = 0.75$, $p = .390$) or the group x feedback type interaction ($F[2,26] = 1$, $p < .380$). As expected, participants in both groups were least accurate following frustration feedback, more accurate following negative feedback and most accurate following positive feedback (see Figure 22).

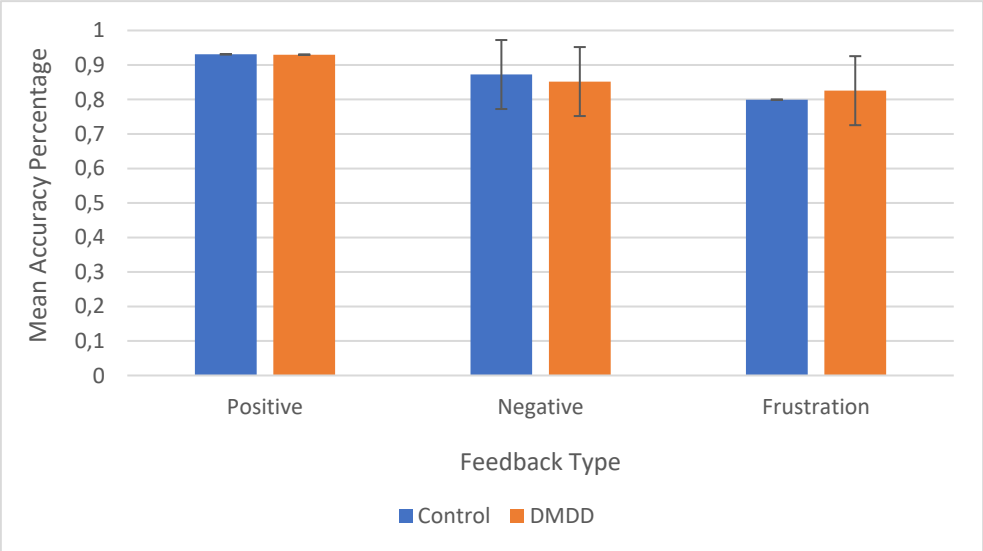

Figure 22. Effect of feedback type on mean accuracy**.**

Post hoc results revealed significant effects for all three feedback types. These results are contained in Table 27.

Table 27

Post hoc Results of Feedback Type Effect on Accuracy during the Affective Posner Cueing Task

Feedback Type 1	Feedback Type 2	*Adj* M_{diff}	*p*	*Adj p*	Lower CI	Upper CI	Adj Lower CI	Adj Upper CI
Negative	Positive	-0.07	.001	.003	-0.10	-0.03	-0.11	-0.02
Frustration	Positive	-0.12	< .001	< .001	-0.15	-0.09	-0.15	-0.08
Frustration	Negative	-0.05	.016	.043	-0.09	-0.009	-0.09	-0.001

Note. Tukey-Kramer adjustment used; α = .05; CI = Confidence Intervals

These results indicate that no significant group differences existed in terms of accuracy as a function of feedback type, which was not expected. Within groups, all three feedback types had a statistically significant effect on participants' accuracy scores.

The final behavioural outcome comparison was the effect of trial type (i.e., reward vs. non-reward) on accuracy. It was anticipated that participants in the DMDD group would be less responsive to rewards, and therefore not show a significant difference in non-reward versus reward accuracy scores, than those in the control group. However, the results did not support this supposition. During game 2, there were no significant effects for group (F[1,9] = 0.02, p = .890), trial type (F[1,27] = 3.71, p = .065) or group x trial type interaction (F[1,27] = 1.11, p = .301). The group x trial type interaction was significant for game 3 (F[1,28] = 6.89, p = .014), however post hoc analyses found no significant differences. In other words, no between-group or within-group differences were found for trial type during the Affective Posner Cueing Task.

Affective data

***Self-Assessment Manikin*.** The next element of the second research aim was to explore group differences in self-reported arousal and valence, as measured by the Self-Assessment Manikin, and after each condition (i.e. baseline, feedback and reward, and

frustration) during the Affective Posner Cueing Task. At baseline (game 1), group had a significant effect on self-reported arousal (F[1,28] = 4.84, p = .036). Post hoc analyses showed that participants in the DMDD group reported significantly more arousal than those in the control group (*adj* M_{diff} = -1.73, *adj* p = .036), 95% CIs [-3.35, -0.12] at pre-test. There were no other significant between-group differences in self-reported valence or arousal at any other test point, contrary to what was expected (see Table 28).

Table 28

Least Squares Means Results Comparing Groups on Self-reported Valence and Arousal (as measured by the SAM)

			Control	DMDD	Control	DMDD	Control	DMDD
	F	*p*	*Adj M*		Lower CI		Upper CI	
Pre-test								
Arousal	F(1,28) = 4.84	.036	5.33 ± 0.56	7.07 ± 0.56	4.19	5.93	6.47	8.21
Valence	F(1,14) = 0.18	.681	6.8 ± 0.46	7.07 ± 0.46	5.86	6.12	7.74	8.01
Game 1 (baseline)								
Arousal	F(1,26) = 4.84	.192	7.33 ± 0.48	6.36 ± 0.5	6.33	5.33	8.32	7.39
Valence	F(1,13) = 0.71	.416	7.29 ± 0.44	7.70 ± 0.43	6.37	6.81	8.2	8.59
Game 2 (feedback & reward)								
Arousal	F(1,27) = 2.43	.281	7.24 ± 0.75	6.02 ± 0.75	5.70	4.48	8.79	7.57
Valence	F(1,14) = 1.21	.142	7.62 ± 0.7	6.31 ± 0.75	6.19	4.88	9.05	7.75
Game 3 (frustration)								
Arousal	F(1,27) = 1.27	.270	5.19 ± 0.78	5.49 ± 0.78	5.19	8.37	3.90	7.08
Valence	F(1,13) = 0.21	.653	5.66 ± 0.72	5.21 ± 0.72	4.19	7.13	3.74	6.69

Note. SAM = Self-Assessment Manikin; Values are adjusted means ± SD; α = .05; CI = Confidence Intervals

Observed Frustration. The final objective of the second research aim was to determine whether the observed frustration scores, as measured by the Observed Frustration Reactivity scale and rated by the researcher during the frustration condition (game 3) of the

Affective Posner Cueing Task, were significantly correlated with the participants' ratings of their valence and arousal after game 3 on the Self-Assessment Manikin.

A Spearman's correlation was run to determine the relationship between the seven observable frustration behaviour variables (i.e., frowning, sighing, pouting, whining, fussing, swearing and shouting), as well as the global distress and affects scores, and the participants' self-reported valence and arousal scores in each group. There were five significant correlations. In the control group, there was a significant correlation (p = .004) between observed whining and self-reported arousal. In the DMDD group, the significant correlations were observed frowning and self-reported valence (p = .025), observed sighing and arousal (p = .042), observed global distress and self-reported valence (p = .017), and observed global affect and self-reported valence (p = .010; see Table 29).

Table 29

Spearman's Correlation Coefficients for Observed Frustration Reactivity and SAM Scores

Observed Frustration	Control (n =15) SAM Arousal		Control (n =15) SAM Valence		DMDD (n =15) SAM Arousal		DMDD (n =15) SAM Valence	
	r_s	p	r_s	p	r_s	p	r_s	p
Frowning	-0.079	.780	-0.349	.202	-0.341	.213	-0.574	.025
Sighing	-0.177	.529	-0.377	.165	-0.530	.042	-0.411	.128
Pouting	-0.150	.594	-0.468	.079	-0.262	.346	-0.306	.268
Whining	0.690	.004	-0.218	.434	-0.054	.848	-0.348	.203
Fussing	0.434	.106	0.135	.631	-0.258	.352	-0.346	.207
Swearing	-	-	-	-	-0.350	.201	-0.378	.165
Shouting	0.317	.250	0.348	.202	-	-	-	-
Global Distress	0.297	.282	-0.168	.551	-0.458	.086	-0.603	.017
Global Affect	0.473	.075	-0.246	.377	0.482	.069	0.640	.010

Note. SAM = Self-Assessment Manikin; N =30; Prob > |r| under H0: Rho=0

The relationship between observed whining and self-reported arousal in the control group was positive. This was expected, as more frustration behaviour would be a logical consequence of increased arousal. The correlations in the DMDD group, however, were mixed and one contradicted expectations. When observed frowning scores increased, DMDD participants reported less positively valenced feelings, which was expected. More observed sighing, in contrast, was associated with less self-reported arousal. This was not expected, as more observed frustration behaviour was anticipated to be associated with higher levels of self-reported arousal. Observed global distress was negatively correlated with self-reported valence, which means the more observed distress participants in the DMDD group displayed, the lower they rated their feelings of self-reported valence. Similarly, the more positive affect participants in this group exhibited, the higher their ratings of self-reported valence were. These findings were expected.

Dynamic Affect Recognition Evaluation Task

The third research aim was to test whether groups varied in their ability to accurately recognise emotions and their latency in doing so. It was hypothesised that participants in the DMDD group would be less accurate and slower to identify core emotions. The results are contained in Table 30.

No significant between-group differences were evident in terms of latency in identifying emotions. Post hoc analyses found that the groups differed significantly only in their ability to correctly identify fear. The mean percentage accuracy for fear was significantly higher for the control group compared to the DMDD group (*adj* M_{diff} = 0.24, *adj p* = .032), 95% CIs [0.02, 0.45]. Both groups were most accurate at recognising happiness and surprise. The DMDD group found it easier to identify disgust than fear, whereas the control group was least accurate in the recognition of fear. Participants in the control group

mostly mistook fear for surprise (38%) and then happiness (26%), while participants in the DMDD group typically misidentified fear as disgust (34%) and then happiness (29%).

Table 30

Least Squares Means Results Comparing Groups on DARE Behavioural Data for Each Emotion

			Control	DMDD	Control	DMDD	Control	DMDD
	F	*p*	*Adj M*		Lower CI		Upper CI	
Percent Accurate								
Anger	$F(1,14) = 2.64$	.1265	0.74 ± 0.06	0.62 ± 0.06	0.62	0.5	0.86	0.74
Disgust	$F(1,28) = 1.85$	.1843	0.66 ± 0.09	0.49 ± 0.09	0.47	0.3	0.84	0.67
Fear	$F(1,28) = 5.12$	.0316	0.7 ± 0.07	0.47 ± 0.07	0.55	0.31	0.86	0.62
Happiness	$F(1,14) = 0.01$	.9277	0.96 ± 0.02	0.96 ± 0.02	0.93	0.92	0.1	0.1
Sadness	$F(1,14) = 0.01$	.9152	0.9 ± 0.04	0.9 ± 0.04	0.83	0.82	0.98	0.98
Surprise	$F(1,14) = 1.56$	.2316	0.96 ± 0.02	0.93 ± 0.02	0.92	0.86	1.0	0.97
Latency								
Anger	$F(1,14) = 3.06$	.1024	13.70	15.95	11.82	14.06	15.59	17.84
Disgust	$F(1,14) = 0.05$	.823	10.78	10.99	9.41	9.62	12.14	12.35
Fear	$F(1,27) = 0.79$	.3813	12.79	13.55	11.53	12.33	14.06	14.77
Happiness	$F(1,14) = 0.4$	.5385	9.92 ± 0.63	10.44 ± 0.63	8.64	9.15	11.21	11.72
Sadness	$F(1,14) = 0.47$	.5036	11.21 ± 0.88	12.07 ± 0.88	9.41	10.26	13.03	13.88
Surprise	$F(1,28) = 1.42$	.2429	10.05 ± 0.6	11.05 ± 0.6	8.83	9.83	11.26	12.27

Note. Values are adjusted means ± SD; α = .05; CI = Confidence Intervals

Prosody Task

The fourth research goal was to compare groups in terms of their prosodic features of vocalisation and to explore the relationship between these features and vagal tone. Prosody, which is the conveyance of emotion in human voice through the modulation of intonation, is hypothesised to be linked to RSA, as the vagal system that influences heart rate is related to functioning of the cranial nerves that also control the striated muscles in the face and head. The prosody task was designed to evoke emotion through the recounting of positive and negative narratives The hypothesis was that participants in the DMDD group,

who were anticipated to have lower vagal tone, would exhibit more less prosody (i.e., less emotion, and therefore, less variability or more monotone), measured by modulation depth, bandwidth of fundamental frequency (BW50), and spectral tilt, than those in the control group.

During the positive prosody task, a main effect was found for spectral tilt (F[1,25] = 4.79, p = .044). This means that the distribution of energy between the low and the high frequencies in the DMDD group's acoustics was significantly more varied (i.e., more prosody) than that of the control group (*adj* M_{diff} = -0.0006, *adj* p = .044), 95% CIs [-0.00119, -0.00002]. No main effects of group were found for BW50 (F[1,25] = 0.42, p = .522), or modulation depth (F[1,13] = 3.37, p = .089).

A statistically significant effect of group was also found for modulation depth during the negative prosody task (F[1,25] = 5.26, p = .031). Again, the vocalisations of the participants in the DMDD group were more varied, or less smoothly modulated, than participants in the control group (*adj* M_{diff} = -0.06746, *adj* p = .029), 95% CIs [-0.13, -0.01]. The results for spectral tilt (F[1,13] = 0.75, p = .403), and BW50 (F[1,26] = 1.23, p = .277) were not significant.

There was a significant negative correlation between modulation depth and vagal tone during the positive prosody task (F[1,14] = 8.24, p = .012) in the control group. During the positive prosody task, RSA decreased by 7 units for every 1 unit increase in modulation depth. There was no evidence of a significant correlation between vagal tone and modulation depth in the DMDD group, with RSA increasing by 1.4 units for every 1 unit increase in modulation depth.

Attachment Style

The fifth research aim was to compare the groups in terms of their self-reported anxious and avoidant attachment scores, as measured by the Attachments Style Classification

Questionnaire. A significant main effect for the avoidant attachment style was found (F[1,28] = 7.07, p = .013). Post hoc analyses showed that the mean avoidant attachment style score was significantly higher for participants in the DMDD group than for participants in the control group (*adj* M_{diff} = -0.99, *adj* p = .013), 95% CIs [-1.75, -0.23], suggesting a greater presence of this type of attachment in the DMDD group.

No significant between-group difference was evident for the anxious attachment style (F[1,28] = 2.01, p = .168). However, this attachment style was significantly correlated with vagal tone at baseline (r(28) = -0.363, p = .049) in both groups. This correlation means that poorer vagal tone is associated with a more anxious attachment style in general.

Social Behaviour and Communication: Parent Questionnaire

Since the control group exhibited age-appropriate social behaviour and communication skills, this analysis was only completed for the DMDD group. The number of problems in each of the categories of the questionnaire reported by the parents was correlated with RSA and HP at pre-test (resting baseline) and during game 3 (frustration condition) of the Posner task to determine whether a significant relationship existed between the number of parent-reported social behaviour and communication skills problems and vagal tone. The number of interaction problems (i.e., using words and sentences spontaneously to communicate thoughts and ideas; talking with others; initiating interactions; interacting with peers; and reading social cues) significantly correlated with vagal tone (RSA) during the frustration condition (r(28) = -.516; p = .049). In other words, as the number of interaction problems on the Social Behaviour and Communication: Parent Questionnaire increased, vagal tone during the frustration condition decreased.

This chapter presented the statistical analyses associated with the research aims of the study. The first main finding is that the two groups were matched in terms of their demographics. Secondly, no significant group difference was found in terms of RSA, i.e.

vagal response, at any test point. The third main finding included significant between-group differences in behavioural data for the Affective Posner Cueing Task. Participants in the DMDD group were significantly slower to respond to all trials (i.e., baseline, feedback and reward, and frustration), regardless of cue type, trial type and feedback type. However, the groups did not vary significantly in accuracy. Apart from reporting significantly more arousal at baseline, groups reported similar affective ratings on the Self-Assessment Manikin after each game of the Affective Posner Cueing Task. The fourth main finding was that response latency was similar for the two groups when recognising emotions during the DARE task, but participants in the DMDD struggled more than those in the control group to correctly identify fear and often mistook it for disgust or happiness. The fifth important finding was that the two groups differed significantly on two prosodic features of vocalisation, namely spectral tilt during the positive experience recording and modulation depth during the negative experience recording. This indicates more smoothly-modulated vocalisations by participants in the control group than by those in the DMDD group. The two groups also varied in self-reported attachment style, with an avoidant attachment style being more prevalent among participants in the DMDD group. An anxious attachment style was found to correlate significantly with baseline RSA in both groups. Finally, the number of social and communication interaction problems reported by parents of children in the DMDD group was significantly correlated with RSA during the frustration condition of the Affective Posner Cueing Task. The next chapter interprets these results in further detail and locates them within the literature reviewed at the outset of this thesis.

Chapter 7: Discussion

The rationale of the current research was to expand the existing knowledge base of the newly defined *DSM-5* (APA, 2013) diagnostic category, disruptive mood dysregulation disorder (DMDD), which was created to make provision for children who are significantly emotionally dysregulated in the form of chronic irritability and frequent temper outbursts, but who do not meet the criteria of other mental health disorders. Due to the recent inclusion of DMDD, and the fact that the majority of research has been done on its predecessor, severe mood dysregulation (SMD), a similar but not identical syndrome, empirical research data on DMDD is lacking and is only now starting to emerge. Available research focused on the prevalence, comorbidity and correlates of DMDD (e.g. Althoff et al., 2016; Dougherty et al., 2014, 2016; Freeman, Youngstrom, Youngstrom, & Findling, 2016; Mayes, Waxmonsky, Calhoun, & Bixler, 2016). Neurobiological studies of DMDD focused on social and emotional information processing and emotion regulation, particularly impaired regulation of emotion and attention; misinterpretation of social, emotional, and threat stimuli; impaired context sensitivity; and reward system dysfunction (e.g. Blair, 2010; Brotman et al., 2007, 2010; Dickstein et al., 2007, 2010; Leibenluft, 2011; Rich et al., 2007, 2008, 2010; Zepf, 2016). The current study was designed to contribute to the existing body of knowledge on DMDD by exploring the potential association between heart rate variability and the externalising symptoms (i.e. frequent temper tantrums and irritable or angry moods between outbursts) of the disorder. The overarching goal was to establish whether the emotion dysregulation and compromised social behaviour associated with DMDD are a function of a compromised social engagement system, as proposed by the polyvagal theory (Porges, 1995, 2004). Innovative aspects of this study include an exploration of vagal tone, and related prosodic features of vocalisation, in the DMDD population, as well as the use of novel methods and technology in doing so (particularly in terms of prosody analyses).

The first aim of the study was to compare the DMDD and control groups in terms of respiratory sinus arrhythmia (RSA), a non-invasive measure of vagal tone, at baseline (or resting) and in response to various challenges, particularly emotion evocation. The hypothesis was that children who were diagnosed with DMDD (i.e., emotionally dysregulated) would exhibit lower baseline RSA (i.e. low vagal tone) and larger reductions in RSA (i.e. excessive vagal withdrawal) in response to emotionally evocative stimuli (Beauchaine, 2015a, 2015b). This prediction was based on previous research that had found unusually low RSA and excessive RSA reactivity to emotion evocation to be associated with a wide range of psychopathological outcomes in children and adults, with either internalising and externalising symptoms or a combination of both (e.g., Åhs, Sollers, Furmark, Fredrikson, & Thayer, 2009; Asmundson & Stein, 1994; Austin et al., 2007; Beauchaine, 2001, 2012, 2015a; Beauchaine et al., 2007; Chambers & Allen, 2002; Crowell et al., 2005; De Wied, van Boxtel, Matthys, & Meeus, 2012; Hansen, Johnsen, Thornton, Waage, & Thayer, 2007; Kemp et al., 2014; Panaite et al., 2016; Porges, 2007; Rash & Aguirre-Camacho, 2012; Rottenberg, 2007; Rottenberg et al., 2005; Sloan et al., 1994). However, this hypothesis was not confirmed by the results. While consistently lower than that of the control group, no significant differences in RSA were found between participants in the DMDD group and the control group at baseline. This is similar to empirical studies by other researchers who did not find a difference in baseline RSA in children with emotional dysregulation and behavioural problems in comparison to controls (Calkins et al., 2007; Schoorl, Van Rijn, De Wied, Van Goozen, & Swaab, 2016b; Scott & Weems, 2014). Other studies have, however, suggested an association between attenuated baseline RSA and emotional dysregulation and behavioural problems in children (e.g., Beauchaine et al., 2001, 2007; Beauchaine, Hong, & Marsh, 2008; Gordis, Feres, Olezeski, Rabkin, & Trickett, 2010; Mezzacappa et al., 1997; Pine et al., 1998). In contrast, another study found an association between externalising problems, higher

RSA and lower heart rate (i.e., higher heart period) at supine rest (Dietrich et al., 2007). One potential reason for the null finding of the current study is the low power of the study due to the small sample size (see Limitations section). Low-powered studies are more likely to produce false negatives than high-powered studies, which means a significant effect for group on RSA may well have existed, but was not detected (Sterne & Smith, 2001).

A small sample size is, however, typical in studies of this nature. For example, the first published evidence of a parasympathetic contribution to different response profiles between adult females (between the ages of 18 and 45) who have been diagnosed with borderline personality disorder (BPD) and controls had a total sample of 20 participants – 9 participants with BPD and 11 controls (Austin et al., 2007). Similarly, 12 young adults with BPD were compared to 28 healthy controls in terms of sympathetic and parasympathetic autonomic nervous system activity before, during and following a social stressor task (Weinberg, Klonsky, & Hajcak, 2009). An exploration of high-frequency heart rate variability and cortico-striatal activity in men and women with social phobia collected data from 28 participants (Åhs et al., 2009). Another study that investigated whether increases in vagal tone would be associated with favourable treatment response with non-pharmacological treatment in adult females (ages 18 to 45) who have been diagnosed with non-chronic major depression, ended with a final sample of 16 (Chambers & Allen, 2002). Panaite et al. (2016) examined RSA reactivity in 37 depressed adults to emotional films to predict symptom improvement and trajectory. Data from 30 young adults (*M* age = 19.28, *SD* = 2.05, 20 females) was used to determine whether cardiac vagal tone and frontal brain electrical asymmetry predicted biased attention to social threat (Miskovic & Schmidt, 2010).

Similar empirical studies with children had small sample sizes as well. A study that demonstrated that infants with difficulties in decreasing vagal tone during a social/attention task at 9 months of age had significantly more behavioural problems at 3 years of age

gathered data from 24 infants (Porges et al., 1996). Another study, which found an association between baseline RSA and externalising behaviour problems in young children (*M* age = 37.79 months) who were born prematurely (before 37 weeks gestation), had a sample of 28 participants (Bagner et al., 2012). Only 20 very low birth weight children participated in a longitudinal study that explored the association between neonatal physiological measures and school-age (6–9 years) outcome measures (Doussard-Roosevelt, McClenny, & Porges, 2001). Another study explored the relationships between cardiac responses (mediated via the vagus nerve) and sustained attention in a sample of 32 typically developing school-age children (Suess, Porges, & Plude, 1994). The sample for a study investigating the psychological, autonomic, and serotonergic correlates of parasuicide among adolescent girls consisted of 23 parasuicidal adolescent girls and 23 controls (Crowell et al., 2005). Studies of children with severe mood dysregulation, which had guided certain aspects of the design of the current study, had comparable sample sizes. Deveney et al. (2013), for example, compared the emotional responses, behaviour, and neural activity of 19 severely irritable children to that of 23 healthy controls during a cued-attention task completed under non-frustrating and frustrating conditions. Similarly, a preliminary study of the neural mechanisms of frustration in paediatric bipolar disorder, using the Affective Posner Cueing Task, compared magnetoencephalography data from 20 medicated adolescence to that of 20 controls (Rich et al., 2010b).

The null finding at baseline in the current study may also be a function of cardioactive medication taken by participants in the DMDD group. None of the participants in the DMDD group were medication-naïve, and while they were asked to delay medication the morning of testing, it is not inconceivable that residual medication may have affected their RSA. Stimulants (such as those prescribed for ADHD), antipsychotic and antidepressant medication, and combinations thereof, which all of the DMDD participants were prescribed

at the time of testing, may reduce RSA (Kemp et al., 2010, 2014). It is therefore possible that baseline RSA measurements may have been higher in participants with DMDD than controls, similar to findings by Dietrich et al. (2007), but that this was not evident due to the medication.

Another potential explanation for the conflicting baseline results may be the lack of standardised baseline measurements to ensure comparability across samples, experiments and laboratories (Laborde et al., 2017). While participants in the current study were asked to adhere to the standard baseline procedure, which involves sitting, with knees at a 90° angle, both feet flat on the floor, and hands on thighs (similar to the procedure recommended for blood pressure), it did not preclude mind-wandering or disruptive thoughts, environmental distractions (such as noise from corridors or adjacent classrooms, bells, lawnmowers, etc.), or non-compliance. Furthermore, time constraints within the school setting did not allow for adequate acclimatisation to the testing environment (i.e., at least five minutes in the resting position) to allow for the potential anxiety and increased attention to respiration and heart rate, which may occur when participants are told that the recording is starting, to fade out (Quintana, Alvares, & Heathers, 2016). In addition, the standard procedure requires participants to close their eyes, but as it was anticipated that this would compound any potential anxiety that children may have been feeling in the unfamiliar testing situation, they were not asked to do so. Thus, these procedural issues may have contributed to differences in findings between the current study and those which found significant differences.

These inconsistent findings suggest that a moderate baseline vagal tone, as opposed to low or high vagal tone, characterises an optimal balance of arousal and regulation that allow children to respond to, and constructively engage with, challenges in their environment (Beauchaine, 2001; Kogan et al., 2014; Miller, Kahle, & Hastings, 2017).

Although RSA results the during the Affective Posner Task session approached statistical significance (p = .058), groups did not significantly differ in RSA during, or at recovery from, the Dynamic Affective Recognition Evaluation (DARE) and the prosody tasks. These findings were unexpected, but in line with null findings in a few other studies (Beauchaine et al., 2001; Beauchaine et al., 2008; Schoorl et al., 2016b). They are, however, in contrast with many studies that did find significant differences in RSA during stress conditions in children with emotion dysregulation and behaviour problems and controls (e.g., Åhs et al., 2009; Asmundson & Stein, 1994; Beauchaine et al., 2007; Crowell et al., 2005; De Wied et al., 2012; Hansen et al., 2007; Kemp et al., 2014; Porges, 2007; Rash & Aguirre-Camacho, 2012; Rottenberg, 2007; Rottenberg et al., 2005; Sloan et al., 1994). Despite the non-significant results, the current study did, however, yield interesting observations in terms of RSA trajectory between the groups. During the Affective Posner Cueing Task, the control group exhibited no RSA decrease (i.e., vagal withdrawal) from baseline to the practice game, while the DMDD displayed a decrease in RSA, which is consistent with the hypothesised evolutionary adaptive function of vagal tone in preparedness for fight/flight responses during challenging situations. This finding may be an indication that the DMDD group detected threat (via neuroception) in their environment where no threat was present. Throughout the challenge, the control group exhibited, as expected, a decrease in RSA during games and an increase in RSA during recovery, indicative of adaptive vagal functioning. The DMDD group also showed a further decrease in RSA from the practice game to game 1, and marginally more withdrawal than the control group. In contrast with the control group, however, the DMDD group exhibited an increase in RSA from game 1 to game 2 (when rewards and emotive feedback were introduced), and no decrease in RSA from game 1 recovery to game 2, which could be interpreted as an insensitivity to rewards. However, an index of sympathetic nervous system responses to reward (such as cardiac pre-ejection period) would

be needed in order to draw a definite conclusion (see Limitations section; Beauchaine, 2009, 2015a). Both groups showed a decrease in RSA from game 2 recovery (after rewards and emotive feedback) to game 3 (frustration condition), again with the DMDD group displaying only slightly more withdrawal than the control group. Presuming that the degree of vagal influence, and not the degree of vagal withdrawal, is key to reaching the threshold for fight and flight responding, the lower RSA at baseline may put the DMDD participants closer to the threshold for fight and flight responding even when reductions in RSA were not significantly different to those of the control group. Thus, normative reductions in RSA may be more costly for children with DMDD as they act on a system that is already compromised (Beauchaine et al., 2007).

During the DARE test of facial emotion recognition, RSA remained largely unaffected in both groups, suggesting that neither group found the task emotionally evocative. The prosody task yielded similar results to that of the DARE for the control group, with RSA remaining unchanged throughout the recounting of positive and negative stories. This implies that the control group either did not experience the task as stressful (i.e., no threat detection) or that they did have a robust social engagement system that is able to effectively regulate and maintain a calm physiological state to enable such social interactions (such as the telling of positive and negative stories during the prosody task). The DMDD group, however, exhibited a steady decline in RSA from baseline to positive to negative during the prosody task, potentially indicating excessive vagal withdrawal, compared to that of the control group, in reaction to emotion evocation.

While no firm conclusions can be drawn from these results, as they were not statistically significant, they are aligned to results from research that identified a trend for children with externalising behavioural problems to exhibit either excessive RSA withdrawal in response to emotionally evocative stimuli, or no RSA withdrawal, less RSA withdrawal

(i.e., smaller reductions in RSA), or even higher tonic RSA and increased RSA, during laboratory tests, particularly when the stimulus conditions are attentionally demanding (as are the Affective Posner Cueing Task and the DARE) and not emotionally evocative (Beauchaine, 2015b; Boyce et al., 2001; Calkins et al., 2007; Dietrich et al., 2007; Graziano & Derefinko, 2013; Mezzacappa et al., 1997; Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010; Scott & Weems, 2014). Little, less or no RSA withdrawal during laboratory tasks may be the result of attentional difficulties often experienced by children with externalising behaviour problems (Beauchaine, 2001, 2015b; Rash & Aguirre-Camacho, 2012) and may explain why, during the only emotionally evocative task without attentional demands (i.e., the prosody task), the DMDD group showed increased vagal withdrawal compared to the control group, particularly during the negative prosody task.

A potential lack of vagal suppression may also point to the absence of behavioural regulation strategies that are used by typically developing children in affect-eliciting situations (Calkins, 1997). Children with DMDD may not be able to regulate their emotion and behaviour using top-down strategies (i.e. selective attention and executive functioning) as they are in an autonomic state that facilitates flight or fight behaviour.

An analysis of heart period data indicated statistically significant differences between groups during the first session, when the Affective Posner Cueing Task was completed. At baseline, during, and after each game of the Affective Posner Cueing Task, the DMDD group displayed significantly shorter heart period (i.e., faster heart rate) than the control group. During the second session, when the DARE facial emotion labelling task and the prosody task were completed, the DMDD group also had shorter heart period than the control group, but not significantly so. The control group had a significantly shorter heart period than the DMDD group during recovery from the DARE, when controlling for baseline, perhaps indicative of adaptive vagal regulation in the control group during this task. It is thought that

RSA suppression facilitates heart rate increases under challenging conditions, and so a shorter heart period in the DMDD group could indicate parasympathetic influences to the heart and a physiological state of mobilisation (i.e., failure of the newest phylogenetic vagal system). However, in the absence of evidence of significantly attenuated RSA, it is not possible to conclude that increased heart rate is not due to other influences, such as sympathetic, hormonal or homeostatic processes. Again, it is possible that medication was a confounding variable in this case.

Thus, results did not fully confirm the hypothesis that children who have been diagnosed with DMDD would exhibit lower baseline RSA and excessive reductions in RSA in response to emotionally evocative stimuli compared to healthy controls. Although not statistically significant, dissimilar trends emerged for the two groups in terms of RSA trajectories, and warrant further investigation. Also noteworthy were findings that suggest moderate vagal regulation is fundamental to emotion regulation and prosocial behaviour, as opposed to the notion that high vagal tone equates optimal emotional and social functioning.

The second research goal was to establish whether any significant between-group differences existed in behavioural (i.e., response time/latency and accuracy) data during the Affective Posner Cueing Task. As expected, participants in the DMDD group exhibited more attentional difficulties that participants in the control group, resulting in significantly slower response times regardless of cue type (i.e., valid, invalid, and neutral), reward type (i.e., reward versus no-reward) and feedback type (i.e., positive, negative, and frustrative). This is in line with a study that found behavioural impairments during the Affective Posner Cueing Task in children with severe mood dysregulation in comparison to healthy controls (Deveney et al., 2013). However, the hypothesis that rewards, emotive feedback and frustrating feedback would produce a significantly smaller reduction in response time in the DMDD group than in the control group was not supported by the findings of the current study. One

plausible explanation for this null finding is that the under-active behavioural activation system (BAS), which is mediated by the striatum and its frontal projections, is partially normalised by the administration of methylphenidate – a medication prescribed to almost half (46%) of the DMDD group.

Unexpectedly, no group differences emerged for accuracy of scores during the Affective Posner Cueing Task. These results are, however, consistent with previous null findings for children with emotion dysregulation in comparison to typical controls (Deveney et al., 2013; Rich et al., 2005; Rich et al., 2010b). A possible explanation for this finding is that, due to the nature of the Affective Posner Cueing Task, participants had a 50% chance of being correct even when responding randomly.

The last aspect of the second research aim was to test whether group differences existed in the ability to identify emotions in the facial expressions of others and the response times in doing so. No statistically significant differences emerged for response times, although the trend for the control group to increase their response times for fear and disgust compared to other emotions was noteworthy. In terms of accuracy of identifying target emotions, the only statistically significant difference was in the groups' ability to recognise fear, on which the DMDD group scored much lower than the control group. Although not statistically significant, the DMDD group also struggled more than the control group to correctly identify the other negatively valenced emotions (control: sadness, 90.48%; fear 70.48%; anger, 74.29%; disgust, 65.71%. DMDD: anger, 61.90%; disgust, 48.57%; and fear, 46.67%). Impairment in the DMDD group in face labelling tasks, such as the DARE, was anticipated, as children with severe mood regulation appear to have difficulty with correctly identifying emotions in facial expressions (Guyer et al., 2007; McClure et al., 2003, 2005; Schenkel et al., 2007).

These findings have several implications. Firstly, as mentioned in the descriptive data section (Chapter 6), emotion identification of the control group was a slightly faster than that of the DMDD group (although not statistically significant), except on fear and disgust. This suggests of a lack of self-awareness in participants in the DMDD group that more time is required to process emotional cues that may start off as ambiguous (for example, both fear and surprise share certain features of facial expression, such as wide eyes and an information-gathering appearance), an awareness which the children in the control group exhibited.

Secondly, the inability to correctly identify fear in particular suggests that children with DMDD, like children with severe mood dysregulation, have impaired activity in the areas in the brain, specifically the amygdala, that are vital for the processing of threat and fear in facial expressions. The fact that they also struggled more than the control group to accurately identify sadness, anger and disgust supports the theory that amygdala dysfunction extends to impairment in recognising various highly arousing emotions that are similar to fear (i.e., negatively valenced emotions), including anger, disgust and sadness (Adolphs, et al., 1999; Calder et al., 1996; Schmolck & Squire, 2001). Findings in the DMDD group did not, however, mirror findings from Schmolck and Squire (2001), who found that the most common error was the same for both the control and patient (with bilateral damage to the amygdala) groups. In the aforementioned study, when presented with a sad expression, the most common error by controls and patients was to describe it as disgust (on 22% of the trials for patients and 10% of the trials for controls). Fearful expressions were most commonly erroneously described as surprise (in 33% of the trials for patients and in 17% of the trials for controls). In the current study, controls mostly erroneously described fear as surprise (as in the previous study), whereas DMDD participants mostly mistook fear for disgust and not surprise. This finding was unexpected, as fear and surprise have morphological similarities (i.e., wide eyes and raised eyebrows), while disgust and anger share common facial

characteristics (i.e., narrowing of the eyes and lowered eyebrows; Aviezer et al., 2008; Ekman, 1993). However, the finding coincides with that of Surguladze et al. (2010) who found that participants with major depressive disorder, which children with DMDD are likely to be diagnosed with in early adulthood (Brotman et al., 2006), displayed greater activation in brain regions involved with disgust (i.e., the left insula, the left orbito-frontal gyrus, the left middle/inferior temporal gyrus, and the right middle/inferior temporal gyrus). As in the previous study, the most common error made by participants in the control group when presented with a sad expression, was to describe it as disgust. Participants in the DMDD group, in contrast, incorrectly described sad expressions as either surprise, fear or disgust (26.67% respectively), and then as anger (13.33%). These findings support Porges' (1995, 2001, 2004) theory that faulty neuroception (i.e., the unconscious misinterpretation of non-threatening social and environmental cues as threatening) and a compromised social engagement system contribute to the dysregulated emotion and arousal often seen in psychiatric disorders like DMDD. It is therefore conceivable that children with DMDD are in a physiological state of defence, have attentional bias to environmental cues of threat, are more likely to misinterpret facial expressions of negative emotions and therefore are more prone to exhibit defensive behaviour (e.g., aggression and tantrums).

Thus, the hypothesis that participants in the DMDD group would exhibit reduced speed and accuracy during the Affective Posner Cueing Task and the DARE emotion recognition task was partially supported. Children in the DMDD group were significantly slower to respond during all three conditions of the Affective Posner Cueing Task and had significantly impaired ability to accurately recognise fear during the DARE task, in comparison with healthy controls.

The third research aim entailed a comparison between groups on self-reported and observed feelings of valence (i.e., on a scale that ranges from feelings of displeasure,

unhappiness, annoyance, dissatisfaction, gloominess, hopelessness and boredom to feelings of pleasure, happiness, satisfaction, content, hope, and interest) and activation (i.e., an indication of the intensity of the valence, ranging from calm, relaxed, sluggish, dull, sleepy and unaroused to excited, stimulated, frenzied, jittery, wide-awake and aroused) during the frustration condition of the Affective Posner Cueing Task. The hypothesis was that children who have been diagnosed with DMDD would report more unhappiness and arousal following trials that resulted in punishment (i.e., losing points) and frustration (i.e., rigged feedback on accuracy or speed) and present more behaviours associated with frustration. The DMDD group exhibited statistically significantly more arousal than the control group at baseline (p = .036) and no statistically significant differences in valence. However, statistically significant correlations between observed frustration behaviour and self-reported data were evident. In the control group, observed whining was the only behaviour that was positively correlated with self-reported arousal (p = .004), indicating a self-awareness of increased arousal and the ability to correctly identify and describe it. In the DMDD group, observed frowning was negatively correlated with self-reported valance (p = .025), whereas observed sighing was negatively correlated with self-reported arousal (p = .042). Observed global distress (p = .017) was negatively correlated with self-reported valence, while observed global affect (p = .010) were positively correlated with self-reported valence. This means that participants in the DMDD group exhibited non-verbal expressions of frustration and reported feelings of displeasure, but under-reported or under-estimated the intensity of these feelings (i.e., arousal). These findings only partially support the hypothesis in that DMDD participants reported lower levels of valence that correlated with observed global affect and distress. The finding that they reported more feelings of pleasure when exhibiting difficulty in regulating emotions are similar to that of previous research, which suggested that boys with externalising symptoms (i.e., oppositional defiant disorder and conduct disorder) and

attention/deficit hyperactivity disorder, which is often comorbid with DMDD (Driver & Thomas, 2018; Rigling et al., 2017), did not perceive themselves as having impairments in regulating their emotions and displayed reduced awareness of external, as well as internal emotional cues (Factor, Rosen, & Reyes, 2016; Schoorl, Van Rijn, De Wied, Van Goozen, & Swaab, 2016a; Zimmermann, 2006). These results are also aligned with those of another study where male adolescents with severe behavioural problems had more difficulties in describing their feelings than did controls (Manninen et al., 2011). It is therefore a possibility that children with DMDD have reduced awareness of their emotions and, unlike the control group, are not able to adequately to appropriately describe their feelings. However, this is in contradiction to studies that found higher self-reported levels of frustration in children with severe mood dysregulation than in control subjects in response to blocked goal attainment, possibly attributable to a failure to regulate the approach system (i.e., the inability to inhibit approach responses when these responses are not successful; Deveney et al., 2013; Thomas et al., 2014). This discrepancy between self-reported levels of frustration and observed frustration in the current study could, in part, be explained by differences in social desirability levels between the two groups, with the DMDD group under-reporting feelings of frustration in order to appear more favourable than the participants in the control group (Dadds, Perrin, & Yule, 1998).

The fourth research aim, which follows directly from the proposed failure of the social engagement system, was to determine whether children with DMDD have significantly reduced acoustic modulation when compared to healthy controls. Only one statistically significant difference emerged during the negative prosody task, which was that the participants in the DMDD group appeared to have more prosodic voices relative to the control group, as indicated by significantly increased modulation depth ($p = .031$), during the recounting of the negative story. This finding is unexpected, as it was hypothesised that

children in the DMDD group would have reduced acoustic modulation relative to the control group. However, this may be an indication of greater instability in their voices (such as, quivering) or, as parents indicated in the social behaviour and communication questionnaire (discussed under the sixth research aim), the inability to perceive and adapt to social situations appropriately when the demands on social behaviour are changed or unstated (i.e., it is possible that the participants in the control group used quieter voices because the strange, indoor or more formal context demanded it, while participants in the DMDD group did not perceive such cues and failed to adapt to the new situation). A follow-up study with a bigger sample may help to separate some of these nuances.

Also unexpected, were results that indicated significant negative correlations between RSA and modulation depth in the control group during the positive (p = .012) and negative (p = .003) prosody tasks and at recovery (p = .004). Less variability in acoustic features of vocalisations (i.e., usually described as monotone) would be expected with vagal withdrawal (i.e., reduced RSA). However, studies that investigate modulation of vocalisation features in relation to autonomic state in humans are limited and typically restricted to infants (e.g., Kolacz, 2016; Kolacz et al., 2018; Shinya, Kawai, Niwa, & Myowa-Yamakoshi, 2016; Stewart et al., 2013; Unwin et al., 2017). One possible explanation for this anomaly is that, perhaps, in older children and adults, more smoothly modulated voices are associated with a calmer autonomic state and that, as with vagal tone, moderate acoustic variation, as compared to low or high variability, characterises an optimal balance of arousal and regulation that allow individuals to adaptively respond to, and constructively engage with, their environment. This appears to be an important question to explore as efforts are continued to understand the relationship between autonomic state and psychopathology.

Therefore, results for the fourth research aim suggested that vagal regulation, and associated acoustic modulation, differ between children who have been diagnosed with

DMDD and typically developing controls, particularly when associated with negative emotional stimuli. Findings also support a growing body of evidence suggesting a potential non-linear (i.e., inverted U-shaped curve) relationship between vagal tone, and by implication, acoustic modulation, and wellbeing (Kogan, Gruber, Shallcross, Ford, & Mauss, 2013).

The fifth research aim, which is directly related to the development of the social engagement system, as proposed by the polyvagal theory (Porges, 1995, 1997, 1998, 2001, 2004), was to determine whether children with DMDD and children in the control group revealed significantly different self-reported attachment styles. Children with DMDD scored significantly higher on the avoidant attachment scale than those in the control group, suggesting that avoidant attachment, and by implication, parenting style (Beauchaine et al., 2007), may be a contributing factor in the development of DMDD. This confirms evidence that dysregulation of negative emotion is prominent in children with an avoidant attachment style (Movahed Abtahi & Kerns, 2017). The hypothesis that vagal tone would be linked to both avoidant and anxious attachment styles was only partially supported. Only anxious attachment was significantly negatively correlated with vagal tone, regardless of group membership (i.e., the higher the score on the anxious attachment scale, the lower vagal regulation). Research on the relationship between attachment style and vagal regulation is inconsistent, and, again, mostly limited to infants and younger children. An association has been established between more vagal withdrawal in avoidant-attached infants compared to securely attached infants in one study (Hill-Soderlund et al., 2008) during the Strange Situation procedure (Ainsworth, Blehar, Waters, & Wall, 1978), while several other studies found that RSA increased in insecure-avoidant infants (Movahed Abtahi & Kerns, 2017; Smith, Woodhouse, Clark, & Skowron, 2016) and decreased in insecure-resistant infants (Smith et al., 2016). In contrast, no associations have been found between attachment quality

and RSA reactivity in preschool children (Stevenson-Hinde & Marshall, 1999). Adolescents who classified themselves as both avoidantly and ambivalently attached displayed more symptoms of internalising and externalising symptoms than those who classified themselves as securely attached (Muris, Meesters, & van den Berg, 2003). Currently, there is no clear pattern of association between vagal regulation and attachment style and the results may be linked to developmental stage (i.e., cognitive development improves with age, particularly the regulation of executive functions). It is possible that parasympathetic reactivity varies according to the type of stressor (e.g., social, emotionally evocative, or cognitive) and other contextual factors (e.g. attachment style of caregivers, confounding variables, etc.), as well as the age of the child (Pietromonaco & Powers, 2015). The potential link between avoidant attachment style and DMDD evident from this study warrants further investigation in a larger sample.

The final aim of the study was to explore whether the parent-reported levels of impaired social communication and behaviour skills correlated with vagal tone during the frustration condition of the Affective Posner Cueing Task, and whether these symptoms improved as a consequence of starting medication. From a polyvagal perspective (Porges, 1995, 2004), it would follow that children with a compromised social engagement system, indicated by attenuated vagal tone, would exhibit a higher number of social interaction problems than those with a functioning social engagement system. The results from this study support this hypothesis. As expected, in the DMDD group, the number of interaction problems reported by parents were significantly negatively correlated (p = .049) with vagal tone during the frustration condition. In other words, when children with DMDD perceived threat, and experienced frustration due to blocked goal attainment (Beauchaine, 2001), their vagal regulation was reduced. In children with more interaction problems, this effect was

more pronounced. However, given the small sample size, these findings need to be replicated in a larger study.

Parents indicated, at most, moderate improvement in some of the social communication and behaviour problem areas, but medication did not seem to significantly improve symptoms of DMDD. This may indicate that there is pathophysiological component of DMDD that is not being addressed by the current treatment regimes.

Findings from this, and other studies, indicate that a multi-faceted, multi-level analysis is necessary when investigating the aetiology of psychological disorders, such as DMDD. Nonetheless, the findings have shed some light on the potential contributions of the parasympathetic nervous system (in particular, the vagus nerve) to the symptoms of DMDD and identified some observations and limitations that suggest several directions for future research.

Limitations of the Study and Directions for Future Research

Several limitations of the present study should be acknowledged and suggestions are given for how these could be improved on in future studies.

In light of the small sample size, which is not uncommon in studies that apply to niche sections of the population (as discussed under the first research aim above, e.g., Åhs et al., 2009; Austin et al., 2007; Bagner et al., 2012; Beauchaine, 2002; Beauchaine et al., 2001; Chambers & Allen, 2002; Crowell et al., 2005; Degangi, Dipietro, Greenspan, & Porges, 1991; Doussard-Roosevelt et al., 2001; Miskovic & Schmidt, 2010; Panaite et al., 2016; Porges et al., 1996; Suess et al., 1994; Rich et al., 2010b; Weinberg et al., 2009), the findings of the current study should be interpreted with caution and replication with a larger sample is recommended.

From previous research (e.g., Austin et al., 2007; Beauchaine et al., 2007; Bernston et al., 1993; Bernston et al., 1994; Friedman & Thayer, 1998) and data from the current study, it

is also evident that heightened physiological preparedness for defence behaviours have both a parasympathetic and a sympathetic component, but only the parasympathetic component was quantified during this study due the adoption of the polyvagal perspective (Porges, 1995), which focuses on parasympathetic cardiac influences. Future studies that include measures of both components may be able to elucidate the contribution of each to the symptoms of DMDD.

Reliance on self-report measures, which lend themselves to a number of biases (e.g., social desirability bias) is another limitation of this study, particularly in a sample that may not be able to accurately identify and describe their feelings (Rosenman, Tennekoon, & Hill, 2011). In some instances, however, the lack of appropriate and valid psychometric measures necessitates the use of self-report measures. For example, there is no gold-standard measure for attachment style in middle childhood and adolescence (Jewell et al., 2019).

A number of ethical and logistical considerations precluded the control of certain confounding variables that influence heart rate variance, as well as the exclusion of participants based on these variables (Laborde et al, 2017). For example, it was not ethical to wean participants off their medication (classes of cardioactive medication used by participants in this study included antidepressants, antipsychotics, and stimulants, and combinations of these) prior to testing, nor was it possible to control their diets (e.g., caffeine and sugar may affect heart rate variance), sleeping, eating and physical activity patterns before measuring their heart rates. Future studies may benefit from using the demographic questionnaire supplied by Laborde et al. (2017) in a larger sample in order to control for these variables.

Recently, there has been a shift in focus in psychophysiological research with emphasis on the microbiome-gut-brain axis and the role that the bidirectional communication between the intestine and the brain (to which the vagus nerve is central) plays in the

regulation of both metabolism and behaviour (Bonaz, Bazin, & Pellissier, 2018; Groen, de Clercq, Nieuwdorp, Hoenders, & Groen, 2018; Kolacz, Kovacic, & Porges, 2019). Growing evidence suggests that intestinal permeability (which is highly influenced by psychological stress), and the resultant immune activation, has a pertinent role in the pathophysiology of many psychiatric disorders, such as major depression, schizophrenia, bipolar disorder, alcoholism and autism (Rudzki & Szulc, 2018). An investigation into whether gut microbiome and immune activation contribute to the development DMDD, and by the same token, may offer new therapeutic options.

Conclusion

The results of the present study partially support the polyvagal theory (Porges, 1995, 1997, 1998, 2001) in relation to children with DMDD, as they suggest that these children do not exhibit the level of vagal regulation that is fundamental to appropriate and adaptive emotion regulation and social functioning, particularly in response to emotionally evocative stimuli. The symptoms of DMDD can therefore be interpreted as emergent properties of a compromised social engagement system, as proposed by the polyvagal theory (Porges, 2004).

The current study also provided a clearer picture of children with DMDD in relation to typically developing controls. The former children seem to have faulty neuroception (i.e., they misinterpret non-threatening cues as threatening) and attentional bias to threat in their environment, as suggested by divergent RSA trajectories in response to emotionally evocative stimuli. Children with DMDD also appear to have significantly poorer recognition of negative emotions in facial expressions and largely avoidant attachment styles. Finally, attenuated vagal tone was related to the number of interaction problems reported by their parents. Collectively, these findings contribute to the existing knowledge base regarding this new diagnostic category.

References

Abela, J. R., Brozina, K., & Haigh, E. P. (2002). An examination of the response styles theory of depression in third-and seventh-grade children: A short-term longitudinal study. *Journal of Abnormal Child Psychology*, *30*(5), 515-527. doi: 10.1023/A:1019873015594

Abell, B. (2012, August). *Actiwave Cardio: The Feasibility and Validation of an Innovative New Ambulatory Monitoring Device* (Paper presentation). Australian Cardiovascular Health and Rehabilitation Association. Queensland, Australia

Adam, D. (2013). On the spectrum. *Nature*, 496(7446), 416-418. doi:10.1038/496416a

Adolphs, R. (2010). What does the amygdala contribute to social cognition? *Annals of the New York Academy of Sciences*, *1191*(1), 42–61. doi: 10.1111/j.1749-6632.2010.05445.x

Adolphs, R., Russell, J. A., & Tranel, D. (1999). A role for the human amygdala in recognizing emotional arousal from unpleasant stimuli. *Psychological Science*, *10*(2), 167-171. doi: 10.1111/1467-9280.00126

Adolphs, R., Tranel, D., & Damasio, A. R. (1998). The human amygdala in social judgment. *Nature*, *393*(6684), 470-474. doi: 10.1038/30982

Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. (1994). Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature*, *372*(6507), 669. doi: 10.1038/372669a0

Åhs, F., Sollers, J. J. III, Furmark, T., Fredrikson, M. & Thayer, J. F. (2009). High-frequency heart rate variability and cortico-striatal activity in men and women with social phobia. *NeuroImage*, *47*, 815–820. doi:10.1016/j.neuroimage.2009.05.091

Ainsworth, M. D. S. (1978). Toward a general theory of infantile attachment: A comparative review of aspects of the social bond. *Behavioral and Brain Sciences*, *1*, 436.

Ainsworth, M. D. S., & Bell, S. M. (1970). Attachment, exploration, and separation: Illustrated by the behavior of one-year-olds in a strange situation. *Child Development*, 49-67. doi: 10.2307/1127388

Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. N. (1978). *Patterns of Attachment: A Psychological Study of the Strange Situation*. Hillsdale, NJ: Erlbaum.

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review*, 30(2), 217-237. doi: 10.1016/j.cpr.2009.11.004

Allgood, R., & Heaton, P. (2015). Developmental change and cross-domain links in vocal and musical emotion recognition performance in childhood. *British Journal of Developmental Psychology*, 33, 398–403. doi:10.1111/bjdp.2015.33.issue-3

Althoff, R. R., Crehan, E. T., He, J. P., Burstein, M., Hudziak, J. J., & Merikangas, K. R. (2016). Disruptive mood dysregulation disorder at ages 13–18: results from the national comorbidity survey—adolescent supplement. *Journal of Child and Adolescent Psychopharmacology*, *26*(2), 107-113. doi: 10.1089/cap.2015.0038

American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.). Washington, DC: Author. doi:10.1176/appi.books.9780890423349

American Psychiatric Association (APA; 2013a). Depressive Disorders. In *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Washington, DC: Author. doi: 10.1176/appi.books.9781585625031.rh11

American Psychological Association (APA; 2013b). *Disruptive Mood Dysregulation Disorder Fact sheet*. Retrieved from http://www.dsm5.org/Documents/Disruptive%20Mood%20Dysregulation%20Disorder%20Fact%20Sheet.pdf

Ametti, M. R. (2019). *Parsing Heterogenity In Non-Episodic, Pediatric Irritability: A Transdiagnostic, Research Domain Criteria Informed Approach*. Retrieved from https://scholarworks.uvm.edu/graddis/1083/

Anderson, A. K., Christoff, K., Panitz, D., De Rosa, E., & Gabrieli, J. D. (2003). Neural correlates of the automatic processing of threat facial signals. *Journal of Neuroscience*, *23*(13), 5627-5633. Retrieved from http://www.jneurosci.org/content/jneuro/23/13/5627.full.pdf

Anderson, A. K., & Phelps, E. A. (2000a). Perceiving emotion: There's more than meets the. *Current Biology*, *10*(15), R551-R554. doi: 10.1016/S0960-9822(00)00612-6

Anderson, A. K., & Phelps, E. A. (2000). Expression without recognition: contributions of the human amygdala to emotional communication. *Psychological Science*, *11*(2), 106-111.

Appelhans, B. M., & Luecken, L. J. (2006). Heart rate variability as an index of regulated emotional responding. *Review* eye *of General Psychology*, 10(3), 229-240. doi:10.1037/1089-2680.10.3.229

Arnold, M. B. (1960). *Emotion and personality. Vol. I. Psychological aspects*.

Asmundson, G. J. & Stein, M. B. (1994). Vagal attenuation in panic disorder: An assessment of parasympathetic nervous system function and subjective reactivity to respiratory manipulations. *Psychosomatic Medicine*, *56*, 187–193. doi:10.1097/00006842-199405000-00002

Austin, M. A., Riniolo, T. C., & Porges, S. W. (2007). Borderline personality disorder and emotion regulation: Insights from the Polyvagal Theory. *Brain and Cognition*, *65*(1), 69-76. doi: 10.1016/j.bandc.2006.05.007

Averill, J. R. (1980). A constructivist view of emotion. In *Theories of Emotion*, 305-339. Academic Press. doi: 10.1016/B978-0-12-558701-3.50018-1

Aviezer, H., Hassin, R. R., Ryan, J., Grady, C., Susskind, J., Anderson, A., ... & Bentin, S. (2008). Angry, disgusted, or afraid? Studies on the malleability of emotion perception. *Psychological Science*, *19*(7), 724-732. doi: 10.1111/j.1467-9280.2008.02148.x

Ax, A. F. (1953). The physiological differentiation between fear and anger in humans. *Psychosomatic Medicine*, *15*(5), 433-442. doi: 10.1097/00006842-195309000-00007

Axelson, D. (2013). Taking Disruptive Mood Dysregulation Disorder Out for a Test Drive. *American Journal of Psychiatry*, 170(2), 136–139. doi: 10.1176/appi.ajp.2012.12111434

Axelson, D., Findling, R. L., Fristad, M. A., Kowatch, R. A., Youngstrom, E. A., Horwitz, S. M., ... & Gill, M. K. (2012). Examining the proposed disruptive mood dysregulation disorder diagnosis in children in the Longitudinal Assessment of Manic Symptoms study. *The Journal of Clinical Psychiatry*, *73*(10), 1342. doi: 10.4088/JCP.12m07674

Bagner, D. M., Graziano, P. A., Jaccard, J., Sheinkopf, S. J., Vohr, B. R., & Lester, B. M. (2012). An initial investigation of baseline respiratory sinus arrhythmia as a moderator of treatment outcome for young children born premature with externalizing behavior problems. *Behavior Therapy*, *43*(3), 652-665. doi: 10.1016/j.beth.2011.12.002

Bal, E., Harden, E., Lamb, D., Van Hecke, A. V., Denver, J. W., & Porges, S. W. (2010). Emotion recognition in children with autism spectrum disorders: Relations to eye gaze and autonomic state. *Journal of Autism and Developmental Disorders*, *40*(3), 358-370. doi: 10.1007/s10803-009-0884-3

Ball, T., Derix, J., Wentlandt, J., Wieckhorst, B., Speck, O., Schulze-Bonhage, A., & Mutschler, I. (2009). Anatomical specificity of functional amygdala imaging of responses to stimuli with positive and negative emotional valence. *Journal of Neuroscience Methods*, 180(1), 57-70. doi: 10.1016/j.jneumeth.2009.02.022

Banks, S. J., Eddy, K. T., Angstadt, M., Nathan, P. J., & Phan, K. L. (2007). Amygdala–frontal connectivity during emotion regulation. *Social Cognitive and Affective Neuroscience*, 2(4), 303-312. doi: 10.1093/scan/nsm029

Barbas-Henry, H. A., & Lohman, A. H. (1984). The motor nuclei and primary projections of the IXth, Xth, XIth, and XIIth cranial nerves in the monitor lizard, Varanus exanthematicus. *Journal of Comparative Neurology*, *226*(4), 565-579. doi: 10.1002/cne.902260409

Barrett, L. F. (2009). The future of psychology: Connecting mind to brain. *Perspectives on Psychological Science*, *4*(4), 326-339. doi: 10.1111/j.1745-6924.2009.01134.x

Barrett, L. F. (2016). Navigating the science of emotion. In H. Meiselman (Ed.), *Emotion Measurement* (pp. 31–63). Cambridge, UK: Woodhead Publishing. doi: 10.1016/B978-0-08-100508-8.00002-3

Barrett, L. F. (2017a). Functionalism cannot save the classical view of emotion. *Social Cognitive and Affective Neuroscience*, *12*, 34–36. doi: 10.1093/scan/nsw156

Barrett, L. F. (2017b). *How emotions are made: The secret life of the brain*. Houghton Mifflin Harcourt.

Barret, K.C., & Campos, J.J. (1987) Perspectives on emotional development: II. A functionalist approach to emotions. In J.D. Osofsky (Ed.), *Handbook of Infant Development* (2nd ed., pp. 555-578). New York: Wiley.

Bayle, D. J., Henaff, M. A., & Krolak-Salmon, P. (2009). Unconsciously perceived fear in peripheral vision alerts the limbic system: a MEG study. *PLoS One*, *4*(12), e8207. doi: 10.1371/journal.pone.0008207

Bazhenova, O. V., & Porges, S. W. (2000). *Listening Project Parent Questionnaire*.

Bear, M. F., Connors, B. W., & Paradiso, M. A. (Eds.). (2007). *Neuroscience* (Vol. 2). Lippincott Williams & Wilkins.

Beauchaine, T.P. (2001). Vagal tone, development, and Gray's motivational theory: toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology*, 13(2), 183-214. doi: 10.1017/S0954579401002012

Beauchaine, T. P. (2009). Some difficulties in interpreting psychophysiological research with children. *Monographs of the Society for Research in Child Development, 74*, 80–88. doi:10.1111/j.1540-5834.2009.00509.x

Beauchaine, T. P. (2015a). Future directions in emotion dysregulation and youth psychopathology. *Journal of Clinical Child and Adolescent Psychology*, 44, 875-896. doi:10.1080/15374416.2015.1038827.

Beauchaine, T. P. (2015b). Respiratory sinus arrhythmia: A transdiagnostic biomarker of emotion dysregulation and psychopathology. *Current Opinion in Psychology*, 3, 43-47. doi:10.1016/j.copsyc.2015.01.017

Beauchaine, T. P., & Bell, Z. E. (2020). Respiratory sinus arrhythmia as a transdiagnostic biomarker of emotion dysregulation. *The Oxford Handbook of Emotion Dysregulation*, 153. doi: 10.1093/oxfordhb/9780190689285.013.12

Beauchaine, T. P., Bell, Z., Knapton, E., McDonough-Caplan, H., Shader, T., & Zisner, A. (2019). Respiratory sinus arrhythmia reactivity across empirically based structural dimensions of psychopathology: A meta-analysis. *Psychophysiology*, *56*(5). doi: 10.1111/psyp.13329

Beauchaine, T. P., & Haines, N. (2019). Functionalist and constructionist perspectives on emotion dysregulation. In T. P. Beauchaine & S. E. Crowell (Eds.), *The Oxford Handbook of Emotion Dysregulation*. New York, NY: Oxford University Press. epublished ahead of print. doi: 10.1093/oxfordhb/9780190689285.013.1

Beauchaine, T. P., Gatzke-Kopp, L., & Mead, H. K. (2007). Polyvagal Theory and Developmental Psychopathology: Emotion Dysregulation and Conduct Problems from Preschool to Adolescence. *Biological Psychology*, 74(2), 174–184. doi:10.1016/j.biopsycho.2005.08.008

Beauchaine, T. P., Hong, J., & Marsh, P. (2008). Sex differences in autonomic correlates of conduct problems and aggression. *Journal of the American Academy of Child & Adolescent Psychiatry*, *47*(7), 788-796. doi:

Beauchaine, T. P., & Thayer, J. F. (2015). Heart rate variability as a transdiagnostic biomarker of psychopathology. *International Journal of Psychophysiology*, 98, 338-350. doi:10.1016/j.ijpsycho.2015.08.004

Beauchaine, T. P., Zisner, A. R., & Sauder, C. L. (2017). Trait impulsivity and the externalizing spectrum. *Annual Review of Clinical Psychology*, *13*, 343-368. doi; 10.1146/annurev-clinpsy-021815-093253

Berntson, G. G., Cacioppo, J. T., Binkley, P. F., Uchino, B. N., Quigley, K. S., & Fieldstone, A. (1994). Autonomic cardiac control. III. Psychological stress and cardiac response in autonomic space as revealed by pharmacological blockades. *Psychophysiology*, *31*(6), 599-608. doi: 10.1111/j.1469-8986.1994.tb02352.x

Bernston, G.G., Cacioppo, J.T. and Quigley, K.S. (1993), Respiratory sinus arrhythmia: Autonomic origins, physiological mechanisms, and psychophysiological implications. *Psychophysiology*, 30, 183–196. doi: 10.1111/j.1469-8986.1993.tb01731.x

Biederman, J., Mick, E., Faraone, S., Spencer, T., Wilens, T., & Wozniak, J. (2000). Pediatric mania: A developmental subtype of bipolar disorder? *Acta Neuropsychiatrica*, *12*(3), 131-131. doi:10.1017/S0924270800035596

Blader, J. C., & Carlson, G. A. (2007). Increased rates of bipolar disorder diagnoses among US child, adolescent, and adult inpatients, 1996–2004. *Biological Psychiatry*, 62(2), 107-114. doi:10.1016/j.biopsych.2006.11.006

Blair, R. J. R. (2010). Psychopathy, frustration, and reactive aggression: the role of ventromedial prefrontal cortex. *British Journal of Psychology*, 101(3), 383-399. doi: 10.1348/000712609X418480

Blandon, A. Y., Calkins, S. D., Grimm, K. J., Keane, S. P., & O'Brien, M. (2010). Testing a developmental cascade model of emotional and social competence and early peer acceptance. *Development and Psychopathology*, 22(04), 737-748. doi: 10.1017/S0954579410000428

Bonaz, B., Bazin, T., & Pellissier, S. (2018). The vagus nerve at the interface of the microbiota-gut-brain axis. *Frontiers in Neuroscience*, *12*, 49. doi: 10.3389/fnins.2018.00049

Bornstein, M. H., Arterberry, M. E., & Lamb, M. E. (2013). *Development in Infancy: A Contemporary Introduction*. Taylor & Francis.

Bowlby, J. (1958). The nature of the child's tie to his mother. *International Journal of Psycho-Analysis*, *39*, 350-373. doi: 10.4324/9780429475931-15

Bowlby, J. (1969). Attachment and loss. *Vol. I: Attachment.* New York: Basic.

Boyce, W. T., Quas, J., Alkon, A., Smider, N. A., Essex, M. J., & Kupfer, D. J. (2001). Autonomic reactivity and psychopathology in middle childhood. *The British Journal of Psychiatry*, *179*(2), 144-150. doi: 10.1192/bjp.179.2.144

Bozikas, V. P., Kosmidis, M. H., Tonia, T., Andreou, C., Focas, K., & Karavatos, A. (2007). Impaired perception of affective prosody in remitted patients with bipolar disorder. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *19*(4), 436-440. doi: 10.1176/appi.neuropsych.19.4.436

Bradley, M. M., & Lang, P. J. (1994). Measuring emotion: The Self-Assessment Manikin and the Semantic Differential. *Journal of Behavior Therapy and Experimental Psychiatry*, *25*(1), 49-59. doi: 10.1016/0005-7916(94)90063-9

Breiter, H. C., Etcoff, N. L., Whalen, P. J., Kennedy, W. A., Rauch, S. L., Buckner, R. L., ... & Rosen, B. R. (1996). Response and habituation of the human amygdala during visual processing of facial expression. *Neuron*, *17*(5), 875-887. doi: 10.1016/S0896-6273(00)80219-6

Bretherton, I. (1985). Attachment Theory: Retrospect and Prospect. *Monographs of the Society for Research in Child Development*, *50*(1/2), 3–35. doi: 0.2307/3333824

Bridges, L. J., Denham, S. A., & Ganiban, J. M. (2004). Definitional Issues in Emotion Regulation Research. *Child Development*, *75*(2), 340-345. doi: 10.1111/j.1467-8624.2004.00675.x

Brodal, P. (2010). *The Central Nervous System: Structure and Function* (4th ed.). Oxford University Press.

Broks, P., Young, A. W., Maratos, E. J., Coffey, P. J., Calder, A. J., Isaac, C. L., ... & Roberts, N. (1998). Face processing impairments after encephalitis: amygdala damage and recognition of fear. *Neuropsychologia*, *36*(1), 59-70. doi: 10.1016/S0028-3932(97)00105-X

Brotman, M. A., Kassem, L., Reising, M. M., Guyer, A. E., Dickstein, D. P., Rich, B. A., ... & Leibenluft, E. (2007). Parental diagnoses in youth with narrow phenotype bipolar disorder or severe mood dysregulation. *The American Journal of Psychiatry*, 164(8), 1238-1241. doi: 10.1176/appi.ajp.2007.06101619

Brotman, M. A., Rich, B. A., Guyer, A. E., Lunsford, J. R., Horsey, S. E., Reising, M. M., ... & Leibenluft, E. (2010). Amygdala activation during emotion processing of neutral faces in children with severe mood dysregulation versus ADHD or bipolar disorder. *The American Journal of Psychiatry*, 167(1), 61-69. doi: 10.1176/appi.ajp.2009.09010043

Brotman, M. A., Schmajuk, M., Rich, B. A., Dickstein, D. P., Guyer, A. E., Costello, E. J., ... & Leibenluft, E. (2006). Prevalence, clinical correlates, and longitudinal course of severe mood dysregulation in children. *Biological Psychiatry*, *60*(9), 991-997. doi:10.1016/j.biopsych.2006.08.042

Brotman, M. A., Skup, M., Rich, B. A., Blair, K. S., Pine, D. S., Blair, J. R., & Leibenluft, E. (2008). Risk for bipolar disorder is associated with face-processing deficits across emotions. *Journal of the American Academy of Child & Adolescent Psychiatry*, *47*(12), 1455-1461. doi: 10.1097/CHI.0b013e318188832e

Bufferd, S. J., Dougherty, L. R., Carlson, G. A., Rose, S., & Klein, D. N. (2012). Psychiatric disorders in preschoolers: continuity from ages 3 to 6. *American Journal of Psychiatry*, *169*(11), 1157-1164. doi: 10.1176/appi.ajp.2012.12020268.

Cacioppo, J. T., & Berntson, G. G. (1994). Relationship between attitudes and evaluative space: A critical review, with emphasis on the separability of positive and negative substrates. *Psychological Bulletin, 115*(3), 401. doi: 10.1037//0033-2909.115.3.401

Cahill, L., Haier, R. J., Fallon, J., Alkire, M. T., Tang, C., Keator, D., ... & Mcgaugh, J. L. (1996). Amygdala activity at encoding correlated with long-term, free recall of emotional information. *Proceedings of the National Academy of Sciences*, *93*(15), 8016-8021. Retrieved from http://www.jstor.org/stable/40155

Calder, A. J., Young, A. W., Rowland, D., Perrett, D. I., Hodges, J. R., & Etcoff, N. L. (1996). Facial emotion recognition after bilateral amygdala damage: Differentially severe impairment of fear. *Cognitive Neuropsychology*. doi: 10.1080/026432996381890

Calkins, S. D. (1997). Cardiac vagal tone indices of temperamental reactivity and behavioral regulation in young children. *Developmental Psychobiology: The Journal of the International Society for Developmental Psychobiology*, *31*(2), 125-135. doi: 10.1002/(SICI)1098-2302(199709)31:2<125::AID-DEV5>3.0.CO;2-M

Calkins, S., & Dedmon, S. E. (2000). Physiological and behavioral regulation in two-year-old children with aggressive/destructive behavior problems. *Journal of Abnormal Child Psychology*, 28(2), 103–118. doi: 10.1023/A:1005112912906

Calkins, S. D., Gill, K. L., Johnson, M. C., & Smith, C. L. (1999). Emotional reactivity and emotional regulation strategies as predictors of social behavior with peers during

toddlerhood. *Social Development*, *8*(3), 310-334. doi: 10.1111/1467-9507.00098

Calkins, S. D., Graziano, P. A., & Keane, S. P. (2007). Cardiac vagal regulation differentiates among children at risk for behavior problems. *Biological Psychology*, *74*(2), 144-153. doi: 10.1016/j.biopsycho.2006.09.005

Calkins, S. D., & Hill, A. (2007). Caregiver Influences on Emerging Emotion Regulation: Biological and Environmental Transactions in Early Development. In J. J. Gross (Ed.), *Handbook of Emotion Regulation* (p. 229–248). The Guilford Press.

Calkins, S.D. and Keane, S.P. (2004), Cardiac vagal regulation across the preschool period: Stability, continuity, and implications for childhood adjustment. *Developmental Psychobiology*, 45, 101–112. doi: 10.1002/dev.20020

Campbell, D. T., & Stanley, J. C. (2015). *Experimental and Quasi-Experimental Designs for Research*. Ravenio Books.

Campos, J. J., Campos, R. G., & Barrett, K. C. (1989). Emergent themes in the study of emotional development and emotion regulation. *Developmental Psychology*, *25*(3), 394. doi: 10.1037/0012-1649.25.3.394

Campos, J. J., Frankel, C. B., & Camras, L. (2004). On the nature of emotion regulation. *Child Development*, *75*(2), 377-394. doi: 10.1111/j.1467-8624.2004.00681.x

Cannon, W. B. (1927). The James-Lange theory of emotions: A critical examination and an alternative theory. *The American Journal of Psychology*, 39(1/4), 106-124. doi: 10.2307/1415404

Carlson, G. A., Potegal, M., Margulies, D., Basile, J., & Gutkovich, Z. (2010). Liquid risperidone in the treatment of rages in psychiatrically hospitalized children with possible bipolar disorder. *Bipolar Disorders, 12*(2), 205-212. doi: 10.1111/j.1399-5618.2010.00793.x

Carstensen, L. L., Fung, H. H., & Charles, S. T. (2003). Socioemotional selectivity theory and the regulation of emotion in the second half of life. *Motivation and Emotion*, 27(2), 103-123. doi: 10.1023/A:102456980

Carter, C. S. (1998). Neuroendocrine perspectives on social attachment and love. *Psychoneuroendocrinology*, *23*(8), 779-818. doi: 10.1016/S0306-4530(98)00055-9

Carver, C. S. (2001). Affect and the functional bases of behavior: On the dimensional structure of affective experience. *Personality and Social Psychology Review*, *5*(4), 345-356. doi: 10.1207/S15327957PSPR0504_4

Carver, C. S. (2004). Negative affects deriving from the behavioral approach system. *Emotion*, *4*(1), 3. doi: 10.1037/1528-3542.4.1.3

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS Scales. *Journal of Personality and Social Psychology, 67*(2), 319-333. doi: 10.1037/0022-3514.67.2.319

Cassidy, J. (1994). Emotion regulation: Influences of attachment relationships. *Monographs of the Society for Research in Child Development*, *59*(2-3), 228-249. doi: 10.1111/j.1540-5834.1994.tb01287.x

Chamberlain, S. R., Müller, U., Blackwell, A. D., Clark, L., Robbins, T. W., & Sahakian, B. J. (2006). Neurochemical modulation of response inhibition and probabilistic learning in humans. *Science*, *311*(5762), 861-863. doi: 10.1126/science.1121218

Chambers, A. S., & Allen, J. J. (2002). Vagal tone as an indicator of treatment response in major depression. *Psychophysiology*, *39*(6), 861-864. doi: 10.1017/S0048577202010442

Chang, L., Schwartz, D., Dodge, K. A., & McBride-Chang, C. (2003). Harsh parenting in relation to child emotion regulation and aggression. *Journal of Family Psychology*, 17(4), 598. doi: 10.1037/0893-3200.17.4.598

Chapleau, M. W., & Sabharwal, R. (2011). Methods of assessing vagus nerve activity and reflexes. *Heart Failure Reviews*, *16*(2), 109-127. doi: 10.1007/s10741-010-9174-6

Chou, C. Y., La Marca, R., Steptoe, A., & Brewin, C. R. (2014). Biological responses to trauma and the development of intrusive memories: An analog study with the trauma film paradigm. *Biological Psychology, 103*, 135-143. doi: 10.1016/j.biopsycho.2014.08.002

Chronaki, G., Hadwin, J. A., Garner, M., Maurage, P., & Sonuga-Barke, E. J. S. (2014). The development of emotion recognition from facial expressions and non-linguistic vocalizations during childhood. *British Journal of Developmental Psychology*, 33, 218–236. doi:10.1111/bjdp.12075

Cohn, J. F., Zlochower, A. J., Lien, J., & Kanade, T. (1999). Automated face analysis by feature point tracking has high concurrent validity with manual FACS coding. *Psychophysiology, 36*(1), 35-43. doi: 10.1017/S0048577299971184Published

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development,* 317-333. doi: 10.1111/j.1467-8624.2004.00673.x

Cole, P. M., Michel, M. K., & Teti, L. O. D. (1994). The development of emotion regulation and dysregulation: A clinical perspective. *Monographs of the Society for Research in Child Development,* 59(2-3), 73-102. doi: 10.2307/1166139

Cole, P. M., Zahn-Waxler, C., Fox, N. A., Usher, B. A., & Welsh, J. D. (1996). Individual differences in emotion regulation and behavior problems in preschool children. *Journal of Abnormal Psychology*, 105(4), 518-529. doi:10.1037/0021-

843X.105.4.518

Connor, D. F., Glatt, S. J., Lopez, I. D., Jackson, D., & Melloni Jr, R. H. (2002). Psychopharmacology and aggression. I: A meta-analysis of stimulant effects on overt/covert aggression–related behaviors in ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry*, *41*(3), 253-261. doi: 10.1097/00004583-200203000-00004

Copeland, W. E., Angold, A., Costello, E. J., & Egger, H. (2013). Prevalence, comorbidity, and correlates of DSM-5 proposed disruptive mood dysregulation disorder. *American Journal of Psychiatry*. doi: 10.1176/appi.ajp.2012.12010132

Copeland, W. E., Shanahan, L., Egger, H., Angold, A., & Costello, E. J. (2014). Adult diagnostic and functional outcomes of DSM-5 disruptive mood dysregulation disorder. *American Journal of Psychiatry*, *171*(6), 668-674. doi: 10.1176/appi.ajp.2014.13091213

Council for Medical Schemes (2015). *Prevalence of chronic diseases in the population covered by medical aid schemes in South Africa: December 2015*. Centurion, SA: Author.

Corr, P. J. (2001). Testing problems in JA Gray's personality theory: A commentary on Matthews and Gilliland (1999). *Personality and Individual Differences*, *30*(2), 333-352. doi: 10.1016/S0191-8869(00)00028-3

Corr, P. J. (2002). JA Gray's reinforcement sensitivity theory and frustrative nonreward: A theoretical note on expectancies in reactions to rewarding stimuli. *Personality and Individual Differences*, *32*(7), 1247-1253. doi: 10.1016/S0191-8869(01)00115-5

Corr, P. J. (2004). Reinforcement sensitivity theory and personality. *Neuroscience & Biobehavioral Reviews*, *28*(3), 317-332. doi: 10.1016/j.neubiorev.2004.01.005.

Craddock, N., & Owen, M. J. (2010). The Kraepelinian dichotomy – going, going... but still not gone. *The British Journal of Psychiatry*, 196(2), 92–95. doi:10.1192/bjp.bp.109.073429

Crowell, S. E., Beauchaine, T. P., McCauley, E., Smith, C. J., Stevens, A. L. & Sylvers, P. (2005). Psychological, autonomic, and serotonergic correlates of parasuicidal among adolescent girls. *Development and Psychopathology*, *17*, 1105–1127. doi:10.10170S0954579405050522

Cummings, E. M., & Davies, P. (1996). Emotional security as a regulatory process in normal development and the development of psychopathology. *Development and Psychopathology*, 8(1), 123-139. doi: 10.1017/S0954579400007008

Cummins, N., Scherer, S., Krajewski, J., Schnieder, S., Epps, J., & Quatieri, T. F. (2015). A review of depression and suicide risk assessment using speech analysis. *Speech Communication*, *71*, 10-49. doi: 10.1016/j.specom.2015.03.004

Dadds, M. R., Perrin, S., & Yule, W. (1998). Social desirability and self-reported anxiety in children: An analysis of the RCMAS Lie Scale. *Journal of Abnormal Child Psychology, 26*(4), 311-317. doi: 10.1023/a:1022610702439

Damasio, A. R. (1994). *Descartes' error: Emotion, reason, and the human brain*. New York: G. P. Putnam.

Damasio, A. R. (1996). The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences, 351*(1346), 1413-1420. doi: 10.1098/rstb.1996.0125

Darwin, C. (1872). The expression of emotions in animals and man. *London: Murray*, 11. Retrieved from https://books.google.co.za/books?id=2vzCDwAAQBAJ&printsec=frontcover&source=gbs_ge_summary_r&cad=0#v=onepage&q&f=false

Damasio, A. R. (2001). Fundamental feelings. *Nature*, 413, 781. doi: 10.1038/35101669

Davidson, R. J. (1984). Affect, Cognition, and Hemispheric Specialization. In C. E. Izard, J. Kagan, & R. Zajonc (Eds.), *Emotion, Cognition, and Behavior*, 320-365. New York: Cambridge University Press.

Davidson, R. J. (1998). Anterior electrophysiological asymmetries, emotion, and depression: Conceptual and methodological conundrums. *Psychophysiology, 35*(5), 607-614. doi: 10.1017/S0048577298000134

Davidson, R. J., Ekman, P., Saron, C. D., Senulis, J. A., & Friesen, W. V. (1990). Approach-withdrawal and cerebral asymmetry: Emotional expression and brain physiology: *International Journal of Personality and Social Psychology*, 58(2), 330. doi: : 10.1037/0022-3514.58.2.33

De Wied, M., van Boxtel, A., Matthys, W. & Meeus, W. (2012). Verbal, facial and autonomic responses to empathy-eliciting film clips by disruptive male adolescents with high versus low callous-unemotional traits. *Journal of Abnormal Child Psychology*, *40*, 211–223. doi:10.1007/s10802-011-9557-8

Degangi, G. A., Dipietro, J. A., Greenspan, S. I., & Porges, S. W. (1991). Psychophysiological characteristics of the regulatory disordered infant. *Infant Behavior and Development*, *14*(1), 37-50. doi: 10.1016/0163-6383(91)90053-U

Degnan, K. A., Calkins, S. D., Keane, S. P., & Hill-Soderlund, A. L. (2008). Profiles of disruptive behavior across early childhood: Contributions of frustration reactivity, physiological regulation, and maternal behavior. *Child Development*, *79*(5), 1357–1376. doi:10.1111/j.1467-8624.2008.01193.x

Denver, J. W. (2004). The social engagement system: Functional differences in individuals with autism. *Dissertation Abstracts International*, 65(3-B), 1591, University of Maryland. Retrieved from http://hdl.handle.net/1903/1351

Depue, R. A., & Collins, P. F. (1999). Neurobiology of the structure of personality: Dopamine, facilitation of incentive motivation, and extraversion. *Behavioral and Brain Sciences, 22*(3), 491-517. doi: 10.1017/S0140525X99002046

Depue, R. A., & Iacono, W. G. (1989). Neurobehavioral aspects of affective disorders. *Annual Review of Psychology, 40*(1), 457-492. doi: 10.1146/annurev.ps.40.020189.002325

Deveney, C. M., Brotman, M. A., Decker, A. M., Pine, D. S., & Leibenluft, E. (2012). Affective prosody labeling in youths with bipolar disorder or severe mood dysregulation. *Journal of Child Psychology and Psychiatry, 53*(3), 262-270. doi: 10.1111/j.1469-7610.2011.02482.x

Deveney, C. M., Connolly, M. E., Haring, C. T., Bones, B. L., Reynolds, R. C., Kim, P., Pine, D.S. & Leibenluft, E. (2013). Neural mechanisms of frustration in chronically irritable children. *American Journal of Psychiatry,* 170(10), 1186-1194. doi:10.1176/appi.ajp.2013.12070917

Deveney, C. M., Hommer, R. E., Reeves, E., Stringaris, A., Hinton, K. E., Haring, C. T., … Leibenluft, E. (2015). A Prospective study of severe irritability in youths: 2- and 4-year follow-up. *Depression and Anxiety, 32*(5), 364–372. doi: 10.1002/da.22336

Diamond, L. M., & Aspinwall, L. G. (2003). Emotion regulation across the life span: An integrative perspective emphasizing self-regulation, positive affect, and dyadic processes. *Motivation and Emotion,* 27(2), 125-156. doi: 10.1023/A:102452192

Diamond, L. M., Fagundes, C. P., & Butterworth, M. R. (2012). Attachment style, vagal tone, and empathy during mother–adolescent interactions. *Journal of Research on Adolescence*, *22*(1), 165-184. doi: 10.1111/j.1532-7795.2011.00762.x

Dickstein, D. P., Finger, E. C., Brotman, M. A., Rich, B. A., Pine, D. S., Blair, J. R., & Leibenluft, E. (2010). Impaired probabilistic reversal learning in youths with mood and anxiety disorders. *Psychological Medicine*, *40*(07), 1089-1100. doi: 10.1017/S0033291709991462

Dickstein, D. P., Nelson, E. E., McClure, E. B., Grimley, M. E., Knopf, L., Brotman, M. A., ... & Leibenluft, E. (2007). Cognitive flexibility in phenotypes of pediatric bipolar disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 46(3), 341-355. doi: 10.1097/chi.0b013e31802d0b3d

Dickstein, D. P., Towbin, K. E., Van Der Veen, J. W., Rich, B. A., Brotman, M. A., Knopf, L., ... & Leibenluft, E. (2009). Randomized double-blind placebo-controlled trial of lithium in youths with severe mood dysregulation. *Journal of Child and Adolescent Psychopharmacology*, *19*(1), 61-73. doi: 10.1089/cap.2008.044

Dietrich, A. A., Riese, H. H., Sondeijker, F. F., Greaves-Lord, K. K., van Roon, A. A., Ormel, J. J., ... & Rosmalen, J. J. (2007). Externalizing and internalizing problems in relation to autonomic function: A population-based study in preadolescents. *American Academy of Child and Adolescent Psychiatry*, *46*(3), 378-386. doi: 10.1097/CHI.0b013e31802b91ea

Dodge, K. A. (1989). Coordinating responses to aversive stimuli: Introduction to a special section on the development of emotion regulation. *Developmental Psychology*, *25*(3), 339. doi: 10.1037/0012-1649.25.3.339

Donovan, S. J., Stewart, J. W., Nunes, E. V., Quitkin, F. M., Parides, M., Daniel, W., ... & Klein, D. F. (2000). Divalproex treatment for youth with explosive temper and mood lability: a double-blind, placebo-controlled crossover design. *American Journal of Psychiatry*, *157*(5), 818-820. doi: 10.1176/appi.ajp.157.5.818

Dougherty, L. R., Bufferd, S. J., Carlson, G. A., Dyson, M., Olino, T. M., Durbin, C. E., & Klein, D. N. (2011). Preschoolers' observed temperament and psychiatric disorders assessed with a parent diagnostic interview. *Journal of Clinical Child & Adolescent Psychology*, *40*(2), 295-306. doi: 10.1080/15374416.2011.546046

Dougherty, L. R., Smith, V. C., Bufferd, S. J., Carlson, G. A., Stringaris, A., Leibenluft, E., & Klein, D. N. (2014). DSM-5 disruptive mood dysregulation disorder: correlates and predictors in young children. *Psychological Medicine*, *44*(11), 2339-2350. doi: 10.1017/S0033291713003115

Dougherty, L. R., Smith, V. C., Bufferd, S. J., Kessel, E. M., Carlson, G. A., & Klein, D. N. (2016). Disruptive mood dysregulation disorder at the age of 6 years and clinical and functional outcomes 3 years later. *Psychological Medicine*, *46*(5), 1103-1114. doi:10.1017/S0033291715002809

Doussard-Roosevelt, J. A., McClenny, B. D., & Porges, S. W. (2001). Neonatal cardiac vagal tone and school-age developmental outcome in very low birth weight infants. *Developmental Psychobiology: The Journal of the International Society for Developmental Psychobiology*, *38*(1), 56-66. doi: 10.1002/1098-2302(2001)38:1<56::AID-DEV5>3.0.CO;2-K

Doussard-Roosevelt, J. A., Montgomery, L. A., & Porges, S. W. (2003). Short-term stability of physiological measures in kindergarten children: Respiratory sinus arrhythmia, heart period, and cortisol. *Developmental Psychobiology: The Journal of the International Society for Developmental Psychobiology*, 43(3), 230-242. doi: 10.1002/dev.10136

Driver, D. I., & Thomas, S. (2018). *Complex Disorders in Pediatric Psychiatry: A Clinician's Guide*. Elsevier Health Sciences.

Driver, J., Vuilleumier, P., Eimer, M., & Rees, G. (2001). Functional magnetic resonance imaging and evoked potential correlates of conscious and unconscious vision in parietal extinction patients. *Neuroimage*, *14*(1), S68-S75. doi: 10.1006/nimg.2001.0842

Duan, X., Dai, Q., Gong, Q., & Chen, H. (2010). Neural mechanism of unconscious perception of surprised facial expression. *Neuroimage*, *52*(1), 401-407. doi: 10.1016/j.neuroimage.2010.04.021

Egan, G. J., Brown, R. T., Goonan, L., Goonan, B. T., & Celano, M. (1998). The development of decoding of emotions in children with externalizing behavioral disturbances and their normally developing peers. *Archives of Clinical Neuropsychology*, *13*(4), 383-396. doi: 10.1016/S0887-6177(97)00019-X

Eisenberg, N. (2000). Emotion, regulation, and moral development. *Annual Review of Psychology*, 51(1), 665-697. doi: 10.1146/annurev.psych.51.1.665

Eisenberg, N., Cumberland, A., Spinrad, T. L., Fabes, R. A., Shepard, S. A., Reiser, M., ... & Guthrie, I. K. (2001). The relations of regulation and emotionality to children's externalizing and internalizing problem behavior. *Child Development*, 1112-1134. doi: 10.1111/1467-8624.00337

Eisenberg, N., Fabes, R. A., Guthrie, I. K., Murphy, B. C., Maszk, P., Holmgren, R., & Suh, K. (1996). The relations of regulation and emotionality to problem behavior in elementary school children. *Development and Psychopathology*, 8(1), 141-162. doi: 10.1017/S095457940000701X

Eisenberg, N., Fabes, R. A., Murphy, B., Maszk, P., Smith, M., & Karbon, M. (1995). The role of emotionality and regulation in children's social functioning: A longitudinal study. *Child Development*, *66*(5), 1360-1384. doi: 10.1111/j.1467-8624.1995.tb00940.x

Ekman, P. (1957). A Methodological Discussion of Nonverbal Behavior. *The Journal of Psychology*, 43, 141-149. doi: 10.1080/00223980.1957.9713059

Ekman, P. (1984). Expression and the Nature of Emotion. In K. Scherer & P. Ekman (Eds.), *Approaches to Emotion*, 319-343. Hillsdale, NJ: Lawrence Erlbaum. Retrieved from https://www.paulekman.com.

Ekman, P. (1992a). An argument for basic emotions. *Cognition & Etmotion*, 6(3-4), 169-200. doi: 10.1080/02699939208411068

Ekman P. (1992b). Are there basic emotions? *Psychological Review*, 99, 550-553. doi: 10.1037/0033-295X.99.3.550

Ekman, P. (1993). Facial expression and emotion. *American Psychologist*, *48*(4), 384. doi: 10.1037/0003-066X.48.4.384

Ekman, P. (1999). Basic Emotions. In Dalgleish, T. & Power, M. J. (Eds.), *Handbook of Cognition and Emotion*, 45-60. New York, NY: John Wiley & Sons Ltd. doi: 10.1002/0470013494.ch3

Ekman, P., & Cordaro, D. (2011). What is meant by calling emotions basic. *Emotion Review*, *3*(4), 364-370. doi: 10.1177/1754073911410740

Ekman, P., Friesen, W. V., & Ellsworth, P. (1972). *Emotion in the Human Face: Guidelines for Research and an Integration of Findings*. Pergamon.

Ekman, P., Levenson, R. W., & Friesen, W. V. (1983). Autonomic nervous system activity distinguishes among emotions. *Science*, 221(4616), 1208-1210. doi: 10.1126/science.6612338

El-Sheikh, M. (2001). Parental drinking problems and children's adjustment: Vagal regulation and emotional reactivity as pathways and moderators of risk. *Journal of Abnormal Psychology*, 110(4), 499. doi: 10.1037/0021-843X.110.4.499

El-Sheikh, M. (2005). Stability of respiratory sinus arrhythmia in children and young adolescents: A longitudinal examination. *Developmental Psychobiology: The Journal of the International Society for Developmental Psychobiology*, *46*(1), 66-74. doi: 10.1002/dev.20036

Else, P. L., & Hulbert, A. J. (1981). Comparison of the "mammal machine" and the "reptile machine": energy production. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, *240*(1), R3-R9. doi: 10.1152/ajpregu.1981.240.1.R3

Emerson, C. S., Harrison, D. W., & Everhart, D. E. (1999). Investigation of receptive affective prosodic ability in school-aged boys with and without depression. *Neuropsychiatry, Neuropsychology, & Behavioral Neurology*.

Factor, P. I., Rosen, P. J., & Reyes, R. A. (2016). The relation of poor emotional awareness and externalizing behavior among children with ADHD. *Journal of Attention Disorders*, *20*(2), 168-177. doi: 10.1177/1087054713494005

Farb, N. A., Chapman, H. A., & Anderson, A. K. (2013). Emotions: Form follows function. *Current Opinion in Neurobiology*, *23*(3), 393-398. doi: 10.1016/j.conb.2013.01.015

Faul, F., Erdfelder, E., Lang, A. G., & Buchner, A. (2007). G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior research methods*, *39*(2), 175-191. doi: 10.3758/BF03193146

Fenton-O'Creevy, M., Lins, J. T., Vohra, S., Richards, D. W., Davies, G., & Schaaff, K. (2012). Emotion regulation and trader expertise: Heart rate variability on the trading floor. *Journal of Neuroscience, Psychology, and Economics*, *5*(4), 227. doi: 10.1037/a0030364

Finlay-Jones, R., & Brown, G. W. (1981). Types of stressful life event and the onset of anxiety and depressive disorders. *Psychological Medicine*, *11*(4), 803-815. doi: 10.1017/S0033291700041301

Finzi, R., Cohen, O., Sapir, Y., & Weizman, A. (2000). Attachment styles in maltreated children: A comparative study. *Child Psychiatry and Human Development*, *31*(2), 113-128.

Finzi, R., Har-Even, D., Weizman, A., Tyano, S., & Shnit, D. (1996). The adaptation of the attachment style questionnaire for latency-aged children. *Psychology*, *5*, 167-177.

Fitzgerald, D. A., Angstadt, M., Jelsone, L. M., Nathan, P. J., & Phan, K. L. (2006). Beyond threat: amygdala reactivity across multiple expressions of facial affect. *Neuroimage*, *30*(4), 1441-1448. doi: 10.1016/j.neuroimage.2005.11.003

Fortunato, C. K., Gatzke-Kopp, L. M., & Ram, N. (2013). Associations between respiratory sinus arrhythmia reactivity and internalizing and externalizing symptoms are emotion specific. *Cognitive, Affective, & Behavioral Neuroscience*, *13*(2), 238-251. doi: 10.3758/s13415-012-0136-4.

Fowles, D. C. (1980). The three arousal model: Implications of Gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. *Psychophysiology*, *17*(2), 87-104. doi: 10.1111/j.1469-8986.1980.tb00117.x

Fowles, D. C. (1988). Psychophysiology and psychopathology: A motivational approach. *Psychophysiology*, *25*(4), 373-391. doi: 10.1111/j.1469-8986.1988.tb01873.x

Fox, N. A. (1989). Psychophysiological correlates of emotional reactivity during the first year of life. *Developmental Psychology*, 25(3), 364. doi: 10.1037/0012-1649.25.3.364

Freegard, G. (2012). *Response Pads: The design, development and results of a bespoke response box, and subsequent testing, for comparison purposes, of various devices used for reaction time measurements in psychological experiments*. Retrieved from http://psy.swan.ac.uk/staff/freegard/Response%20Box%20Report.pdf

Freeman, A. J., Youngstrom, E. A., Youngstrom, J. K., & Findling, R. L. (2016). Disruptive mood dysregulation disorder in a community mental health clinic: prevalence, comorbidity and correlates. *Journal of Child and Adolescent Psychopharmacology*, 26(2), 123-130. doi: 10.1089/cap.2015.0061

Friedman, B. H., & Thayer, J. F. (1998). Autonomic balance revisited: panic anxiety and heart rate variability. *Journal of Psychosomatic Research*, *44*(1), 133-151. doi: 10.1016/S0022-3999(97)00202-X

Frijda, N. H. (1986). *The emotions*. Cambridge University Press.

Geisler, F. C., Kubiak, T., Siewert, K., & Weber, H. (2013). Cardiac vagal tone is associated with social engagement and self-regulation. *Biological Psychology*, *93*(2), 279-286. doi: 10.1016/j.biopsycho.2013.02.013

Gentzler, A. L., Santucci, A. K., Kovacs, M., & Fox, N. A. (2009). Respiratory sinus arrhythmia reactivity predicts emotion regulation and depressive symptoms in at-risk and control children. *Biological Psychology*, 82(2), 156-163. doi: 10.1016/j.biopsycho.2009.07.002

George, D. T., Nutt, D. J., Walker, W. V., Porges, S. W., Adinoff, B., & Linnoila, M. (1989). Lactate and hyperventilation substantially attenuate vagal tone in normal volunteers: A possible mechanism of panic provocation? *Archives of General Psychiatry*, *46*(2), 153-156. doi: 10.1001/archpsyc.1989.01810020055009

Gisunterman, A., Cohn, Y., & Weizman, A. (2018). Disruptive Mood Dysregulation Disorder - A New and Challenging Diagnosis in DSM-5. *Harefuah*, *157*(8), 525-528. Retrieved from http://0-resolver.ebscohost.com.innopac.wits.ac.za/openurl?sid=Entrez%3aPubMed&id=pmid%3a30175571&site=ftf-live

Goldin, P. R., McRae, K., Ramel, W., & Gross, J. J. (2008). The neural bases of emotion regulation: reappraisal and suppression of negative emotion. *Biological Psychiatry*, 63(6), 577-586. doi: 10.1016/j.biopsych.2007.05.031

Gordis, E. B., Feres, N., Olezeski, C. L., Rabkin, A. N., & Trickett, P. K. (2009). Skin conductance reactivity and respiratory sinus arrhythmia among maltreated and comparison youth: Relations with aggressive behavior. *Journal of Pediatric Psychology*, *35*(5), 547-558. doi: 10.1093/jpepsy/jsp113

Gorenstein, E. E., & Newman, J. P. (1980). Disinhibitory psychopathology: A new perspective and a model for research. *Psychological Review*, *87*(3), 301. doi: 10.1037/0033-295X.87.3.301

Gottman, J. M., & Katz, L. F. (2002). Children's emotional reactions to stressful parent-child interactions: The link between emotion regulation and vagal tone. *Marriage & Family Review*, *34*(3-4), 265-283. doi: 10.1300/J002v34n03_04

Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of Psychopathology and Behavioral assessment,* 26(1), 41-54. doi: 10.1023/B:JOBA.0000007455.08539.94

Graziano, P., & Derefinko, K. (2013). Cardiac vagal control and children's adaptive functioning: A meta-analysis. *Biological Psychology,* 94(1), 22-37. doi:10.1016/j.biopsycho.2013.04.011

Graziano, P.A., Keane, S.P., & Calkins, S.D. (2007). Cardiac vagal regulation and early peer status. *Child Development*, 78, 264–278. doi:10.1111/j.1467-8624.2007.00996.x

Gray, J. A. (1970). The psychophysiological basis of introversion-extraversion. *Behaviour Research and Therapy*, *8*(3), 249-266. doi: 10.1016/0005-7967(70)90069-0

Gray, J. A. (1975). *Elements of a two-process theory of learning*. Academic Press.

Gray, J.A. (1976). The behavioral inhibition system: A possible substrate for anxiety. In M.P. Feldman & A. Broadhurst (Eds.), *Theoretical and Experimental Bases of the Behavior Therapies*, Wiley, New York

Gray, J.A. (1982). *The neuropsychology of anxiety: an enquiry into the functions of the septo-hippocampal system*. Oxford University Press, New York.

Gray, J.A. (1994). Three fundamental emotion systems. In P. Ekman & R.J. Davidson (Eds). *The nature of Emotion: Fundamental questions* (pp. 243-247). Oxford: Oxford University Press.

Gray, J., & McNaughton, N. (2003). (Ed.), *The Neuropsychology of Anxiety: An enquiry into the function of the septo-hippocampal system*. Oxford: Oxford University Press. Retrieved 10 Aug. 2018, from http://www.oxfordscholarship.com/view/10.1093/acprof:oso/9780198522713.001.0001/acprof-9780198522713.

Grimmer, Y., Hohmann, S., & Poustka, L. (2014). Is bipolar always bipolar? Understanding the controversy on bipolar disorder in children. *F1000Prime Reports*, 6, 111. doi: 10.12703/P6-111

Groen, R. N., de Clercq, N. C., Nieuwdorp, M., Hoenders, H. R., & Groen, A. K. (2018). Gut microbiota, metabolism and psychopathology: A critical review and novel perspectives. *Critical Reviews in Clinical Laboratory Sciences*, *55*(4), 283-29. doi: 10.1080/10408363.2018.1463507

Gross, J. J. (2001). Emotion regulation in adulthood: Timing is everything. *Current Directions in Psychological Science*, 10(6), 214-219. doi: 10.1111/1467-8721.00152

Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology*, *39*(03), 281-291. doi: 10.1017.S0048577201393198

Gross, J. J. (2015). Emotion regulation: Current status and future prospects. *Psychological Inquiry*, *26*(1), 1-26. doi: 10.1080/1047840X.2014.940781

Gross, J. J., & Feldman Barrett, L. (2011). Emotion generation and emotion regulation: One or two depends on your point of view. *Emotion Review*, *3*(1), 8-16. doi: 10.1177/1754073910380974

Gross, J. J., & Jazaieri, H. (2014). Emotion, emotion regulation, and psychopathology an affective science perspective. *Clinical Psychological Science*, *2*(4), 387-401. doi: 10.1177/2167702614536164

Gross, J. J., & Muñoz, R. F. (1995). Emotion regulation and mental health. *Clinical Psychology: Science and Practice*, 2(2), 151-164. doi: 10.1111/j.1468-2850.1995.tb00036.x

Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J.J. Gross (Ed.), *Handbook of Emotion Regulation* (pp. 3-24). New York: Guilford Press.

Grossman, P., & Taylor, E. W. (2007). understanding respiratory sinus arrhythmia: relations to cardiac vagal tone, evolution and biobehavioral functions. *Biological Psychology*, *74*(2), 263-285. doi: 10.1016/j.biopsycho.2005.11.014

Gunn, R. L., & Finn, P. R. (2015). Applying a dual process model of self-regulation: The association between executive working memory capacity, negative urgency, and negative mood induction on pre-potent response inhibition. *Personality and Individual Differences*, *75*, 210-215. doi: 10.1016/j.paid.2014.11.033

Guyer, A. E., McClure, E. B., Adler, A. D., Brotman, M. A., Rich, B. A., Kimes, A. S., Pine, D. S., Ernst, M. and Leibenluft, E. (2007), Specificity of facial expression labeling deficits in childhood psychopathology. *Journal of Child Psychology and Psychiatry*, 48: 863–871. doi: 10.1111/j.1469-7610.2007.01758.x

Halberstadt, A. G., Denham, S. A., & Dunsmore, J. C. (2001). Affective social competence. *Social Development*, 10, 79–119. doi:10.1111/sode.2001.10.issue-1

Hansen, A. L., Johnsen, B. H., Thornton, D., Waage, L. & Thayer, J. F. (2007). Facets of psychopathy, heart rate variability and cognitive function. *Journal of Personality Disorders*, *21*, 568–582. doi:10.1521/pedi.2007.21.5.568

Harmon-Jones, E. (2003). Anger and the behavioral approach system. *Personality and Individual Differences*, *35*(5), 995-1005. doi: 10.1016/S0191-8869(02)00313-6

Harmon-Jones, E., & Allen, J. J. (1997). Behavioral activation sensitivity and resting frontal EEG asymmetry: Covariation of putative indicators related to risk for mood disorders. *Journal of Abnormal Psychology*, *106*(1), 159. doi: 10.1037/0021-843X.106.1.159

Harmon-Jones, E., & Allen, J. J. (1998). Anger and frontal brain activity: EEG asymmetry consistent with approach motivation despite negative affective valence. *Journal of Personality and Social Psychology, 74*(5), 1310. doi: 10.1037//0022-3514.74.5.1310

Harmon-Jones, E., & Sigelman, J. (2001). State anger and prefrontal brain activity: Evidence that insult-related relative left-prefrontal activation is associated with experienced anger and aggression. *Journal of Personality and Social Psychology, 80*(5), 797. doi: 10.1037/0022-3514.80.5.797

Harmon-Jones, E., Sigelman, J., Bohlig, A., & Harmon-Jones, C. (2003). Anger, coping, and frontal cortical activity: The effect of coping potential on anger-induced left frontal activity. *Cognition and Emotion*, 17(1), 1-24. doi: 10.1080/02699930302278

Harris, A. D., McGregor, J. C., Perencevich, E. N., Furuno, J. P., Zhu, J., Peterson, D. E., & Finkelstein, J. (2006). The use and interpretation of quasi-experimental studies in medical informatics. *Journal of the American Medical Informatics Association : JAMIA*, *13*(1), 16-23. doi: 10.1197/jamia.M1749

Hastings, P. D., Nuselovici, J. N., Utendale, W. T., Coutya, J., McShane, K. E., & Sullivan, C. (2008). Applying the polyvagal theory to children's emotion regulation: Social context, socialization, and adjustment. *Biological Psychology, 79*(3), 299-306. doi: 10.1016/j.biopsycho.2008.07.005

Hay, E. L., & Diehl, M. (2011). Emotion complexity and emotion regulation across adulthood. *European Journal of Ageing*, 8(3), 157-168. doi: 10.1007/s10433-011-0191-7

Hazan, C., & Shaver, P. (1987). Romantic love conceptualized as an attachment process. *Journal of Personality and Social Psychology*, *52*(3), 511. doi: 10.1037%2F0022-3514.52.3.511

Heilman, K. J., Harden, E. R., Weber, K. M., Cohen, M., & Porges, S. W. (2013). Atypical autonomic regulation, auditory processing, and affect recognition in women with HIV. *Biological Psychology*, *94*(1), 143-151. doi: 10.1016/j.biopsycho.2013.06.003

Higgins, E. T., Shah, J., & Friedman, R. (1997). Emotional responses to goal attainment: strength of regulatory focus as moderator. *Journal of Personality and Social Psychology*, *72*(3), 515. doi: 10.1037//0022-3514.72.3.515

Hill-Soderlund, A. L., Mills-Koonce, W. R., Propper, C., Calkins, S. D., Granger, D. A., Moore, G. A., ... & Cox, M. J. (2008). Parasympathetic and sympathetic responses to the strange situation in infants and mothers from avoidant and securely attached dyads. *Developmental Psychobiology: The Journal of the International Society for Developmental Psychobiology*, *50*(4), 361-376. doi: 10.1002/dev.20302

Hillyard, S. A., Hink, R. F., Schwent, V. L., & Picton, T. W. (1973). Electrical signs of selective attention in the human brain. *Science*, *182*(4108), 177-180. doi: 10.1126/science.182.4108.177

Holodynski, M., & Friedlmeier, W. (2006). *Development of emotions and emotion regulation* (Vol. 8). Springer Science & Business Media.

Hommer, R. E., Meyer, A., Stoddard, J., Connolly, M. E., Mogg, K., Bradley, B. P., Pine, D. S., Leibenluft, E. and Brotman, M. A. (2014). Attention bias to threat faces in severe mood dysregulation. *Depression and Anxiety*, 31, 559–565. doi: 10.1002/da.22145

Hongwanishkul, D., Happaney, K. R., Lee, W. S., & Zelazo, P. D. (2005). Assessment of hot and cool executive function in young children: Age-related changes and individual differences. *Developmental Neuropsychology*, *28*(2), 617-644. doi: 10.1207/s15326942dn2802_4

Horan, W. P., Kern, R. S., Tripp, C., Hellemann, G., Wynn, J. K., Bell, M., ... & Green, M. F. (2011). Efficacy and specificity of social cognitive skills training for outpatients with psychotic disorders. *Journal of Psychiatric Research*, *45*(8), 1113-1122. doi: 10.1016/j.jpsychires.2011.01.015

Hugdahl, K., & Nordby, H. (1994). Electrophysiological correlates to cued attentional shifts in the visual and auditory modalities. *Behavioral and neural biology*, *62*(1), 21-32. doi: 10.1016/S0163-1047(05)80055-X

Hutcherson, C. A., & Gross, J. J. (2011). The moral emotions: A social–functionalist account of anger, disgust, and contempt. *Journal of Personality and Social Psychology*, 100(4), 719–737. doi: 10.1037/a0022408

Insel, T. R., & Young, L. J. (2001). The neurobiology of attachment. *Nature Reviews Neuroscience*, *2*(2), 129-136. doi: 10.1038/35053579

Irwin, W., Davidson, R. J., Lowe, M. J., Mock, B. J., Sorenson, J. A., & Turski, P. A. (1996). Human amygdala activation detected with echo-planar functional magnetic resonance imaging. *Neuroreport*, *7*(11), 1765-1769. doi:10.1097/00001756-199607290-00014

Izard, C. E. (1972). *Patterns of emotions: A new analysis of anxiety and depression*. New York: Academic Press.

Izard, C. E. (1977). *Human emotions*. New York, NY: Plenum Press. doi: 10.1007/978-1-4899-2209-0

Izard, C. E. (1990). Facial expressions and the regulation of emotions. *Journal of Personality and Social Psychology*, 58(3), 487. doi: 10.1037/0022-3514.58.3.487

Jacob, J. S., & McDonald, H. S. (1976). Diving bradycardia in four species of North American aquatic snakes. *Comparative Biochemistry and Physiology Part A: Physiology*, *53*(1), 69-72. doi: 10.1016/S0300-9629(76)80013-8

Jacob, S. N., Shear, P. K., Norris, M., Smith, M., Osterhage, J., Strakowski, S. M., ... & Eliassen, J. C. (2015). Impact of functional magnetic resonance imaging (fMRI) scanner noise on affective state and attentional performance. *Journal of Clinical and Experimental Neuropsychology*, *37*(6), 563-570. doi: 10.1080/13803395.2015.1029440

James, W. (1884). What is an emotion? *Mind*, 9(34): 188-205. doi: 10.1093/mind/os-IX.34.188

Jewell, T., Gardner, T., Susi, K., Watchorn, K., Coopey, E., Simic, M., ... & Eisler, I. (2019). Attachment measures in middle childhood and adolescence: A systematic review of measurement properties. *Clinical Psychology Review*, *68*, 71-82. doi: 10.1016/j.cpr.2018.12.004

Jönsson, P., & Sonnby-Borgström, M. (2003). The effects of pictures of emotional faces on tonic and phasic autonomic cardiac control in women and men. *Biological Psychology*, 62(2), 157-173. doi: 10.1016/S0301-0511(02)00114-X

Kakouros, S., & Räsänen, O. (2016). Perception of sentence stress in speech correlates with the temporal unpredictability of prosodic features. *Cognitive Science*, 40(7), 1739-1774. doi: 10.1111/cogs.12306

Kan, Y., Mimura, M., Kamijima, K., & Kawamura, M. (2004). Recognition of emotion from moving facial and prosodic stimuli in depressed patients. *Journal of Neurology, Neurosurgery & Psychiatry*, 75(12), 1667-1671. doi: 10.1136/jnnp.2004.036079

Kashdan, T. B., & Rottenberg, J. (2010). Psychological flexibility as a fundamental aspect of health. *Clinical Psychology Review*, *30*(7), 865-878. doi: 10.1016/j.cpr.2010.03.001

Keenan, K. (2000). Emotion dysregulation as a risk factor for child psychopathology. *Clinical Psychology: Science and Practice, 7*(4), 418-434. doi: 10.1093/clipsy.7.4.418

Keltner, D., & Gross, J. J. (1999). Functional accounts of emotions. *Cognition & Emotion, 13*(5), 467-480. doi: 10.1080/026999399379140

Kemp, A. H., Brunoni, A. R., Santos, I. S., Nunes, M. A., Dantas, E. M., de Figueiredo, R. C., … Lotufo, P. A. (2014). Effects of depression, anxiety, comorbidity, and antidepressants on resting-state heart rate and its variability: An ELSA-Brasil cohort baseline study. *American Journal of Psychiatry*, *171*, 1328–1334. doi:10.1176/appi.ajp.2014.13121605

Kemp, A. H., & Quintana, D. S. (2013). The relationship between mental and physical health: insights from the study of heart rate variability. *International Journal of Psychophysiology*, *89*(3), 288-296. doi: 10.1016/j.ijpsycho.2013.06.018

Kemp, A. H., Quintana, D. S., Gray, M. A., Felmingham, K. L., Brown, K., & Gatt, J. M. (2010). Impact of depression and antidepressant treatment on heart rate variability: a review and meta-analysis. *Biological Psychiatry*, *67*(11), 1067-1074. doi: 10.1016/j.biopsych.2009.12.012

Kessing, L.V., Vradi, E., & Andersen, P. K. (2014). Are rates of pediatric bipolar disorder increasing? Results from a nationwide register study. *International Journal of Bipolar Disorders, 2*(1), 10. doi:10.1186/s40345-014-0010-0

Kim, S., & Boylan, K. (2016). Effectiveness of antidepressant medications for symptoms of irritability and disruptive behaviors in children and adolescents. *Journal of Child and Adolescent Psychopharmacology*, *26*(8), 694-704. doi: 10.1089/cap.2015.0127

Kim, P., Evans, G. W., Angstadt, M., Ho, S. S., Sripada, C. S., Swain, J. E., ... & Phan, K. L. (2013). Effects of childhood poverty and chronic stress on emotion regulatory brain function in adulthood. *Proceedings of the National Academy of Sciences*, 110(46), 18442-18447. doi: 10.1073/pnas.1308240110

Kluender, K. R., Coady, J. A., & Kiefte, M. (2003). Sensitivity to change in perception of speech. *Speech Communication*, *41*(1), 59-69. doi: 10.1016/S0167-6393(02)00093-6

Kogan, A., Gruber, J., Shallcross, A. J., Ford, B. Q., & Mauss, I. B. (2013). Too much of a good thing? Cardiac vagal tone's nonlinear relationship with well-being. *Emotion*, *13*(4), 599. doi: 10.1037/a0032725

Kogan, A., Oveis, C., Carr, E. W., Gruber, J., Mauss, I. B., Shallcross, A., ... & Keltner, D. (2014). Vagal activity is quadratically related to prosocial traits, prosocial emotions, and observer perceptions of prosociality. *Journal of Personality and Social Psychology*, *107*(6), 1051. doi: 10.1037/a0037509

Kok, B. E., Coffey, K. A., Cohn, M. A., Catalino, L. I., Vacharkulksemsuk, T., Algoe, S. B., ... & Fredrickson, B. L. (2013). How positive emotions build physical health: Perceived positive social connections account for the upward spiral between positive emotions and vagal tone. *Psychological Science, 24*(7), 1123-1132. doi: 10.1177/0956797612470827

Kok, B. E., & Fredrickson, B. L. (2010). Upward spirals of the heart: Autonomic flexibility, as indexed by vagal tone, reciprocally and prospectively predicts positive emotions and social connectedness. *Biological Psychology, 85*(3), 432-436. doi: 10.1016/j.biopsycho.2010.09.005

Kolacz, J. (2016). *Investigating Infant Crying Persistence and Cry Acoustic Features as Early Risk Indicators for Social Adjustment: Developmental Associations with Infant Vagal Tone and Attachment Stress* (Doctor of Philosophy thesis). University of North Carolina, Chapel Hill.

Kolacz, J., Kovacic, K. K., & Porges, S. W. (2019). Traumatic stress and the autonomic brain-gut connection in development: Polyvagal Theory as an integrative framework for psychosocial and gastrointestinal pathology. *Developmental Psychobiology, 61*(5), 796-809. doi: 10.1002/dev.21852

Kolacz, J., Lewis, G. F., & Porges, S. W. (2018). The integration of vocal communication and biobehavioral state regulation in mammals: A polyvagal hypothesis. In *Handbook of Behavioral Neuroscience* (Vol. 25, pp. 23-34). doi: doi.org/10.1016/B978-0-12-809600-0.00003-2.

Koole, S. L. (2009). The psychology of emotion regulation: An integrative review. *Cognition and Emotion*, *23*(1), 4-41. doi: 10.1080/02699930802619031

Kopp, C. B. (1982). Antecedents of self-regulation: A developmental perspective. *Developmental Psychology*, 18(2), 199-214. doi: http://dx.doi.org/10.1037/0012-1649.18.2.199

Kopp, C. B. (1989). Regulation of distress and negative emotions: A developmental view. *Developmental Psychology*, *25*(3), 343. doi: 10.1037/0012-1649.25.3.343

Kopp C. B. & Neufeld S. J. (2003). Emotional development during infancy. In: R. Davidson, K. R. Scherer, & H. H. Goldsmith (Eds.), *Handbook of Affective Sciences*, 347–374. Oxford, UK: Oxford University Press.

Kosslyn, S. M., Shin, L. M., Thompson, W. L., McNally, R. J., Rauch, S. L., Pitman, R. K., & Alpert, N. M. (1996). Neural effects of visualizing and perceiving aversive stimuli: a PET investigation. *Neuroreport*, *7*(10), 1569-1576. Retrieved from http://0-resolver.ebscohost.com.innopac.wits.ac.za/openURL?sid=Entrez:PubMed&id=pmid:8904757

Krause, F. & Lindemann, O. (2014). Expyriment: A Python library for cognitive and neuroscientific experiments. *Behavior Research Methods*, 46(2), 416-428. doi:10.3758/s13428-013-0390-6.

Kusserow, M., Amft, O., Gubelmann, H., & Tröster, G. (2010). Arousal pattern analysis of an Olympic champion in ski jumping. *Sports Technology*, *3*(3), 192-203. doi: 10.1080/19346182.2011.564285

Kusserow, M., Candia, V., Amft, O., Hildebrandt, H., Folkers, G., & Tröster, G. (2012). Monitoring Stage Fright Outside the Laboratory: An Example in Professional Musician Using Wearable Sensors. *Medical Problems of Performing Artists*, *27*(1), 21. PMID: 22543319

Laborde, S., Mosley, E., & Thayer, J. F. (2017). Heart rate variability and cardiac vagal tone in psychophysiological research–recommendations for experiment planning, data analysis, and data reporting. *Frontiers in Psychology*, *8*, 213. doi: 10.3389/fpsyg.2017.00213

Lang, P. J. (1979). A bio-informational theory of emotional imagery. *Psychophysiology*, 16(6), 495-512. doi: 10.1111/j.1469-8986.1979.tb01511.x

Lang, P. J. (1980). Behavioral treatment and bio-behavioral assessment: Computer applications. In J. B. Sidowski, J. H. Johnson, & T A. Williams (Eds.), *Technology in Mental Health Care Delivery Systems* (pp. 119-137). Norwood, NJ: Ablex Publishing.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1990). Emotion, attention, and the startle reflex. *Psychological Review*, *97*(3), 377. doi: 10.1037/0033-295X.97.3.377

Lange, C. G. (1887). *Über Gemütsbewegungen* (Org. Om Sindsbevægelser). Leipzig: Thomas Theodor.

Lawton, M. P. (2001). Emotion in later life. *Current Directions in Psychological Science*, 10(4), 120-123. doi: 10.1111/1467-8721.00130

Leary, A., & Katz, L. F. (2004). Coparenting, family-level processes, and peer outcomes: The moderating role of vagal tone. *Development and Psychopathology*, *16*(03), 593-608. doi: 10.1017/S0954579404004687

LeDoux J.E. (1984). Cognition and Emotion. In: M.S. Gazzaniga (Ed.) *Handbook of Cognitive Neuroscience*, 357-368 Springer, Boston, MA. doi: 10.1007/978-1-4899-2177-2_17

LeDoux, J. E. (1992). Emotion in the amygdala. In J. P. Aggleton (Ed.), *The Amygdala: Neurobiological Aspects of Emotion, Memory and Mental Dysfunction*. New York: WileyLiss, pp. 339-351.

LeDoux, J.E. (1996). *The emotional brain: The mysterious underpinnings of emotional life*. New York: Simon and Schuster.

LeDoux, J. E., Ruggiero, D. A., & Reis, D. J. (1985). Projections to the subcortical forebrain from anatomically defined regions of the medial geniculate body in the rat. *Journal of Comparative Neurology, 242*(2), 182-213. doi: 10.1002/cne.902420204

Leibenluft, E., Charney, D. S., Towbin, K. E., Bhangoo, R. K., & Pine, D. S. (2003). Defining clinical phenotypes of juvenile mania. *American Journal of Psychiatry, 160*(3), 430-437. doi: 10.1176/appi.ajp.160.3.430

Leibenluft, E. (2008). Pediatric Bipolar Disorder Comes of Age. *Archives of General Psychiatry*, 65(10), 1122–1124. http://doi.org/10.1001/archpsyc.65.10.1122

Leibenluft, E. (2011). Severe Mood Dysregulation, Irritability, and the Diagnostic Boundaries of Bipolar Disorder in Youths. *The American Journal of Psychiatry*, 168(2), 129–142. doi:10.1176/appi.ajp.2010.10050766

Lench, H. C., Bench, S. W., Darbor, K. E., & Moore, M. (2015). A functionalist manifesto: Goal-related emotions from an evolutionary perspective. *Emotion Review, 7*(1), 90-98. doi: 10.1177/1754073914553001

Levenson, R. W., Ekman, P., & Friesen, W. V. (1990). Voluntary facial action generates emotion-specific autonomic nervous system activity. *Psychophysiology, 27*(4), 363-384. doi: 10.1111/j.1469-8986.1990.tb02330.x

Licht, C. M., de Geus, E. J., van Dyck, R., & Penninx, B. W. (2009). Association between anxiety disorders and heart rate variability in The Netherlands Study of Depression and Anxiety (NESDA). *Psychosomatic Medicine*, 71(5), 508-518. doi: 10.1097/PSY.0b013e3181a292a6

Liddell, B. J., Brown, K. J., Kemp, A. H., Barton, M. J., Das, P., Peduto, A., ... & Williams, L. M. (2005). A direct brainstem–amygdala–cortical 'alarm'system for subliminal signals of fear. *Neuroimage, 24*(1), 235-243. doi: 10.1016/j.neuroimage.2004.08.016

Lindquist, K. A., & Barrett, L. F. (2012). A functional architecture of the human brain: emerging insights from the science of emotion. *Trends in Cognitive Sciences, 16*(11), 533-540. doi: 10.1016/j.tics.2012.09.005

Lochman, J. E., Evans, S. C., Burke, J. D., Roberts, M. C., Fite, P. J., Reed, G. M., ... & Elena Garralda, M. (2015). An empirically based alternative to DSM-5's disruptive mood dysregulation disorder for ICD-11. *World Psychiatry*, *14*(1), 30-33. doi: 10.1002/wps.20176

Lopes, P. N., Salovey, P., Côté, S., Beers, M., & Petty, R. E. (2005). Emotion regulation abilities and the quality of social interaction. *Emotion,* 5(1), 113. doi: 10.1037/1528-3542.5.1.113

Main, M. (1990). Cross-cultural studies of attachment organization: Recent studies, changing methodologies, and the concept of conditional strategies. *Human Development*, *33*(1), 48-61. doi: 10.1159/000276502

Main, M., & Hesse, E. (1990). Parents' unresolved traumatic experiences are related to infant disorganized attachment status: Is frightened or frightening parental behavior the linking mechanism? In M. Greenberg, D. Cicchetti & E. M. Cummings (Eds.), *Attachment in the Preschool Years,* 161-182. Chicago, IL: University of Chicago

Press.

Main, M., & Solomon, J. (1986). Discovery of an insecure-disorganized/disoriented attachment pattern. In T. B. Brazelton & M. W. Yogman (Eds.), *Affective Development in Infancy* (p. 95–124). Ablex Publishing.

Malik, M., Bigger, J. T., Camm, A. J., Kleiger, R. E., Malliani, A., Moss, A. J., & Schwartz, P. J. (1996). Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. *European Heart Journal*, *17*(3), 354-381. doi: 10.1093/oxfordjournals.eurheartj.a014868

Mandler, G. (2013). A constructivist theory of emotion. In *Psychological and Biological Approaches to Emotion* (pp. 39-62). Psychology Press. doi: 10.4324/9780203761588

Manninen, M., Therman, S., Suvisaari, J., Ebeling, H., Moilanen, I., Huttunen, M., & Joukamaa, M. (2011). Alexithymia is common among adolescents with severe disruptive behavior. *The Journal of Nervous and Mental Disease*, *199*(7), 506-509. doi: 10.1097/NMD.0b013e3182214281.

Marcovitch, S., Leigh, J., Calkins, S. D., Leerks, E. M., O'Brien, M., & Blankson, A. N. (2010). Moderate vagal withdrawal in 3.5-year-old children is associated with optimal performance on executive function tasks. *Developmental Psychobiology*, 52(6), 603-608. doi: 10.1002/dev.20462

Margulies, D. M., Weintraub, S., Basile, J., Grover, P. J., & Carlson, G. A. (2012). Will disruptive mood dysregulation disorder reduce false diagnosis of bipolar disorder in children? *Bipolar Disorders, 14*(5), 488-496. doi: 10.1111/j.1399-5618.2012.01029.x

Mathôt, S., Schreij, D., & Theeuwes, J. (2012). OpenSesame: An open-source, graphical experiment builder for the social sciences. *Behavior Research Methods*, 44(2), 314-324. doi:10.3758/s13428-011-0168-7

Mayes, S. D., Mathiowetz, C., Kokotovich, C., Waxmonsky, J., Baweja, R., Calhoun, S. L., & Bixler, E. O. (2015). Stability of disruptive mood dysregulation disorder symptoms (irritable-angry mood and temper outbursts) throughout childhood and adolescence in a general population sample. *Journal of Abnormal Child Psychology*, *43*(8), 1543-1549. doi: 10.1007/s10802-015-0033-8

Mayes, S. D., Waxmonsky, J. D., Calhoun, S. L., & Bixler, E. O. (2016). Disruptive mood dysregulation disorder symptoms and association with oppositional defiant and other disorders in a general population child sample. *Journal of Child and Adolescent Psychopharmacology*, *26*(2), 101-106. doi: 10.1089/cap.2015.0074

Mazilu, S., Blanke, U., Roggen, D., Tröster, G., Gazit, E., & Hausdorff, J. M. (2013). Engineers meet clinicians: augmenting Parkinson's disease patients to gather information for gait rehabilitation. In *Proceedings of the 4th Augmented Human International Conference* (pp. 124-127). doi: 10.1145/2459236.2459257

McClure, E. B., Pope, K., Hoberman, A. J., Pine, D. S., & Leibenluft, E. (2003). Facial expression recognition in adolescents with mood and anxiety disorders. *American Journal of Psychiatry, 160*(6), 1172-1174. doi: 10.1176/appi.ajp.160.6.1172

McClure, E. B., Treland, J. E., Snow, J., Schmajuk, M., Dickstein, D. P., Towbin, K. E., ... & Leibenluft, E. (2005). Deficits in social cognition and response flexibility in pediatric bipolar disorder. *American Journal of Psychiatry*, *162*(9), 1644-1651. doi: 10.1176/appi.ajp.162.9.1644

McCraty, R., & Shaffer, F. (2015). Heart rate variability: new perspectives on physiological mechanisms, assessment of self-regulatory capacity, and health risk. *Global Advances in Health and Medicine*, *4*(1), 46-61. doi: 10.7453/gahmj.2014.073

McLaughlin, K. A., Hatzenbuehler, M. L., Mennin, D. S., & Nolen-Hoeksema, S. (2011). Emotion dysregulation and adolescent psychopathology: A prospective study. *Behaviour Research and Therapy*, *49*(9), 544-554. doi: 10.1016/j.brat.2011.06.003

Mennin, D. S., Heimberg, R. G., Turk, C. L., & Fresco, D. M. (2005). Preliminary evidence for an emotion dysregulation model of generalized anxiety disorder. *Behaviour Research and Therapy, 43*(10), 1281-1310. doi: 10.1016/j.brat.2004.08.008

Mezzacappa, E., Tremblay, R. E., Kindlon, D., Saul, J. P., Arseneault, L., Seguin, J., ... & Earls, F. (1997). Anxiety, antisocial behavior, and heart rate regulation in adolescent males. *Journal of Child Psychology and Psychiatry*, *38*(4), 457-469. doi: 10.1111/j.1469-7610.1997.tb01531.x

Mikulincer, M. (1988). Reactance and helplessness following exposure to unsolvable problems: The effects of attributional style. *Journal of Personality and Social Psychology*, *54*(4), 679. doi: 10.1037/0022-3514.54.4.679

Mikulincer, M., & Segal, J. (1990). A multidimensional analysis of the experience of loneliness. *Journal of Social and Personal Relationships*, *7*(2), 209-230. doi: 10.1177/0265407590072004

Miller, L., Hlastala, S. A., Mufson, L., Leibenluft, E., Yenokyan, G., & Riddle, M. (2018). Interpersonal psychotherapy for mood and behavior dysregulation: Pilot randomized trial. *Depression and Anxiety*, 35(6), 574-582. doi: 10.1002/da.22761

Miller, J. G., Kahle, S., & Hastings, P. D. (2017). Moderate baseline vagal tone predicts greater prosociality in children. *Developmental Psychology*, *53*(2), 274. doi: 10.1037/dev0000238

Miskovic, V., & Schmidt, L. A. (2010). Frontal brain electrical asymmetry and cardiac vagal tone predict biased attention to social threat. *International Journal of Psychophysiology*, *75*(3), 332-338. doi: 10.1016/j.ijpsycho.2009.12.015

Mitchell, R. L., Elliott, R., Barry, M., Cruttenden, A., & Woodruff, P. W. (2004). Neural response to emotional prosody in schizophrenia and in bipolar affective disorder. *The British Journal of Psychiatry*, *184*(3), 223-230. doi: 10.1192/bjp.184.3.223

Moreno, C., Laje, G., Blanco, C., Jiang, H., Schmidt, A. B., & Olfson, M. (2007). National trends in the outpatient diagnosis and treatment of bipolar disorder in youth. *Archives of General Psychiatry*, 64(9), 1032-1039. doi:10.1001/archpsyc.64.9.1032.

Morningstar, M., Dirks, M. A., & Huang, S. (2017). Vocal cues underlying youth and adult portrayals of socio-emotional expressions. *Journal of Nonverbal Behavior*, 41(2), 155-183. doi: 10.1007/s10919-017-0250-7

Morningstar, M., Dirks, M. A., Rappaport, B. I., Pine, D. S., & Nelson, E. E. (2017). Associations between anxious and depressive symptoms and the recognition of vocal socioemotional expressions in youth. *Journal of Clinical Child & Adolescent Psychology*, 1-10. doi: 10.1080/15374416.2017.1350963

Morris, J. S., Friston, K. J., Büchel, C., Frith, C. D., Young, A. W., Calder, A. J., & Dolan, R. J. (1998). A neuromodulatory role for the human amygdala in processing emotional facial expressions. *Brain: A Journal of Neurology*, *121*(1), 47-57. doi: 10.1093/brain/121.1.47

Morris, J. S., Frith, C. D., Perrett, D. I., Rowland, D., Young, A. W., Calder, A. J., & Dolan, R. J. (1996). A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature*, *383*(6603), 812. doi: 10.1038/383812a0

Morris, J. S., Öhman, A., & Dolan, R. J. (1999). A subcortical pathway to the right amygdala mediating "unseen" fear. *Proceedings of the National Academy of Sciences*, *96*(4), 1680-1685. doi: 10.1073/pnas.96.4.1680

Movahed Abtahi, M., & Kerns, K. A. (2017). Attachment and emotion regulation in middle childhood: changes in affect and vagal tone during a social stress task. *Attachment & Human Development*, *19*(3), 221-242. doi: 10.1080/14616734.2017.1291696

Movius, H. L., & Allen, J. J. (2005). Cardiac vagal tone, defensiveness, and motivational style. *Biological Psychology*, *68*(2), 147-162. doi: 10.1016/j.biopsycho.2004.03.019

Muris, P., Meesters, C., & van den Berg, S. (2003). Internalizing and externalizing problems as correlates of self-reported attachment style and perceived parental rearing in normal adolescents. *Journal of Child and Family Studies*, *12*(2), 171-183. doi: 10.1023/A:1022858715598

Murphy, D., & Cutting, J. (1990). Prosodic comprehension and expression in schizophrenia. *Journal of Neurology, Neurosurgery & Psychiatry*, *53*(9), 727-730. doi: 10.1136/jnnp.53.9.727

Musser, E. D., Backs, R. W., Schmitt, C. F., Ablow, J. C., Measelle, J. R., & Nigg, J. T. (2011). Emotion regulation via the autonomic nervous system in children with attention-deficit/hyperactivity disorder (ADHD). *Journal of Abnormal Child Psychology*, 39(6), 841–852. doi: 10.1007/s10802-011-9499-1

National Advisory Mental Health Council Workgroup on Tasks and Measures for Research Domain Criteria. (2016). *Behavioral assessment methods for RDoC constructs*. Bethesda, MD: National Institute of Mental Health.

Newman, J. P. (1987). Reaction to punishment in extraverts and psychopaths: Implications for the impulsive behavior of disinhibited individuals. *Journal of Research in Personality*, *21*(4), 464-480. doi: 10.1016/0092-6566(87)90033-X

Norman-Haignere, S., Kanwisher, N. G., & McDermott, J. H. (2015). Distinct cortical pathways for music and speech revealed by hypothesis-free voxel decomposition. *Neuron*, *88*(6), 1281-1296. doi: 10.1016/j.neuron.2015.11.035

Obradović, J., Bush, N. R., Stamperdahl, J., Adler, N. E. & Boyce, W. T. (2010). Biological sensitivity to context: The interactive effects of stress reactivity and family adversity on socioemotional behavior and school readiness. *Child Development, 81*, 270–289. doi:10.1111/j.1467-8624.2009.01394.x

Ochsner, K. N. (2008). The social-emotional processing stream: five core constructs and their translational potential for schizophrenia and beyond. *Biological Psychiatry*, *64*(1), 48-61. doi: 10.1016/j.biopsych.2008.04.024

Öhman, A. (2002). Automaticity and the amygdala: Nonconscious responses to emotional faces. *Current Directions in Psychological Science*, *11*(2), 62-66. doi: 10.1111/1467-8721.00169

Ong, A. D., & Bergeman, C. S. (2004). The complexity of emotions in later life. The Journals of Gerontology Series B: *Psychological Sciences and Social Sciences*, 59(3), P117-P122. doi: 10.1093/geronb/59.3.P117

Öztuna, D., Elhan, A. H., & Tüccar, E. (2006). Investigation of four different normality tests in terms of type 1 error rate and power under different distributions. *Turkish Journal of Medical Sciences*, *36*(3), 171-176. Retrieved from https://journals.tubitak.gov.tr/medical/abstract.htm?id=8332

Pan, P. Y., Fu, A. T., & Yeh, C. B. (2018). Aripiprazole/Methylphenidate Combination in Children and Adolescents with Disruptive Mood Dysregulation Disorder and Attention-Deficit/Hyperactivity Disorder: An Open-Label Study. *Journal of Child and Adolescent Psychopharmacology*. doi: 10.1089/cap.2018.0068

Panaite, V., Hindash, A. C., Bylsma, L. M., Small, B. J., Salomon, K., & Rottenberg, J. (2016). Respiratory sinus arrhythmia reactivity to a sad film predicts depression symptom improvement and symptomatic trajectory. *International Journal of Psychophysiology*, *99*, 108-113. doi: 10.1016/j.ijpsycho.2015.12.002

Panksepp, J. (1982). Toward a general psychobiological theory of emotions. *Behavioral and Brain Sciences*, 5(3), 407-422. doi: 10.1017/S0140525X00012759

Panksepp, J. (1998). The periconscious substrates of consciousness: Affective states and the evolutionary origins of the SELF. *Journal of Consciousness Studies, 5*(5-6), 566-582.

Panksepp, J. (2011). The basic emotional circuits of mammalian brains: Do animals have affective lives? *Neuroscience and Biobehavioral Reviews*, 35, 1791–1804. doi: 10.1016/j.neubiorev.2011.08.003

Panksepp, J. (2016). The psycho-neurology of cross-species affective/social neuroscience: Understanding animal affective states as a guide to development of novel psychiatric treatments. *Current Topics in Behavioral Neuroscience*, *30*, 109–125. doi: 10.1007/7854_2016_458

Patterson, C. M., & Newman, J. P. (1993). Reflectivity and learning from aversive events: Toward a psychological mechanism for the syndromes of disinhibition. *Psychological Review, 100*(4), 716. doi: 10.1037/0033-295X.100.4.716

Pereyra, P. M., Zhang, W., Schmidt, M., & Becker, L. E. (1992). Development of myelinated and unmyelinated fibers of human vagus nerve during the first year of life. *Journal of the Neurological Sciences*, 110(1), 107-113. doi: 10.1016/0022-510X(92)90016-E

Pérez-Edgar, K., & Fox, N. A. (2005). A behavioral and electrophysiological study of children's selective attention under neutral and affective conditions. *Journal of Cognition and Development*, 6(1), 89-118. doi: 10.1207/s15327647jcd0601_6

Péron, J., El Tamer, S., Grandjean, D., Leray, E., Travers, D., Drapier, D., ... & Millet, B. (2011). Major depressive disorder skews the recognition of emotional prosody. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 35(4), 987-996. doi: 10.1016/j.pnpbp.2011.01.019

Phelps, E. A. (2006). Emotion and cognition: Insights from studies of the human amygdala. *Annual Review of Psychology*, *57*, 27-53. doi: 10.1146/annurev.psych.56.091103.070234

Phelps, E. A., & LeDoux, J. E. (2005). Contributions of the amygdala to emotion processing: from animal models to human behavior. *Neuron*, *48*(2), 175-187. doi: 10.1016/j.neuron.2005.09.025

Pietromonaco, P. R., & Barrett, L. F. (2000). The internal working models concept: What do we really know about the self in relation to others*? Review of General Psychology*, 4(2), 155. doi: 10.1037/1089-2680.4.2.155

Pietromonaco, P. R., & Powers, S. I. (2015). Attachment and health-related physiological stress processes. *Current Opinion in Psychology*, *1*, 34-39. doi: 10.1016/j.copsyc.2014.12.001

Pine, D. S., Wasserman, G. A., Miller, L., Coplan, J. D., Bagiella, E., Kovelenku, P., . . . Sloan, R. P. (1998). Heart period variability and psychopathology in urban boys at risk for delinquency. *Psychophysiology, 35*, 521–529. doi: 10.1017/S0048577298970846

Plutchik, R. (1980). A general psychoevolutionary theory of emotion. In *Theories of Emotion* (pp. 3-33). Academic press. doi: 10.1016/B978-0-12-558701-3.50007-7

Pogge, D. L., Wayland-Smith, D., Zaccario, M., Borgaro, S., Stokes, J., & Harvey, P. D. (2001). Diagnosis of manic episodes in adolescent inpatients: structured diagnostic procedures compared to clinical chart diagnoses. *Psychiatry Research*, *101*(1), 47–54. doi: 10.1016/S0165-1781(00)00248-1

Poon K. (2018). Hot and Cool Executive Functions in Adolescence: Development and Contributions to Important Developmental Outcomes. *Frontiers in Psychology*, *8*, 2311. doi:10.3389/fpsyg.2017.02311

Porges, S. W. (1985). Respiratory sinus arrhythmia: An index of vagal tone. *Psychophysiology of cardiovascular control: Models, methods, and data*, 437-450.

Porges S.W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage: A Polyvagal Theory. *Psychophysiology*, 32:301–318. doi: 10.1111/j.1469-8986.1995.tb01213.x

Porges, S. W. (1997). Emotion: An Evolutionary By-Product of the Neural Regulation of the Autonomic Nervous Systema. *Annals of the New York Academy of Sciences*, *807*(1), 62-77. doi: 10.1111/j.1749-6632.1997.tb51913.x

Porges, S. W. (1998). Love: An emergent property of the mammalian autonomic nervous system. *Psychoneuroendocrinology* 23, 837-861. doi: 10.1016/S0306-4530(98)00057-2

Porges, S. W. (2001). The polyvagal theory: phylogenetic substrates of a social nervous system. *International Journal of Psychophysiology*, 42(2), 123-146. doi:10.1016/S0167-8760(01)00162-3

Porges, S. W. (2003a). The Polyvagal Theory: phylogenetic contributions to social behavior. A Tribute to Paul MacLean: *The Neurobiological Relevance of Social Behavior*, 79(3), 503–513. doi:10.1016/S0031-9384(03)00156-2

Porges, S. W. (2003b). Social engagement and attachment. *Annals of the New York Academy of Sciences*, 1008(1), 31-47.

Porges, S. W. (2007). The polyvagal perspective. *Biological Psychology*, *74*, 116–143. doi:10.1016/j.biopsycho.2006.06.009

Porges, S. W. (2011). *The Polyvagal Theory: Neurophysiological Foundations of Emotions, Attachment, Communication, and Self-regulation (Norton Series on Interpersonal Neurobiology)*. WW Norton & Company.

Porges, S. W. (2015). Making the World Safe for our Children: Down-regulating Defence and Up-regulating Social Engagement to 'Optimise' the Human Experience. *Children Australia*, *40*(02), 114-123. doi: 10.1017/cha.2015.12

Porges, S. W., & Bohrer, R. E. (1990). *The Analysis of Periodic Processes in Psychophysiological Research*. Unpublished manuscript.

Porges, S. W., Cohn, J. F., Bal, E., & Lamb, D. (2007). The dynamic affect recognition evaluation (DARE) software. *University of Illinois-Chicago*.

Porges S.W., Doussard-Roosevelt J.A., Portales A.L., & Greenspan S.I. (1996). Infant regulation of the vagal "brake" predicts child behavior problems: A psychobiological model of social behavior. *Developmental Psychobiology*, 29:697–712. doi: 10.1002/(SICI)1098-2302(199612)29:8<697::AID-DEV5>3.0.CO;2-O

Porges, S. W., & Furman, S. A. (2011). The early development of the autonomic nervous system provides a neural platform for social behaviour: A polyvagal perspective. *Infant and Child Development, 20*(1), 106-118. doi: 10.1002/icd.688

Porges, S. W., & Lewis, G. F. (2010). The polyvagal hypothesis: common mechanisms mediating autonomic regulation, vocalizations and listening. In *Handbook of Behavioral Neuroscience*, (19), 255-264. Elsevier.

Porges, S. W., Macellaio, M., Stanfill, S. D., McCue, K., Lewis, G. F., Harden, E. R., ... & Heilman, K. J. (2013). Respiratory sinus arrhythmia and auditory processing in autism: Modifiable deficits of an integrated social engagement system? *International Journal of Psychophysiology, 88*(3), 261-270. doi: 10.1016/j.ijpsycho.2012.11.009

Porter, F. L., Porges, S. W., & Marshall, R. E. (1988). Newborn pain cries and vagal tone: Parallel changes in response to circumcision. *Child Development*, 495-505. doi: 10.2307/1130327

Posner, M. I. (1988). Structures and function of selective attention. In T. Boll & B. K. Bryant (Eds.), *The Master Lecture Series. Clinical Neuropsychology and Brain Function: Research, Measurement, and Practice*, 7: 173-202. Washington, DC, US: American Psychological Association.

Posner, M. I., & Cohen, Y. (1984). Components of visual orienting. *Attention and Performance X: Control of Language Processes*, *32*, 531-556.

Prencipe, A., Kesek, A., Cohen, J., Lamm, C., Lewis, M. D., & Zelazo, P. D. (2011). Development of hot and cool executive function during the transition to adolescence. *Journal of Experimental Child Psychology*, *108*(3), 621-637. doi: 10.1016/j.jecp.2010.09.008

Prinz, J. J. (2004a). *Gut reactions: A perceptual theory of emotion*. Oxford University Press. Retrieved from https://books.google.co.za/books?hl=en&lr=&id=v0mm6MQM770C&oi=fnd&pg=PR11&ots=QLelTRJhYo&sig=XzSgQeBOxowf4Btvwztph7PaDQI#v=onepage&q&f=false

Prinz, J. J. (2004b). Embodied emotions. In R. C. Solomon (Ed.), *Thinking about Feeling: Contemporary Philosophers on Emotions*, 44–58. Retrieved from https://books.google.co.za/books?id=T4hJCAAAQBAJ&printsec=frontcover&dq=Thinking+about+feeling:+Contemporary+philosophers+on+emotions+(pp.+28%E2%80%9343).+New+York:+Oxford+University+Press.&hl=en&sa=X&ved=0ahUKEwjw-9Dlr_jlAhUWTxUIHYsCD8UQ6AEIQzAD#v=onepage&q&f=false

Quintana, D. S., Alvares, G. A., & Heathers, J. A. J. (2016). Guidelines for Reporting Articles on Psychiatry and Heart rate variability (GRAPH): recommendations to advance research communication. *Translational Psychiatry*, *6*(5), e803. doi: 10.1038/tp.2016.73

Quintana, D. S., Kemp, A. H., Alvares, G. A., & Guastella, A. J. (2013). A role for autonomic cardiac control in the effects of oxytocin on social behavior and psychiatric illness. *Frontiers in Neuroscience*, *7*, 48. doi: 10.3389/fnins.2013.00048

Rapcsak, S. Z., Galper, S. R., Comer, J. F., Reminger, S. L., Nielsen, L., Kaszniak, A. W., ... & Cohen, R. A. (2000). Fear recognition deficits after focal brain damage: a cautionary note. *Neurology*, *54*(3), 575-575. doi: 10.1212/WNL.54.3.575

Rash, J. A. & Aguirre-Camacho, A. (2012). Attention-deficit hyperactivity disorder and cardiac vagal control: A systematic review. *ADHD Attention Deficit and Hyperactivity Disorders*, *4*, 167–177. doi:10.1007/s12402-012-0087-1

Regal, P. J. (1978). Behavioral differences between reptiles and mammals: an analysis of activity and mental capabilities. In N. Greenberg & P. D. MacLean (Eds.), *Behavior and Neurology of Lizards*, 183-202.

Reiman, E. M., Lane, R. D., Ahern, G. L., Schwartz, G. E., Davidson, R. J., Friston, K. J., ... & Chen, K. (1997). Neuroanatomical correlates of externally and internally generated human emotion. *American Journal of Psychiatry*, *154*(7), 918-925. doi: 10.1176/ajp.154.7.918

Reitz, S., Kluetsch, R., Niedtfeld, I., Knorz, T., Lis, S., Paret, C., ... & Bohus, M. (2015). Incision and stress regulation in borderline personality disorder: neurobiological mechanisms of self-injurious behaviour. *The British Journal of Psychiatry*, *207*(2), 165-172. doi: 10.1192/bjp.bp.114.153379

Rich, B. A., Brotman, M. A., Dickstein, D. P., Mitchell, D. G., Blair, R. J. R., & Leibenluft, E. (2010a). Deficits in attention to emotional stimuli distinguish youth with severe mood dysregulation from youth with bipolar disorder. *Journal of Abnormal Child Psychology*, *38*(5), 695-706. doi: 10.1007/s10802-010-9395-0

Rich, B. A., Grimley, M. E., Schmajuk, M., Blair, K. S., Blair, R. J. R., & Leibenluft, E. (2008). Face emotion labeling deficits in children with bipolar disorder and severe mood dysregulation. *Development and Psychopathology*, 20(2), 529–546. doi:10.1017/S0954579408000266

Rich, B. A., Holroyd, T., Carver, F. W., Onelio, L. M., Mendoza, J. K., Cornwell, B. R., ... & Leibenluft, E. (2010b). A preliminary study of the neural mechanisms of frustration in pediatric bipolar disorder using magnetoencephalography. *Depression and Anxiety*, 27(3), 276-286. doi: 10.1002/da.20649

Rich, B. A., Schmajuk, M., Perez-Edgar, K. E., Fox, N. A., Pine, D. S., & Leibenluft, E. (2007). Different Psychophysiological and Behavioral Responses Elicited by Frustration in Pediatric Bipolar Disorder and Severe Mood Dysregulation. *American Journal of Psychiatry*, 164(2), 309–317. doi: 10.1176/ajp.2007.164.2.309

Rich, B. A., Schmajuk, M., Perez-Edgar, K. E., Pine, D. S., Fox, N. A., & Leibenluft, E. (2005). The impact of reward, punishment, and frustration on attention in pediatric bipolar disorder. *Biological Psychiatry*, 58(7), 532-539. doi: 10.1016/j.biopsych.2005.01.006

Riglin, L., Eyre, O., Cooper, M., Collishaw, S., Martin, J., Langley, K., ... & O'Donovan, M. C. (2017). Investigating the genetic underpinnings of early-life irritability. *Translational Psychiatry*, *7*(9), e1241. doi: 10.1038/tp.2017.212

Riniolo, T., & Porges, S. W. (1997). Inferential and descriptive influences on measures of respiratory sinus arrhythmia: sampling rate, R-wave trigger accuracy, and variance estimates. *Psychophysiology*, *34*(5), 613-621.

Roberson-Nay, R., Leibenluft, E., Brotman, M. A., Myers, J., Larsson, H., Lichtenstein, P., & Kendler, K. S. (2015). Longitudinal stability of genetic and environmental influences on irritability: from childhood to young adulthood. *American Journal of Psychiatry, 172*(7), 657-664. doi: 10.1176/appi.ajp.2015.14040509

Robinson, J. (1995). Startle. *The Journal of Philosophy*, 92(2), 53-74. doi:10.2307/2940940

Robinson, J. (2004). Emotion: Biological fact or social construction? In R. C. Solomon (Ed.), *Thinking about Feeling: Contemporary Philosophers on Emotions*, 28–43. Retrieved from https://books.google.co.za/books?id=T4hJCAAAQBAJ&printsec=frontcover&dq=Thinking+about+feeling:+Contemporary+philosophers+on+emotions+(pp.+28%E2%80%9343).+New+York:+Oxford+University+Press.&hl=en&sa=X&ved=0ahUKEwjw-9Dlr_jlAhUWTxUIHYsCD8UQ6AEIQzAD#v=onepage&q&f=false

Roiser, J. P., & Sahakian, B. J. (2013). Hot and cold cognition in depression. *CNS Spectrums*, *18*(3), 139-149. doi: 10.1017/S1092852913000072

Rolls, E. T. (2002). A theory of emotion, its functions, and its adaptive value. In *Memory and Emotion*, 349-360. doi: 10.1142/9789812776563_0028

Romanski, L. M., & LeDoux, J. E. (1992). Equipotentiality of thalamo-amygdala and thalamo-cortico-amygdala circuits in auditory fear conditioning. *Journal of Neuroscience*, *12*(11), 4501-4509. Retrieved from http://www.jneurosci.org/content/12/11/4501.long

Rombouts, S. A., Machielsen, W., Witter, M. P., Barkhof, F., Lindeboom, J., & Scheltens, P. (1997). Visual association encoding activates the medial temporal lobe: a functional magnetic resonance imaging study. *Hippocampus*, 7(6), 594-601. doi: 10.1002/(SICI)1098-1063(1997)7:6<594::AID-HIPO2>3.0.CO;2-F

Rorden, C., & Hanayik, T. (2014). StimSync: Open-source hardware for behavioral and MRI experiments. *Journal of Neuroscience Methods*, *227*, 90-99. doi: 10.1016/j.jneumeth.2013.11.017

Rosato, N. S., Correll, C. U., Pappadopulos, E., Chait, A., Crystal, S., Jensen, P. S., & Treatment of Maladaptive Aggressive in Youth Steering Committee. (2012). Treatment of maladaptive aggression in youth: CERT guidelines II. Treatments and ongoing management. *Pediatrics*, peds-2010. doi: 10.1542/peds.2010-1360

Rose, J., Gilbert, L., & Richards, V. (2015). *Health and Well-being in Early Childhood*. SAGE.

Rosenman, R., Tennekoon, V., & Hill, L. G. (2011). Measuring bias in self-reported data. *International Journal of Behavioural & Healthcare Research*, *2*(4), 320. doi: 10.1504/IJBHR.2011.043414

Rottenberg, J. (2007). Cardiac vagal tone in depression: A critical analysis. *Biological Psychology*, 74, 200–211. doi:10.1016/j.biopsycho.2005.08.010

Rottenberg, J., Salomon, K., Gross, J.J., Gotlib, I.H. (2005). Vagal withdrawal to a sad film predicts subsequent recovery from depression. *Psychophysiology*, 42, 277-281. doi: 10.1111/j.1469-8986.2005.00289.x

Rubin, K. H., Coplan, R. J., Fox, N. A., & Calkins, S. D. (1995). Emotionality, emotion regulation, and preschoolers' social adaptation. *Development and Psychopathology*, 7(1), 49-62. doi: 10.1017/S0954579400006337

Rudzki, L., & Szulc, A. (2018). "Immune Gate" of Psychopathology - The Role of Gut Derived Immune Activation in Major Psychiatric Disorders. *Frontiers in Psychiatry*, *9*, 205. doi: 10.3389/fpsyt.2018.00205

Russell, J. A. (2009). Emotion, core affect, and psychological construction. *Cognition and Emotion*, *23*(7), 1259-1283. doi: 10.1080/02699930902809375

Saarni, C. (1999). *The development of emotional competence*. Guilford Press.

Saarni, C., Campos, J. J., Camras, L. A., & Witherington, D. (2006). Emotional Development: Action, Communication, and Understanding. In N. Eisenberg, W. Damon, & R. M. Lerner (Eds.), *Handbook of Child Psychology*: *Vol. 3, Social, Emotional, and Personality Development* (6th ed.), 226-299. Hoboken, NJ: John Wiley. do1i: 10.1002/9780470147658.chpsy0305

Sahar, T., Shalev, A. Y., & Porges, S. W. (2001). Vagal modulation of responses to mental challenge in posttraumatic stress disorder. *Biological Psychiatry*, *49*(7), 637-643. doi: 10.1016/S0006-3223(00)01045-3

Santucci, A. K., Silk, J. S., Shaw, D. S., Gentzler, A., Fox, N. A., & Kovacs, M. (2008). Vagal tone and temperament as predictors of emotion regulation strategies in young children. *Developmental Psychobiology*, *50*(3), 205-216. doi:

SAS Institute Inc., *SAS Software, version 9.3 for Windows*, Cary, NC, USA: SAS Institute Inc. (2002-2010).

Saul, J. (1990). Beat-To-Beat Variations of Heart Rate Reflect Modulation of Cardiac Autonomic Outflow. *Physiology*, 5(1), 32–37. Retrieved from http://physiologyonline.physiology.org/content/5/1/32.full-text.pdf+html

Sauter, D. A., Panattoni, C., & Happé, F. (2013). Children's recognition of emotions from vocal cues. *British Journal of Developmental Psychology*, 31, 97–113. doi:10.1111/j.2044-835X.2012.02081.x

Savage, J., Verhulst, B., Copeland, W., Althoff, R. R., Lichtenstein, P., & Roberson-Nay, R. (2015). A genetically informed study of the longitudinal relation between irritability and anxious/depressed symptoms. *Journal of the American Academy of Child & Adolescent Psychiatry*, *54*(5), 377-384. doi: 10.1016/j.jaac.2015.02.010

Schachter, J. (1957). Pain, fear, and anger in hypertensives and normotensives: A psychophysiological study. *Psychosomatic Medicine*, *19*(1), 17-29. doi: 10.1097/00006842-195701000-00003

Schachter, S., & Singer, J. (1962). Cognitive, social, and physiological determinants of emotional state. *Psychological Review*, 69(5), 379. doi: 10.1037/h0046234

Schenkel, L. S., Pavuluri, M. N., Herbener, E. S., Harral, E. M., & Sweeney, J. A. (2007). Facial emotion processing in acutely ill and euthymic patients with pediatric bipolar disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, *46*(8), 1070-1079. doi: 10.1097/chi.0b013e3180600fd6

Scherer, K. R. (1984). On the nature and function of emotion: A component process approach. *Approaches to Emotion*, 2293, 317. doi: 10.1177/053901882021004001

Scherer, K. R. (2003). Vocal communication of emotion: A review of research paradigms. *Speech Communication*, 40, 227–256. doi:10.1016/S0167-6393(02)00084-5

Schmolck, H., & Squire, L. R. (2001). Impaired perception of facial emotions following bilateral damage to the anterior temporal lobe. *Neuropsychology*, *15*(1), 30. doi: 10.1037/0894-4105.15.1.30

Schoorl, J., van Rijn, S., de Wied, M., Van Goozen, S., & Swaab, H. (2016a). Emotion regulation difficulties in boys with oppositional defiant disorder/conduct disorder and the relation with comorbid autism traits and attention deficit traits. *PloS one*, *11*(7). doi: 10.1371/journal.pone.0159323

Schoorl, J., Van Rijn, S., de Wied, M., Van Goozen, S. H., & Swaab, H. (2016b). Variability in emotional/behavioral problems in boys with oppositional defiant disorder or conduct disorder: the role of arousal. *European child & adolescent psychiatry*, *25*(8), 821-830. doi: 10.1007/s00787-015-0790-5

Schroeder, U., Hennenlotter, A., Erhard, P., Haslinger, B., Stahl, R., Lange, K. W., & Ceballos-Baumann, A. O. (2004). Functional Neuroanatomy of Perceiving Surprised Faces. *Human Brain Mapping, 23,* 181-187. doi: 10.1002/hbm.20057

Scott, N., Green, C., & Fairley, S. (2016). Investigation of the use of eye tracking to examine tourism advertising effectiveness. *Current Issues in Tourism, 19*(7), 634-642. doi: 10.1080/13683500.2014.1003797

Scott, B. G., & Weems, C. F. (2014). Resting vagal tone and vagal response to stress: associations with anxiety, aggression, and perceived anxiety control among youths. *Psychophysiology, 51*(8), 718-727. doi: 10.1111/psyp.12218

Sergerie, K., Chochol, C., & Armony, J. L. (2008). The role of the amygdala in emotional processing: a quantitative meta-analysis of functional neuroimaging studies. *Neuroscience & Biobehavioral Reviews*, *32*(4), 811-830. doi: 10.1016/j.neubiorev.2007.12.002

Sheybani, H., Mikaeili, N., & Narimani, M. (2018). The Efficacy of Emotion Regulation Training on Stress, Anxiety, Depression and Irritability of the Students with Disruptive Mood Dysregulation Disorder. *Iranian Journal of Psychiatric Nursing*, *5*(6), 37-44. doi: 10.21859/ijpn-05066

Shields, A., & Cicchetti, D. (1997). Emotion regulation among school-age children: The development and validation of a new criterion Q-sort scale. *Developmental Psychology*, 33(6), 906. doi: 10.1037//0012-1649.33.6.906

Shinya, Y., Kawai, M., Niwa, F., & Myowa-Yamakoshi, M. (2016). Associations between respiratory arrhythmia and fundamental frequency of spontaneous crying in preterm and term infants at term-equivalent age. *Developmental Psychobiology*, *58*(6), 724-733. doi: 10.1002/dev.21412

Singh, N. C., & Theunissen, F. E. (2003). Modulation spectra of natural sounds and ethological theories of auditory processing. *The Journal of the Acoustical Society of America, 114*(6), 3394-3411. doi: 10.1121/1.1624067

Shipman, K., Schneider, R., & Brown, A. (2004). Emotion dysregulation and psychopathology. In M. Beauregard (Ed.), *Consciousness, Emotional Self-Regulation and the Brain*, 61–85. Amsterdam: John Benjamins.

Sloan, R. P., Shapiro, P. A., Bigger, J. T., Bagiella, M., Steinman, R. C. & Gorman, J. M. (1994). Cardiac autonomic control and hostility in healthy subjects. *The American Journal of Cardiology*, *74*, 298–300. doi:10.1016/0002-9149(94)90382-4

Smith, J. D., Woodhouse, S. S., Clark, C. A., & Skowron, E. A. (2016). Attachment status and mother–preschooler parasympathetic response to the strange situation procedure. *Biological Psychology, 114*, 39-48. doi: 10.1016/j.biopsycho.2015.12.008

Sperling, R. A., Bates, J. F., Cocchiarella, A. J., Schacter, D. L., Rosen, B. R., & Albert, M. S. (2001). Encoding novel face-name associations: A functional MRI study. *Human Brain Mapping*, 14(3), 129-139. doi: 10.1002/hbm.1047

Sprengelmeyer, R., Rausch, M., Eysel, U. T., & Przuntek, H. (1998). Neural structures associated with recognition of facial expressions of basic emotions. *Proceedings of the Royal Society of London B: Biological Sciences, 265*(1409), 1927-1931. doi: 10.1098/rspb.1998.0522

Stern, C. E., Corkin, S., González, R. G., Guimaraes, A. R., Baker, J. R., Jennings, P. J., ... & Rosen, B. R. (1996). The hippocampal formation participates in novel picture encoding: evidence from functional magnetic resonance imaging. *Proceedings of the National Academy of Sciences, 93*(16), 8660-8665. doi: 10.1073/pnas.93.16.8660

Sterne, J. A., & Smith, G. D. (2001). Sifting the evidence - what's wrong with significance tests? *Physical Therapy*, *81*(8), 1464-1469.

Stevenson-Hinde, J., & Marshall, P. J. (1999). Behavioral inhibition, heart period, and respiratory sinus arrhythmia: An attachment perspective. *Child Development*, *70*(4), 805-816. doi: 10.1111/1467-8624.00058

Stewart, A. M., Lewis, G. F., Heilman, K. J., Davila, M. I., Coleman, D. D., Aylward, S. A., & Porges, S. W. (2013). The covariation of acoustic features of infant cries and autonomic state. *Physiology & Behavior*, *120*, 203-210. doi: 10.1016/j.physbeh.2013.07.003

Stigler, K. A., & McDougle, C. J. (2008). Pharmacotherapy of irritability in pervasive developmental disorders. *Child and Adolescent Psychiatric Clinics of North America*, *17*(4), 739-752. doi: 10.1016/j.chc.2008.06.002

Stoddard, J., Sharif-Askary, B., Harkins, E. A., Frank, H. R., Brotman, M. A., Penton-Voak, I. S., ... & Leibenluft, E. (2016). An open pilot study of training hostile interpretation bias to treat disruptive mood dysregulation disorder. *Journal of Child and Adolescent Psychopharmacology*, *26*(1), 49-57. doi: 10.1089/cap.2015.0100

Stringaris, A., Cohen, P., Pine, D. S., & Leibenluft, E. (2009). Adult outcomes of youth irritability: a 20-year prospective community-based study. *The American Journal of Psychiatry*, *166*(9), 1048-1054. doi: 10.1176/appi.ajp.2009.08121849

Stringaris, A., & Goodman, R. (2009). Longitudinal Outcome of Youth Oppositionality: Irritable, Headstrong, and Hurtful Behaviors Have Distinctive Predictions. *Journal of the American Academy of Child & Adolescent Psychiatry*, *48*(4), 404–412. doi:10.1097/CHI.0b013e3181984f30

Suess, P. E., Porges, S. W., & Plude, D. J. (1994). Cardiac vagal tone and sustained attention in school-age children. *Psychophysiology*, *31*(1), 17-22. doi: 10.1111/j.1469-8986.1994.tb01020.x

Sun, X. (2002-2016). Pitch determination algorithm. Retrieved from http://www.mathworks.com/matlabcentral/fileexchange/1230-pitch-determination-algorithm), MATLAB Central File Exchange. Retrieved Apr 9, 2016.

Surguladze, S. A., El-Hage, W., Dalgleish, T., Radua, J., Gohier, B., & Phillips, M. L. (2010). Depression is associated with increased sensitivity to signals of disgust: a functional magnetic resonance imaging study. *Journal of Psychiatric Research*, *44*(14), 894-902. doi: 10.1016/j.jpsychires.2010.02.010

Sutton, S. K., & Davidson, R. J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science*, *8*(3), 204-210. doi: 10.1111/j.1467-9280.1997.tb00413.x

Tamir, M. (2011). The maturing field of emotion regulation. *Emotion Review*, *3*(1), 3-7. doi: 10.1177/1754073910388685

Tapia, V., & John, R. M. (2018). Disruptive Mood Dysregulation Disorder. *The Journal for Nurse Practitioners*, *14*(8), 573-578. doi: 0.1016/j.nurpra.2018.07.007

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. *Circulation*, *93*, 1043–1065.

Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. *Biological Psychiatry*, *39*(4), 255-266. doi: 10.1016/0006-3223(95)00136-0

Thayer, J. F., Hansen, A. L., Saus-Rose, E., & Johnsen, B. H. (2009). Heart rate variability, prefrontal neural function, and cognitive performance: the neurovisceral integration perspective on self-regulation, adaptation, and health. *Annals of Behavioral Medicine*, *37*(2), 141-153. doi: 10.1007/s12160-009-9101-z

Theunissen, F. E., & Elie, J. E. (2014). Neural processing of natural sounds. *Nature Reviews Neuroscience*, *15*(6), 355. doi: 10.1038/nrn3731

Thomas, L. A., Brotman, M. A., Bones, B. L., Chen, G., Rosen, B. H., Pine, D. S., & Leibenluft, E. (2014). Neural circuitry of masked emotional face processing in youth with bipolar disorder, severe mood dysregulation, and healthy volunteers. *Developmental Social and Affective Neuroscience*, 8(0), 110–120. doi: 10.1016/j.dcn.2013.09.007

Thomas, L. A., Brotman, M. A., Muhrer, E. J., Rosen, B. H., Bones, B. L., Reynolds, R. C., ... & Leibenluft, E. (2012). Parametric modulation of neural activity by emotion in youth with bipolar disorder, youth with severe mood dysregulation, and healthy volunteers. *Archives of General Psychiatry*, *69*(12), 1257-1266. doi: 10.1001/archgenpsychiatry.2012.913

Thompson, R. A. (1991). Emotional regulation and emotional development. *Educational Psychology Review*, 3(4), 269-307. doi: 10.1007/BF01319934

Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development*, 59(2-3), 25-52. doi: 10.1111/j.1540-5834.1994.tb01276.x

Thompson, R. A. (2011). Emotion and emotion regulation: Two sides of the developing coin. *Emotion Review*, *3*(1), 53-61. doi: 10.1177/1754073910380969

Thompson, R. A., Lewis, M. D., & Calkins, S. D. (2008). Reassessing emotion regulation. *Child Development Perspectives*, *2*(3), 124-131. doi: 10.1111/j.1750-8606.2008.00054.x

Tomkins, S. S. (1962). *Affect, Imagery, Consciousness. Vol. 1, The positive affects*. New York: Springer. doi: 10.1037/14351-000

Tourian, L., LeBoeuf, A., Breton, J. J., Cohen, D., Gignac, M., Labelle, R., ... & Renaud, J. (2015). Treatment options for the cardinal symptoms of disruptive mood dysregulation disorder. *Journal of the Canadian Academy of Child and Adolescent Psychiatry*, *24*(1), 41. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4357333/

Tull, M. T., & Aldao, A. (2015). Editorial overview: New directions in the science of emotion regulation. *Current Opinion in Psychology, 1(*3). doi: 10.1016/j.copsyc.2015.03.009

Uekermann, J., Abdel-Hamid, M., Lehmkaemper, C., Vollmoeller, W., & Daum, I. (2008). Perception of affective prosody in major depression: a link to executive functions? *Journal of the International Neuropsychological Society*, *14*(4), 552-561. doi: 10.1017/S1355617708080740

Unwin, L. M., Bruz, I., Maybery, M. T., Reynolds, V., Ciccone, N., Dissanayake, C., ... & Whitehouse, A. J. (2017). Acoustic properties of cries in 12-month old infants at high-risk of autism spectrum disorder. *Journal of Autism and Developmental Disorders*, *47*(7), 2108-2119. doi: 10.1007/s10803-017-3119-z

Valent, Paul. (1998). *From Survival to Fulfillment: A Framework for the Life-trauma Dialectic*. Psychology Press.

Van Der Kolk, B. (2014). *The Body Keeps the Score: Brain, Mind, and Body in the Healing of Trauma*. Penguin.

Van Dinteren, R., Arns, M., Jongsma, M. L. A., & Kessels, R. P. C. (2014). Combined frontal and parietal P300 amplitudes indicate compensated cognitive processing across the lifespan. *Frontiers in Aging Neuroscience*, *6*, 294. doi: 10.3389/fnagi.2014.00294

Van Leeuwen, J., Vink, M., Joëls, M., Kahn, R. S., & Vinkers, C. H. (2016). Psychosocial stress and emotion processing in siblings of schizophrenia patients. *European Neuropsychopharmacology*, *26*, S306-S307. doi: 10.1016/S0924-977X(16)31211-1

Vassar, M., & Holzmann, M. (2013). The retrospective chart review: important methodological considerations. *Journal of Educational Evaluation for Health Professions*, *10*, 12. doi: 10.3352/jeehp.2013.10.12

Vuilleumier, P. (2005). How brains beware: neural mechanisms of emotional attention. *Trends in Cognitive Sciences*, *9*(12), 585-594. doi: 10.1016/j.tics.2005.10.011

Vuilleumier, P., Armony, J. L., Driver, J., & Dolan, R. J. (2001). Effects of attention and emotion on face processing in the human brain: an event-related fMRI study. *Neuron*, *30*(3), 829-841. doi: 10.1016/S0896-6273(01)00328-2

Vuilleumier, P., Richardson, M. P., Armony, J. L., Driver, J., & Dolan, R. J. (2004). Distant influences of amygdala lesion on visual cortical activation during emotional face processing. *Nature Neuroscience*, *7*(11), 1271-1278. doi: 10.1038/nn1341

Wallace, J. F., & Newman, J. P. (1990). Differential effects of reward and punishment cues on response speed in anxious and impulsive individuals. *Personality and Individual Differences*, *11*(10), 999-1009. doi: 10.1016/0191-8869(90)90127-D

Watkins, L. L., Grossman, P., Krishnan, R., & Sherwood, A. (1998). Anxiety and vagal control of heart rate. *Psychosomatic Medicine*, 60(4), 498-502. doi: 10.1097/00006842-199807000-00018

Watson, L. (2015). Is Bipolar on the rise? *Moneyweb*. Retrieved from http://www.moneyweb.co.za/news/economy/is-bipolar-on-the-rise/

Watson, D., Wiese, D., Vaidya, J., & Tellegen, A. (1999). The two general activation systems of affect: Structural findings, evolutionary considerations, and psychobiological evidence. *Journal of Personality and Social Psychology, 76*(5), 820. doi: 10.1037/0022-3514.76.5.820

Waxmonsky, J., Pelham, W. E., Gnagy, E., Cummings, M. R., O'Connor, B., Majumdar, A., ... & Fabiano, G. A. (2008). The efficacy and tolerability of methylphenidate and behavior modification in children with attention-deficit/hyperactivity disorder and severe mood dysregulation. *Journal of Child and Adolescent Psychopharmacology*, *18*(6), 573-588. doi: 10.1089/cap.2008.065

Waxmonsky, J. G., Wymbs, F. A., Pariseau, M. E., Belin, P. J., Waschbusch, D. A., Babocsai, L., ... & Pelham, W. E. (2013). A novel group therapy for children with ADHD and severe mood dysregulation. *Journal of Attention Disorders*, *17*(6), 527-541. doi: 10.1177/1087054711433423

Weinberg, A., Klonsky, E. D., & Hajcak, G. (2009). Autonomic impairment in borderline personality disorder: a laboratory investigation. *Brain and Cognition*, *71*(3), 279-286. doi: 10.1016/j.bandc.2009.07.014

Weisfeld, G. E., & Goetz, S. M. (2013). Applying evolutionary thinking to the study of emotion. *Behavioral Sciences*, *3*(3), 388-407. doi: 10.3390/bs3030388

Weymar, M., & Schwabe, L. (2016). Amygdala and Emotion: The Bright Side of It. *Frontiers in Neuroscience, 10*, 224. doi:10.3389/fnins.2016.00224

Whalen, P. J. (1998). Fear, vigilance, and ambiguity: Initial neuroimaging studies of the human amygdala. *Current Directions in Psychological Science*, *7*(6), 177-188. doi: 10.1111/1467-8721.ep10836912

Whalen, P. J., Rauch, S. L., Etcoff, N. L., McInerney, S. C., Lee, M. B., & Jenike, M. A. (1998). Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *Journal of Neuroscience*, *18*(1), 411-418. Retrieved from http://www.jneurosci.org/content/18/1/411.long

Whalen, P. J., Shin, L. M., McInerney, S. C., Fischer, H., Wright, C. I., & Rauch, S. L. (2001). A functional MRI study of human amygdala responses to facial expressions of fear versus anger. *Emotion*, *1*(1), 70. doi: 10.1037/1528-3542.1.1.70

White, H., & Sabarwal, S. (2014). Quasi-experimental design and methods. *Methodological Briefs: Impact Evaluation*, *8*, 1-16. Retrieved from https://ideas.repec.org/p/ucf/metbri/innpub753.html

Wiggins, J. L., Mitchell, C., Stringaris, A., & Leibenluft, E. (2014). Developmental trajectories of irritability and bidirectional associations with maternal depression. *Journal of the American Academy of Child & Adolescent Psychiatry*, *53*(11), 1191-1205. doi: 10.1016/j.jaac.2014.08.005

Willemen, A. M., Goossens, F. A., Koot, H. M., & Schuengel, C. (2008). Physiological reactivity to stress and parental support: Comparison of clinical and non-clinical adolescents. *Clinical Psychology & Psychotherapy*, *15*(5), 340. doi: 10.1002/cpp.578

Winston, J. S., Strange, B. A., O'Doherty, J., & Dolan, R. J. (2002). Automatic and intentional brain responses during evaluation of trustworthiness of faces. *Nature Neuroscience*, *5*(3), 277-283. doi: 10.1038/nn816

Winters, D. E., Fukui, S., Leibenluft, E., & Hulvershorn, L. A. (2018). Improvements in Irritability with Open-Label Methylphenidate Treatment in Youth with Comorbid Attention Deficit/Hyperactivity Disorder and Disruptive Mood Dysregulation Disorder. *Journal of Child and Adolescent Psychopharmacology*. doi: 10.1089/cap.2017.0124

Witherington, D.C., Campos, J.J., & Hertenstein, M.J. (2001). Principles of emotion and its development. In G. Bremner & A. Fogel (Eds.), *Blackwell Handbook of Infant Development*, 427 – 464. Oxford, England: Blackwell.

Witherington, D. C., & Crichton, J. A. (2007). Frameworks for understanding emotions and their development: functionalist and dynamic systems approaches. *Emotion, 7*(3), 628. doi: 10.1037/1528-3542.7.3.628

World Health Organisation (1992). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: World Health Organisation.

Wozniak, J., Biederman, J., Kiely, K., Ablon, J. S., Faraone, S. V., Mundy, E., & Mennin, D. (1995). Mania-like symptoms suggestive of childhood-onset bipolar disorder in clinically referred children. *Journal of the American Academy of Child & Adolescent Psychiatry, 34*(7), 867-876. doi: 10.1097/00004583-199507000-00010

Yang, T. T., Menon, V., Eliez, S., Blasey, C., White, C. D., Reid, A. J., ... & Reiss, A. L. (2002). Amygdalar activation associated with positive and negative facial expressions. *Neuroreport*, *13*(14), 1737-1741. doi: 10.1097/00001756-200210070-00009

Zajonc, R. B. (1980). Feeling and thinking: Preferences need no inferences. *American Psychologist*, *35*(2), 151. doi: 10.1037%2F0003-066X.35.2.151

Zajonc, R. (1984). On the primacy of affect. *The American Psychologist*, *39*(2), 117-123. doi: 10.1037/0003-066X.39.2.117

Zahn-Waxler, C., Crick, N. R., Shirtcliff, E. A., & Woods, K. E. (2015). The origins and development of psychopathology in females and males. *Developmental Psychopathology: Volume One: Theory and Method*, 76-138. doi: 10.1002/9780470939383.ch4

Zelazo, P. D., & Carlson, S. M. (2012). Hot and cool executive function in childhood and adolescence: Development and plasticity. *Child Development Perspectives*, *6*(4), 354-360. doi: 10.1111/j.1750-8606.2012.00246.x

Zepf, F. D., Biskup, C. S., Holtmann, M. & Runions, K. C. (2015). Disruptive mood dysregulation disorder. *IACAPAP e-Textbook of Child and Adolescent Mental Health.* In J. M. Rey (ed.). Geneva: International Association for Child and Adolescent Psychiatry and Allied Professions (IACAPAP). Retrieved from https://iacapap.org/content/uploads/E.3-MOOD-DYSREGULATION-2016.pdf

Zepf, F. D., & Holtmann, M. (2012). Disruptive mood dysregulation disorder. *IACAPAP e-Textbook of Child and Adolescent Mental Health. Geneva, Switzerland: International Association for Child and Adolescent Psychiatry and Allied Professions*, 1-11. Retrieved from https://iacapap.org/content/uploads/E.3-MOOD-DYSREGULATION-2016.pdf

Zhao, K., Yan, W.-J., Chen, Y.-H., Zuo, X.-N., & Fu, X. (2013). Amygdala Volume Predicts Inter-Individual Differences in Fearful Face Recognition. *PLoS ONE, 8*(8), e74096. doi: 10.1371/journal.pone.0074096

Zhao, K., Zhao, J., Zhang, M., Cui, Q., & Fu, X. (2017). Neural Responses to Rapid Facial Expressions of Fear and Surprise. *Frontiers in Psychology, 8,* 761. Doi: 10.3389/fpsyg.2017.00761

Zimmermann, G. (2006). Delinquency in male adolescents: The role of alexithymia and family structure. *Journal of Adolescence, 29*(3), 321-332. doi: 10.1016/j.adolescence.2005.08.001

Zimmermann, P., & Iwanski, A. (2014). Emotion regulation from early adolescence to emerging adulthood and middle adulthood: Age differences, gender differences, and emotion-specific developmental variations. *International Journal of Behavioral Development*, 38(2), 182-194. doi: 10.1177/0165025413515405

Zorrilla, L. T. E., Jeste, D. V., Paulus, M., & Brown, G. G. (2003). Functional abnormalities of medial temporal cortex during novel picture learning among patients with chronic schizophrenia. *Schizophrenia Research*, 59(2), 187-198. doi: 10.1016/S0920-9964(01)00340-1

Zucker, N., Mauro, C., Craske, M., Wagner, H. R., Datta, N., Hopkins, H., ... & Mayer, E. (2017). Acceptance-based interoceptive exposure for young children with functional abdominal pain. *Behaviour Research and Therapy*, *97*, 200-212. doi: 10.1016/j.brat.2017.07.009

Appendix A: Control Group Parent Letter and Consent Form

Psychology
School of Human & Community Development
University of the Witwatersrand
Private Bag 3, Wits, 2050
Tel: 011 717 4503 Fax: 011 717 4559

Dear Parent/Guardian

My name is Michelle Leal. I am a Psychology PhD student at the University of the Witwatersrand. I am conducting research in partial fulfilment of this degree. The purpose of the study is to examine the role of the autonomic nervous system and interaction with the environment in Disruptive Mood Dysregulation Disorder (DMDD) in children. This study is of value because very little is known about this disorder and the findings could have important implications for children who struggle to regulate their emotions at school, home and with friends. The reason for this letter is to ask your permission for your child/ward to participate in my research project.

My study will comprise of two short experiments. During the first, participants will play a computer game while their heart rate is recorded using a non-invasive ECG monitoring device. The game will consist of three sets of trials during which participants will be shown a cross, then three white squares, a blue cue block and a white target block. They will be asked to press the button corresponding to the square on the screen in which the target block appears. Before, during and after the computer game, participants will also be asked to complete a short questionnaire to indicate how they are feeling. The second experiment consists of a short video, during which the participants will be asked to identify six emotions. They will also be asked to speak about a typical day for 3-5 minutes, which will be audio

recorded (participant codes will be used during the recording, not your child's name). During these tasks, their heart rates will be monitored too. Finally, I will ask them and you to complete a short questionnaire. In total, this should take approximately one hour.

The aim is to determine whether children with DMDD show differences in the way their autonomic nervous systems respond to cues in the environment compared to children without the disorder. In order to gather data, I require the voluntary participation of children without a diagnosis of a mental health disorder and who don't suffer from chronic medical conditions or disorders from various schools. Immediate family members must also be free of a psychiatric diagnosis to ensure that the data is not distorted.

In addition, your child/ward should not exhibit the following:

- Temper outbursts occur at least three times per week
- Irritable/angry mood present for most of day, on most days, in at least two settings (at home, school or with friends) for more than one year
- These symptoms are severe in at least one setting

I am therefore inviting your child/ward to participate in this study. Your child's/ward's participation is completely voluntary. Whether you give permission for your child/ward to take part in the study or not will not affect his/her education or marks in any way. However, the school is aware of the project and has given its permission for the study to be conducted. If you allow your child/ward to participate, please complete and sign the form below and return the form to your child's/ward's class teacher as soon as possible.

Please be assured that all data collected will be kept strictly confidential and that all audio recordings will be deleted once they have been transcribed. No child will be identified in any written or spoken report. No identifying information will be requested from your child/ward, thus preserving your child's/ward's anonymity. This does mean that individual feedback

cannot be given. A summary of the findings of the study and implications thereof can be sent to you on request. Should you have any questions or if you wish to request feedback, my contact details, together with those of my supervisor, appear in the signatures below.

There are no risks associated with this study. Although there are no direct benefits for the children who choose to participate, the study will contribute to a broader understanding of this disorder and hopefully assist in the development of appropriate treatment and/or intervention strategies.

If you agree to your child's/ward's participation in this study, please complete the attached consent form and return it to your child's/ward's class teacher as soon as possible.

Thank you for taking the time to read this and for considering letting your child/ward participate in my study.

Please detach and keep this letter.

Yours sincerely,

Michelle Leal
083-200-5134
lealmic@gmail.com

Prof Kate Cockcroft(supervisor)
(011) 717-4511
kate.cockcroft@wits.ac.za

Psychology
School of Human & Community Development
University of the Witwatersrand
Private Bag 3, Wits, 2050
Tel: 011 717 4503 Fax: 011 717 4559

I,_____ consent for my child/ ward

_____ (name of child) to participate in the research study to be conducted by Michelle Leal. I am aware that:

- All details will be kept confidential at all times.
- Participation in this study is completely voluntary.
- My child's participation or non-participation in the study will have no impact on his/her current treatment.
- No information that may identify my child or me will be included in the research report.
- My child will not be harmed in any way during the assessment.
- During the assessment, my child may receive misleading information about the exact nature of the experiment as not to influence results.
- My child's information will be kept confidential at all times.
- There are no risks or benefits associated with participation in this study.

Name of Parent/Guardian: _____

Cell number: _____

Email: _____

Signed: _____

Date: _____

Appendix B: DMDD Group Parent Letter and Consent Form

Psychology
School of Human & Community Development
University of the Witwatersrand
Private Bag 3, Wits, 2050
Tel: 011 717 4503 Fax: 011 717 4559

Dear Parent(s)/Guardian(s)

My name is Michelle Leal. I am a Psychology PhD student at the University of the Witwatersrand. I am conducting research in partial fulfilment of this degree. The purpose of the study is to examine the role of the autonomic nervous system and interaction with the environment in Disruptive Mood Dysregulation Disorder (DMDD) in children. This study is of value because very little is known about this disorder and the findings could have important implications for children who struggle to regulate their emotions at school, home and with friends. The reason for this letter is to ask your permission for your child/ward to participate in my research project.

My study will comprise of two short experiments. During the first, participants will play a computer game while their heart rate is recorded using a non-invasive ECG monitoring device. The game will consist of three sets of trials during which participants will be shown a cross, then three white squares, a blue cue block and a white target block. They will be asked to press the button corresponding to the square on the screen in which the target block appears. Before, during and after the computer game, participants will also be asked to complete a short questionnaire to indicate how they are feeling. The aim is to determine whether children with DMDD show differences in the way their autonomic nervous systems respond to cues in the environment. In order to gather data, I require the voluntary

participation of children who have been diagnosed with DMDD. The second experiment consists of a short video, during which the participants will be asked to identify six emotions. They will also be asked to speak about a typical day for 3-5 minutes, which will be audio recorded (participant codes will be used during the recording, not your child's name). During these tasks, their heart rates will be monitored too. Finally, I will ask them and you to complete a short questionnaire. In total, this should take approximately one hour. If your child has already completed the first task, the second session will take no longer than 20 minutes.

I am therefore inviting your child/ward to participate in this study. Your child's/ward's participation is completely voluntary. Whether you give permission for your child/ward to take part in the study or not will not affect his/her current treatment in any way. However, your doctor/psychologist is aware of the project and has agreed to assist me with the referral of potential participants. If you allow your child/ward to participate, please complete and sign the form below and return the form to me.

Please be assured that all data collected will be kept strictly confidential and that audio recordings will be deleted once they have been transcribed. No child will be identified in any written or spoken report. No identifying information will be requested from your child/ward, thus preserving your child's/ward's anonymity. This does mean that individual feedback cannot be given. A summary of the findings of the study and implications thereof can be sent to you on request. Should you have any questions or if you wish to request feedback, my contact details, together with those of my supervisor, appear in the signatures below.

There are no risks associated with this study. Although there are no direct benefits for the children who choose to participate, the study will contribute to a broader understanding of

this disorder and hopefully assist in the development of appropriate treatment and/or intervention strategies.

If you agree to your child's/ward's participation in this study, please complete the attached consent form and return it to me.

Thank you for taking the time to read this and for considering letting your child/ward participate in my study.

Please detach and keep this letter.

Yours sincerely,

Michelle Leal

083-200-5134

lealmic@gmail.com

Prof Kate Cockcroft(supervisor)

(011) 717-4511

kate.cockcroft@wits.ac.za

Appendix C: Participant Letter and Assent Form

Psychology
School of Human & Community Development
University of the Witwatersrand
Private Bag 3, Wits, 2050
Tel: 011 717 4503 Fax: 011 717 4559

Dear Participant

My name is Michelle Leal. I am a Psychology PhD student at the University of the Witwatersrand. As part of my research project, I am doing a study on the way your nervous system responds to a computer game.

Part of this research requires that you play a computer game while I measure your heart rate. This doesn't hurt at all and involves putting two stickers on your chest and attaching a cable that wirelessly "talks" to my computer. The game will consist of three sets of trials during which you will be shown a cross, then three white squares, a blue cue block and a white target block. You will be asked to press the button corresponding to the square on the screen in which the target block appears. Before, during and after the computer game, you will also be asked to complete a short questionnaire to indicate how you are feeling. I will also ask you to watch a short video and click on faces that you think show certain feelings, and talk to me for 3-5 minutes about what your day is usually like. It should take you approximately an hour to complete. If you've already played the game, the video and audio recording should only take about 20 minutes. Your participation is valuable, as it will help us learn more about how your body reacts to things in your environment and what role it plays in how you feel. I hope

this information will eventually lead ways of helping kids who struggle to manage their emotions. I would therefore like to invite you to participate in this research.

Your responses will remain confidential and anonymity is guaranteed. At no time will I know who you are, since the game and short questionnaires request no identifying information. Participation will be considered as permission to use your responses for my research project. You can choose to withdraw at any time during the study. There are no negative consequences of participating in this study and, should you choose not to participate, this will not be held against you in any way.

As I am only interested in group trends, and have no way of linking any individual's identity to a particular questionnaire, I will not be able to give you individual feedback. You may request general feedback from me on the results of this study after 3 months. If you have any further questions or require feedback on the research, please feel free to contact me. My contact details appear below.

Thank you for considering taking part in the research project. Please detach and keep this sheet.

Yours sincerely,

Michelle Leal

083-200-5134

lealmic@gmail.com

Prof Kate Cockcroft(supervisor)

(011) 717-4511

kate.cockcroft@wits.ac.za

Psychology
School of Human & Community Development
University of the Witwatersrand
Private Bag 3, Wits, 2050
Tel: 011 717 4503 Fax: 011 717 4559

If you decide you want to be in this study, please print/write your name. If you decide that you don't want to be in the study, even if you have started in the study, then all you have to do is tell your parent/guardian or me (Michelle Leal).

I,_____ (Print your name) would like to be in this research study.

_____ (Date of assent)

_____ (Name of parent/guardian who obtained assent)

_____ (Signature of parent/guardian who obtained assent and Date)

_____ (Researcher name)

_____ (Researcher signature and Date)

Appendix D: Recording Consent Form

Psychology
School of Human & Community Development
University of the Witwatersrand
Private Bag 3, Wits, 2050
Tel: 011 717 4503 Fax: 011 717 4559

Consent Form (Recording)

I, _____ give my consent for my/my child's interview with Michelle Leal to be audio recorded for her study. I understand that:

- The audio files and transcripts will not be seen or heard by anyone other than the researcher, the bioengineer who will analyse the vocalisation features, and her supervisor.

- The recordings and transcripts will be kept in a safe place and recordings will be deleted once transcribed.

- No identifying information will be used in the transcripts or the research report.

- Although direct quotes from my/my child's interview may be used in the research report, I/my child will be referred to by a pseudonym (Respondent X, Respondent Y etc.)

Signed: _____

Date: _____

Appendix E: Gauteng Department of Education Approval Form

For administrative use:
Reference no. D2016 / 335 A
Enquiries: Diane Bunting 011 843 6503

GAUTENG PROVINCE
EDUCATION
REPUBLIC OF SOUTH AFRICA

GDE AMENDED RESEARCH APPROVAL LETTER

Date:	13 November 2015
Validity of Research Approval:	8 February 2016 to 30 September 2016
Previous GDE Research Approval letter reference number	D2016 / 049 dated 14 May 2015
Name of Researcher:	Leal M.
Address of Researcher:	36 Fairlands Estate; 174 Wilson Street; Fairland; 2170
Telephone / Fax Number/s:	011 431 0934; 083 200 5134
Email address:	1280858@students.wits.ac.za
Research Topic:	Vagal tone in Disruptive Mood Dysregulation Disorder
Number and type of schools:	FOUR Primary and THREE Secondary Schools
District/s/HO	Gauteng North

Re: Approval in Respect of Request to Conduct Research

This letter serves to indicate that approval is hereby granted to the above-mentioned researcher to proceed with research in respect of the study indicated above. The onus rests with the researcher to negotiate appropriate and relevant time schedules with the school/s and/or offices involved. A separate copy of this letter must be presented to the Principal, SGB and the relevant District/Head Office Senior Manager confirming that permission has been granted for the research to be conducted. However participation is VOLUNTARY.

The following conditions apply to GDE research. The researcher has agreed to and may proceed with the above study subject to the conditions listed below being met. Approval may be withdrawn should any of the conditions listed below be flouted:

CONDITIONS FOR CONDUCTING RESEARCH IN GDE

1. *The District/Head Office Senior Manager/s concerned, the Principal/s and the chairperson/s of the School Governing Body (SGB.) must be presented with a copy of this letter.*

Mudo
16/11/13

Office of the Director: Knowledge Management and Research
9th Floor, 111 Commissioner Street, Johannesburg, 2001
P.O. Box 7710, Johannesburg, 2000 Tel: (011) 355 0506
Email: David.Makhado@gauteng.gov.za
Website: www.education.gpg.gov.za

2. *The Researcher will make every effort to obtain the goodwill and co-operation of the GDE District officials, principals, SGBs, teachers, parents and learners involved. Participation is voluntary and additional remuneration will not be paid.*
3. *Research may only be conducted after school hours so that the normal school programme is not interrupted. The Principal and/or Director must be consulted about an appropriate time when the researcher/s may carry out their research at the sites that they manage.*
4. *Research may only commence from the second week of February and must be concluded by the end of the THIRD quarter of the academic year. If incomplete, an amended Research Approval letter may be requested to conduct research in the following year.*
5. *Items 6 and 7 will not apply to any research effort being undertaken on behalf of the GDE. Such research will have been commissioned and be paid for by the Gauteng Department of Education.*
6. *It is the researcher's responsibility to obtain written consent from the SGBs; principal/s, educator/s, parents and learners, as applicable, before commencing with research.*
7. *The researcher is responsible for supplying and utilizing his/her own research resources, such as stationery, photocopies, transport, faxes and telephones and should not depend on the goodwill of the institution/s, staff and/or the office/s visited for supplying such resources.*
8. *The names of the GDE officials, schools, principals, parents, teachers and learners that participate in the study may not appear in the research title, report or summary.*
9. *On completion of the study the researcher must supply the Director: Education Research and Knowledge Management, with electronic copies of the Research Report, Thesis, Dissertation as well as a Research Summary (on the GDE Summary template).*
10. *The researcher may be expected to provide short presentations on the purpose, findings and recommendations of his/her research to both GDE officials and the schools concerned.*
11. *Should the researcher have been involved with research at a school and/or a district/head office level, the Director/s and school/s concerned must also be supplied with a brief summary of the purpose, findings and recommendations of the research study.*

The Gauteng Department of Education wishes you well in this important undertaking and looks forward to examining the findings of your research study.

Kind regards

.....

Dr David Makhado

Director: Education Research and Knowledge Management

DATE: 2013/11/13

Making education a societal priority

Office of the Director: Knowledge Management and Research

9th Floor, 111 Commissioner Street, Johannesburg, 2001
P.O. Box 7710, Johannesburg, 2000 Tel: (011) 355 0506
Email: David.Makhado@gauteng.gov.za
Website: www.education.gpg.gov.za

For administrative use:
Reference no. D2017 / 319 AA
Enquiries: Diane Buntting 011 843 6503

GAUTENG PROVINCE
EDUCATION
REPUBLIC OF SOUTH AFRICA

GDE AMENDED RESEARCH APPROVAL LETTER

Date:	5 October 2016
Validity of Research Approval:	6 February 2017 to 30 September 2017
Previous GDE Research Approval letter reference number	D2016 / 335 A dated 13 November 2015 and D2016 / 049 dated 14 May 2015
Name of Researcher:	Leal M.
Address of Researcher:	36 Fairlands Estate; 174 Wilson Street; Fairland; 2170
Telephone / Fax Number/s:	011 431 0934; 083 200 5134
Email address:	1280858@students.wits.ac.za
Research Topic:	Vagal tone in Disruptive Mood Dysregulation Disorder
Number and type of schools:	FOUR Primary and THREE Secondary schools
District/s/HO	Gauteng North

Re: Approval in Respect of Request to Conduct Research

This letter serves to indicate that approval is hereby granted to the above-mentioned researcher to proceed with research in respect of the study indicated above. The onus rests with the researcher to negotiate appropriate and relevant time schedules with the school/s and/or offices involved. A separate copy of this letter must be presented to the Principal, SGB and the relevant District/Head Office Senior Manager confirming that permission has been granted for the research to be conducted. However participation is VOLUNTARY.

The following conditions apply to GDE research. The researcher has agreed to and may proceed with the above study subject to the conditions listed below being met. Approval may be withdrawn should any of the conditions listed below be flouted:

CONDITIONS FOR CONDUCTING RESEARCH IN GDE

1. *The District/Head Office Senior Manager/s concerned, the Principal/s and the chairperson/s of the School Governing Body (SGB.) must be presented with a copy of this letter.*
2. *The Researcher will make every effort to obtain the goodwill and co-operation of the GDE District officials, principals, SGBs, teachers, parents and learners involved. Participation is voluntary and additional remuneration will not be paid.*

2016/10/06

3. *Research may only be conducted after school hours so that the normal school programme is not interrupted. The Principal and/or Director must be consulted about an appropriate time when the researcher/s may carry out their research at the sites that they manage.*
4. *Research may only commence from the second week of February and must be concluded by the end of the THIRD quarter of the academic year. If incomplete, an amended Research Approval letter may be requested to conduct research in the following year.*
5. *Items 3 and 4 will not apply to any research effort being undertaken on behalf of the GDE. Such research will have been commissioned and be paid for by the Gauteng Department of Education.*
6. *It is the researcher's responsibility to obtain written consent from the SGB/s; principal/s, educator/s, parents and learners, as applicable, before commencing with research.*
7. *The researcher is responsible for supplying and utilizing his/her own research resources, such as stationery, photocopies, transport, faxes and telephones and should not depend on the goodwill of the institution/s, staff and/or the office/s visited for supplying such resources.*
8. *The names of the GDE officials, schools, principals, parents, teachers and learners that participate in the study may not appear in the research title, report or summary.*
9. *On completion of the study the researcher must supply the Director: Education Research and Knowledge Management, with electronic copies of the Research Report, Thesis, Dissertation as well as a Research Summary (on the GDE Summary template). Failure to submit your Research Report, Thesis, Dissertation and Research Summary on completion of your studies / project – a month after graduation or project completion - may result in permission being withheld from you and your Supervisor in future.*
10. *The researcher may be expected to provide short presentations on the purpose, findings and recommendations of his/her research to both GDE officials and the schools concerned;*
11. *Should the researcher have been involved with research at a school and/or a district/head office level, the Director/s and school/s concerned must also be supplied with a brief summary of the purpose, findings and recommendations of the research study.*

The Gauteng Department of Education wishes you well in this important undertaking and looks forward to examining the findings of your research study.

Kind regards

.....

Dr David Makhado

Director: Education Research and Knowledge Management

DATE: 2016/10/06

GAUTENG PROVINCE
Department: Education
REPUBLIC OF SOUTH AFRICA

8/4/4/1/2

GDE AMENDED RESEARCH APPROVAL LETTER

Date:	14 March 2017
Validity of Research Approval:	06 February 2017 – 29 September 2017 M2017/319AAA
Name of Researcher:	Leal M.
Address of Researcher:	36 Fairlands Estate,
	174 Wilson Street
	Fairland, 2170
Telephone Number:	011 431 0934 083 200 5134
Email address:	1280858@stuidents@wits.ac.za
Research Topic:	Vagal tone in Disruptive Mood Dysregulation Disorder
Number and type of schools:	Four Primary and Three Secondary Schools
District/s/HO	Gauteng North

Re: Approval in Respect of Request to Conduct Research

This letter serves to indicate that approval is hereby granted to the above-mentioned researcher to proceed with research in respect of the study indicated above. The onus rests with the researcher to negotiate appropriate and relevant time schedules with the school/s and/or offices involved to conduct the research. A separate copy of this letter must be presented to both the School (both Principal and SGB) and the District/Head Office Senior Manager confirming that permission has been granted for the research to be conducted.

17/03/2017

The following conditions apply to GDE research. The researcher may proceed with the above study subject to the conditions listed below being met. Approval may be withdrawn should any of the conditions listed below be flouted:

Office of the Director: Education Research and Knowledge Management
7th Floor, 17 Simmonds Street, Johannesburg, 2001
Tel: (011) 355 0488
Email: Faith.Tshabalala@gauteng.gov.za
Website: www.education.gpg.gov.za

1. *The District/Head Office Senior Manager/s concerned must be presented with a copy of this letter that would indicate that the said researcher/s has/have been granted permission from the Gauteng Department of Education to conduct the research study.*
2. *The District/Head Office Senior Manager/s must be approached separately, and in writing, for permission to involve District/Head Office Officials in the project.*
3. *A copy of this letter must be forwarded to the school principal and the chairperson of the School Governing Body (SGB) that would indicate that the researcher/s have been granted permission from the Gauteng Department of Education to conduct the research study.*
4. *A letter / document that outlines the purpose of the research and the anticipated outcomes of such research must be made available to the principals, SGBs and District/Head Office Senior Managers of the schools and districts/offices concerned, respectively.*
5. *The Researcher will make every effort obtain the goodwill and co-operation of all the GDE officials, principals, and chairpersons of the SGBs, teachers and learners involved. Persons who offer their co-operation will not receive additional remuneration from the Department while those that opt not to participate will not be penalised in any way.*
6. *Research may only be conducted after school hours so that the normal school programme is not interrupted. The Principal (if at a school) and/or Director (if at a district/head office) must be consulted about an appropriate time when the researcher/s may carry out their research at the sites that they manage.*
7. *Research may only commence from the second week of February and must be concluded before the beginning of the last quarter of the academic year. If incomplete, an amended Research Approval letter may be requested to conduct research in the following year.*
8. *Items 6 and 7 will not apply to any research effort being undertaken on behalf of the GDE. Such research will have been commissioned and be paid for by the Gauteng Department of Education.*
9. *It is the researcher's responsibility to obtain written parental consent of all learners that are expected to participate in the study.*
10. *The researcher is responsible for supplying and utilising his/her own research resources, such as stationery, photocopies, transport, faxes and telephones and should not depend on the goodwill of the institutions and/or the offices visited for supplying such resources.*
11. *The names of the GDE officials, schools, principals, parents, teachers and learners that participate in the study may not appear in the research report without the written consent of each of these individuals and/or organisations.*
12. *On completion of the study the researcher/s must supply the Director: Knowledge Management & Research with one Hard Cover bound and an electronic copy of the research.*
13. *The researcher may be expected to provide short presentations on the purpose, findings and recommendations of his/her research to both GDE officials and the schools concerned.*
14. *Should the researcher have been involved with research at a school and/or a district/head office level, the Director concerned must also be supplied with a brief summary of the purpose, findings and recommendations of the research study.*

The Gauteng Department of Education wishes you well in this important undertaking and looks forward to examining the findings of your research study.

Kind regards

.....

Ms Faith Tshabalala
CES: Education Research and Knowledge Management

DATE: 14/03/2017

Office of the Director: Education Research and Knowledge Management
7th Floor, 17 Simmonds Street, Johannesburg, 2001
Tel: (011) 355 0488
Email: Faith.Tshabalala@gauteng.gov.za
Website: www.education.gpg.gov.za

Appendix F: Medical Ethics Approval Form

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)

01 March 2017

Ms Michelle Leal
Faculty of Health Sciences
Department of Psychology
University of the Witwatersrand
Parktown
Johannesburg

Sent by email to:michellel@scarab.co.za

Dear Ms Leal,

Re: **Protocol Ref no: M150526**
Protocol Title: Disruptive Mood Dysregulation Disorder : A Polyvagal Perspective
Principal Investigator: Ms Michelle Leal
Protocol Amendment

- **An Affect Recognition task**
- **A Prosody Task**
- **Request to ask Participants their Race**

This letter serves to confirm that the Chairman of the Human Research Ethics Committee (Medical) has approved the protocol amendments on the abovementioned protocol, as detailed in your letter dated 01 February 2017.

The following documents were received:

- Cover Letter dated 01 February 2017.
- HREC (Medical) Application (with track changes)
- Study Proposal (With track changes)
- HREC (Medical) Clearance Certificate

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)

Thank you for keeping us informed and updated.

Yours Sincerely,

L. moeng

Mr Lebohang Moeng
Administrative Assistant
Human Research Ethics Committee (Medical)

UNIVERSITY OF THE WITWATERSRAND
OFFICE OF THE DEPUTY VICE CHANCELLOR RESEARCH
01 MAR 2017
RESEARCH OFFICE HREC (Medical)
JOHANNESBURG

Research Office Secretariat: Faculty of Health Sciences, Phillip Tobias Building, 3rd Floor, Office 302, Corner York Road and 29 Princess of Wales Terrace, Parktown, 2193 Private Bag 3, Wits 2050 | **T**+27 (0)11-717-1234/2656/2700/1252 **E**: Lebo.Moeng@wits.ac.za | **Office E** HREC-Medical.ResearchOffice@wits.ac.za |Website: www.wits.ac.za/research/about-our-research/ethics-and-research-integrity/

Appendix G: Guilford Press Reprint Permission

Guilford Publications

370 Seventh Avenue, Suite 1200
New York, NY 10001-1020
permissions@guilford.com
(212) 431 9800

5 April 2016

NEW ADDRESS!

PERMISSION AGREEMENT

Michelle Leal
University of Witwatersrand
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Appendix H: Demographic Questionnaire

Child Demographic Information **Participant Code: _____**

Session: _____

(Please cross the option that applies to you, where appropriate)

1. Age: _____ Date of birth: _____

2. Gender: MALE FEMALE

3. School: _____

4. Grade: _____

5. Do you belong to a medical aid: YES NO

6. Parent 1/Guardian 1 highest level of education:

MATRIC VOCATION DIPLOMA DEGREE POST-GRAD

OTHER

If other, please specify _____

7. Parent 2/Guardian 2 highest level of education:

MATRIC VOCATION DIPLOMA DEGREE POST-GRAD

OTHER

If other, please specify _____

8. Parent 1/Guardian 1 occupation: _____

9. Parent 2/Guardian 2 occupation: _____

10. OPTIONAL: How would you describe your racial/ethnic background:
_____

11. Home language:

AFRIKAANS	ENGLISH	isiNDEBELE	sePEDI
sISWATI	seSOTHO	xiTSONGA	seTWANA
tshiVENDA	isiXHOSA	isiZULU	OTHER

If other, please specify:_____

12. If English is not your home language, please indicate for how many years you have been exposed to English: _____

13. If English is not your home language, please rate your English understanding/ comprehension skills with 1 being 'not so good' and 5 being 'excellent'.

1	2	3	4	5

14. Have you been diagnosed with a learning disability: YES NO
If yes, please
specify:_____

15. Have you been diagnosed with a mental health disorder: YES NO
If yes, please
specify:_____

16. Are you currently taking or have you taken any medication in the last 3 months:

YES NO

If yes, please
specify:_____

Appendix I: Actiwave Cardio Placement Depiction

Source: CamNtech (http://www.camntech.com/products/actiwave-cardio/actiwave-cardio-overview)

Appendix J: Observed Frustration Reactivity

Global Negative Reactivity

During the frustration condition, the child exhibited:

	0	1	2	3	4
	No distress	Negligible distress	Minor distress	Major distress	Extreme distress
Pouting					
Whining					
Fussing					
Crying					
Swearing					
Shouting/Screaming					
Tantrumming					

Global Affect

During the frustration condition, the child exhibited the following affect:

-3	-2	-1	0	1	2	3
Highly negative	Somewhat negative	Slightly negative	Neutral	Slightly positive	Somewhat positive	Highly positive

Notes

Appendix K: Attachment Style Questionnaire

Attachment Style Classification Questionnaire for Latency Age Children - Ricky Finzi-Dottan

Here are 15 sentences. How true is each of the sentences for you? Everyone has his or her own answer. Try to answer only what you feel. This is not a test, and there are no right or wrong answers. Read each sentence carefully. Then choose one of the five answers in the box below. Every answer has a number. Circle the number of the answer that best describes you.

1 All wrong	2 Wrong	3 A bit wrong/a bit right	4 Right	5 Very right

		All wrong	Wrong	A bit wrong/a bit right	Right	Very right
1	I make friends easily	1	2	3	4	5
2	I don't feel comfortable making new friends	1	2	3	4	5
3	It is easy for me to depend on people, especially if they are my good friends.	1	2	3	4	5
4	I feel uncomfortable if others get too friendly or too close to me	1	2	3	4	5
5	Sometimes I feel afraid when other kids no longer want to be my friend.	1	2	3	4	5
6	I like having good friends and being with them all the time	1	2	3	4	5
7	It's all right with me if good friends trust and depend on me.	1	2	3	4	5
8	It's hard for me to trust others completely.	1	2	3	4	5
9	I sometimes feel that others don't want to be good friends with me as much as I do with them.	1	2	3	4	5
10	I believe that those who are close to me will not leave me.	1	2	3	4	5
11	I'm sometimes afraid that no one really loves me.	1	2	3	4	5
12	I feel uncomfortable and get annoyed when someone tries to get too close to me.	1	2	3	4	5
13	It's hard for me to really trust others, even if they're good friends of mine	1	2	3	4	5
14	Children sometimes avoid me when I want to be good friends with them.	1	2	3	4	5
15	I don't mind when people get too close to me.	1	2	3	4	5

Appendix L: Social Behaviour and Communication: Parent Questionnaire

The purpose of this questionnaire is to obtain your perceptions of your child's social behaviour and communication. Thank you for your input! It is greatly valued!
PLEASE BE SURE TO ANSWER QUESTIONS ON BOTH SIDES OF THE QUESTIONNAIRE.

Directions: The questionnaire is divided into five areas: Gestural and Facial Expression; Language and Social Interaction; Listening and Response to Sound; Emotional Regulation and Reciprocity; and Behaviour.

The "light bulb" provides insight as to the type of situation in which the skill or ability may commonly be observed. Its purpose is to direct your thoughts and provide memory cues to aide in the accuracy of assessment.

A. In the "A" section, please note whether or not this is an area of concern for your child. Circle "Yes" or "No." If you circled "Yes", go on to the next item. If you circled "No," complete items "B", "C", "D".

B. In the "B" section, please give an example of behaviours which you have observed. Please provide rich details describing particular episodes and behaviours. Be sure to describe the settings in which these behaviours occurred, for example, at home, at school etc.

C. In the "C" section, please note where your child is on an ability continuum. The far-left marker indicates the complete absence of a skill; the far-right marker indicates the skills of a typically developing child. Use the symbol "X" to mark your child's skill level prior to starting medication and the symbol "O" to mark your child's skill level after starting medication. This provides a visual, subjective representation of your opinion of how far your child has come and how far he/she has yet to go.

D. In the "D" section, compare your child's current performance to his/her performance prior to medication. Is it "a lot worse," "a little worse," "about the same," "a little better," or "a lot better?"

Adapted, with permission, from the Listening Project Parent Questionnaire
(Bazhenova & Porges, 2000)

Gestures and Facial Expression

I. Using gestures spontaneously such as nodding yes/no, waving hello/goodbye, pointing finger to direct attention, "high-five," or "thumbs-up."

Use of body language and facial expression in communication. In a quiet movie theatre or religious service where talking is prohibited, how would your child communication something?

A. Prior to medication, my child used gestures in an appropriate manner (circle one):

YES If Yes, go to **II** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of your child's gestures not previously observed, but used in communication following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence of typically gestural use — Gesture use of developing child

/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's ability to use gestures in communication is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

II. Making eye contact connections during communication

Use of eye contact. When sitting face to face at a table, does your child catch your eye? Does he/she look back and forth between you and a toy when you play?

A. Prior to medication, my child used eye contact in an appropriate manner (circle one):

YES If Yes, go to **III** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of eye contact not previously observed, but used in communication following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence of eye contact — Eye contact of typically developing child

/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's use of eye contact is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

III. Opening eyes wide and lifting eye brows when making eye contact

Use of facial expression. Can you tell when your child is listening because of the expression in his/her eyes?

A. Prior to medication, my child used appropriate facial expressions while listening (circle one):

YES If Yes, go to **IV** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of facial expression not previously observed, but used while listening following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence of facial expression — Facial expression of typically developing child

/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's facial expression while listening is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

IV. Turning to face towards another person when communicating

Looking at you when talking or when wanting something. Does he/she look to find you before beginning to speak? When reading or watching a movie together sitting in lap or next to each other, does he/she turn towards you to talk?

A. Prior to medication, my child looked at the speaker in an appropriate manner (circle one):

YES If Yes, go to **V** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of looking at speaker not previously observed, but used following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence of orientation — Orientation of typically developing child

/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's orientation towards the speaker is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

V. Tilting head in an expression of interest when listening

Expressing interest with head posture. Does your child tilt head position to indicate listening or interest?

A. Prior to medication, my child tilted head in an appropriate manner (circle one):

YES If Yes, go to **VI** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

E. Description of head tilting not previously observed, but used while listening, following medication:

F. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence of typically head tilting — Head tilting of developing child

/_____/_____/_____/_____/_____/_____/

G. In comparison to before medication, my child's head tilting while listening is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

Language and Social Interaction

VI. Using words and sentences spontaneously to communicate thoughts and ideas

Use of verbal language to relate thoughts, feelings and events which have happened, are happening, or will happen. What does your child tell you when arriving home from school or at dinner table conversations?

A. Prior to medication, my child used verbal language appropriately (circle one):

YES If Yes, go to **VII** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of spontaneous verbal language not previously observed, but used following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence of verbal language — Verbal language of typically developing child

/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's spontaneous verbal language is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

VII. Talking with others

Reciprocal conversation exchanges. Can your child participate in a reciprocal conversation which continues beyond two exchanges (asking a question and answering it)? Can he or she switch topics and talk about something not on his or her own agenda? Can he or she talk without using scripts from movies or television?

A. Prior to medication, my child talked to others in an appropriate manner (circle one):

YES If Yes, go to **VIII** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of reciprocal conversations not previously observed, but used following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence of talking with others child — Talking w/others of typically developing

/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's reciprocal conversation is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

VIII. Initiating interactions

Relating to others. Does your child come to you to share an idea or thought? If he/she she is playing with a toy, does he/she connect with you to share excitement and joy? Think about the difference between initiations just for social pleasure and for those of requesting. Does your child initiate just to connect, not just to get something?

A. Prior to medication, my child initiated social interactions for the sake of social pleasure (circle one):

YES If Yes, go to **IX** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of initiating behaviour not previously observed, but used following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence of verbal initiation — Verbal initiation of typically developing child

/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's spontaneous engagement with others is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

IX. Interacting with peers

Responding with reciprocity in peer relationships. How does your child relate to other children? Can he/she take turns and participate in a group game?

A. Prior to medication, my child engaged with peers in an appropriate manner (circle one):

YES If Yes, go to **X** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of peer interactions not previously observed, but engaged in following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence of peer interaction — Peer interaction of typically developing child

/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's peer interaction is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

X. Reading social cues

Perceiving and adapting to convert social cues. How does your child react when the demands on social behaviour are changed or unstated? Does he or she know to be quiet in an audience setting or that when inside you use a quiet voice?

A. Prior to medication, my child read social situations appropriately (circle one):

YES If Yes, go to **XI** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of reading social cues not previously observed, but used following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence ofsocial cue reading

Social cue reading of typically developing child

/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's social cue reading is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

Listening and Processing of Sound

XI. Listening to verbal requests

Understanding of spoken words without visual or contextual cues. How does your child respond if you verbally request an object from another room or if you say something out of context?

A. Prior to medication, my child listened in an appropriate manner (circle one):

YES If Yes, go to **XII** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of listening not previously observed, but noticed following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence of listening — Listening of typically developing child

/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's response to verbal requests is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

XII. Tolerating sound and noise

Tolerance of sound. Are there any places that your child doesn't like because it is too noisy? How does your child react to common household noises (e.g., vacuum cleaner, garbage disposal, etc.)? Is he or she irritated by common noises or frightened by particular noises?

A. Prior to medication, my child tolerated different sounds without distress (circle one):

YES If Yes, go to **XIII** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of sound tolerance not previously observed, but observed following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

No tolerance of **low** sounds — Tolerance of **low** sounds of typically developing child

/_____/_____/_____/_____/_____/_____/

No tolerance of **high** sounds — Tolerance of **high** sounds of typically developing child

/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's tolerance of environmental sounds is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

Emotional Regulation and Reciprocity

XIII. Maintaining emotional control

Regulating emotional control. How does your child react to unexpected changes? Can he or she switch gears to flow with changing circumstances without fear, tantrums, or over-excitement?

A. Prior to medication, regulated emotions in an appropriate manner (circle one):

YES If Yes, go to **XIV** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of emotional control not previously observed, but observed following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence of emotional control — Emotional control of typically developing child

/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's emotional control is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

XIV. Overcoming fears

Response to fears. Are there specific things of which your child is fearful? Any unusual fears or fears which you cannot explain?

A. Prior to medication, my child experienced daily life without unusual fears (circle one):

YES If Yes, go to **XV** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of reduction in fear response observed following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Atypical fear reactions — Fear reactions of typically developing child

/_____/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's fear or fear management is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

XV. Feeling expression

Emotional reciprocity. Does your child express interest in the feelings of others or express his/her feelings towards you and others?

A. Prior to medication, my child expressed and responded to emotions appropriately (circle one):

YES If Yes, go to **XVI** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

B. Description of feeling expressions not previously observed, but used following medication:

C. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence of feeling expression — Feeling expression of typically developing child

/_____/_____/_____/_____/_____/_____/

D. In comparison to before medication, my child's feeling expression is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

Behaviour

XVI. Organisation of play behaviour

Organising play behaviour. Can your child play independently in an organised manner? For example, when you are busy fixing dinner, is your child able to occupy him or herself in some play activity?

A. Prior to medication, my child engaged effectively in play activities (circle one):

YES If Yes, go to **XVII** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

E. Description of my child's organisation of play not previously observed, but used following medication:

F. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence of organised play behaviour — Organisation of play behaviour of typically developing child

/_____/_____/_____/_____/_____/_____/

G. In comparison to before medication, my child's ability to play on his/her own is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

XVII. Tolerating change

Response to change in routine or environment. Is your child able to flow with unexpected changes in traditional routines? Is your child able to change from one activity to another without disruption?

A. Prior to medication, my child flowed with change easily (circle one):

YES If Yes, go to **XVIII** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

E. Description of new response to change demonstrated following medication:

F. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Absence ofability to tolerate change — Tolerance of change of typically developing child

/_____/_____/_____/_____/_____/_____/

G. In comparison to before medication, my child's ability to tolerate change is:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

XVIII. Unusual interests

Engaging in preoccupations or unusual interests. Does your child have interests in unusual objects or is he or she interested in some activities or objects to an excessive degree?

B. Prior to medication, my child's interests were appropriate (circle one):

YES If Yes, go to **XIX** (next page).
NO If No, go to sections "B", "C" & "D" (this page)

H. Description of reduction in unusual interests observed following medication:

I. Please estimate your child's performance before and after medication. Place an "X" on the scale to indicate your child's ability before and an "O" to indicate ability after.

Presence of unusual interests — Interests of typically developing child

/_____/_____/_____/_____/_____/_____/

J. In comparison to before medication, my child's unusual interests are:

___a lot worse ___a little worse ___the same ___a little better ___a lot better

XIX. Any thoughts or observations regarding specific changes in your child's behaviour following medication?