Parental Stress, Anxiety, and Depression and Child Emotional Intelligence in Children with 22q11.2 Deletion Syndrome

Megan A. Goldfarb
University of New Orleans, mgoldfar@uno.edu

Follow this and additional works at: https://scholarworks.uno.edu/td

Part of the Biological Psychology Commons, Child Psychology Commons, and the Developmental Psychology Commons

Recommended Citation

This Thesis-Restricted is protected by copyright and/or related rights. It has been brought to you by ScholarWorks@UNO with permission from the rights-holder(s). You are free to use this Thesis-Restricted in any way that is permitted by the copyright and related rights legislation that applies to your use. For other uses you need to obtain permission from the rights-holder(s) directly, unless additional rights are indicated by a Creative Commons license in the record and/or on the work itself.

This Thesis-Restricted has been accepted for inclusion in University of New Orleans Theses and Dissertations by an authorized administrator of ScholarWorks@UNO. For more information, please contact scholarworks@uno.edu.
Parental Stress, Anxiety, and Depression and Child Emotional Intelligence in Children with 22q11.2 Deletion Syndrome

A Thesis

Submitted to the Graduate Faculty of the University of New Orleans in partial fulfillment of the requirements for the degree of Master of Science in Psychology

by

Megan Anne Goldfarb

B.S. Tulane University, 2003
M.A. John Carroll University, 2009

August, 2018
# Table of Contents

List of Tables ........................................................................................................... iii  
Abstract .................................................................................................................. iv
Introduction ............................................................................................................ 1
Method ...................................................................................................................... 11
Results ..................................................................................................................... 14
Discussion .............................................................................................................. 20
References ............................................................................................................. 25
Vita ......................................................................................................................... 33
List of Tables

Table 1. Means and standard deviations of main study variables by diagnostic group.........15

Table 2. Correlations of Main Study Variables by Diagnosis: 22q........................................15

Table 3. Correlations of Main Study Variables by Diagnosis: TD........................................16

Table 4: Regression analyses predicting parental stress from 22q11.2DS diagnosis and EI.................................................................18

Table 5: Regression analyses predicting parental anxiety from 22q11.2DS diagnosis and EI.................................................................18

Table 6: Regression analyses predicting parental depression from 22q11.2DS diagnosis and EI.................................................................19
Abstract

Children with chromosome 22q11.2 deletion syndrome (22q11.2DS) have serious medical, psychological, and behavioral symptoms that are stressful to their parents. Higher general intelligence quotients (IQ) and emotional intelligence (EI) in children could allay parental stress. Self-reported stress, anxiety, and depression were measured in parents of children with 22q11.2DS (n=42) and a healthy control group (n=20) in relation to children’s IQ and EI. Children with 22q1.2DS had lower IQ and EI scores. Parental groups did not differ in their reported stress, anxiety, or depression. Children’s IQ and EI levels did not relate to parental measures of affect even in the 22q11.2DS group. Based on these families at this measurement point, it appears that parents are coping well with the challenges of raising a child with a complex neurodevelopmental disorder and that IQ and EI do not play a significant role in parental affect. Further interpretation and future directions are discussed.

Keywords: 22q11.2DS, parents, EI, emotional status
Parental Stress, Anxiety and Depression and Child Emotional Intelligence in Children with 22q11.2 Deletion Syndrome

Introduction

Chromosome 22q11.2 deletion syndrome (22q11.2DS) presents with complex and heterogeneous medical, psychological, and behavioral symptoms. There are at least 180 physical and behavioral symptoms ascribed to this neurodevelopmental syndrome (Sphrintzen, 2008). 22q11.2DS is the result of a hemizygotic deletion that includes upwards of 30 million base pairs encompassing 40 genes. It is the most common survivable microdeletion identified in humans (Sphrintzen, 2005; van den Bree et al., 2013). 22q11.2DS is most often de novo with 90 percent of cases reporting no family history of the syndrome. However, 22q11.2DS can also be inherited from a parent with 22q11.2DS via autosomal dominance (Firth, 2009).

Physical symptoms of 22q11.2DS vary widely across individuals, and can include conotruncal heart malformations, cognitive and developmental deficits, velopharyngeal insufficiency, craniofacial abnormalities (e.g., long faces and hooded eyelids), and short stature, as well as deficits in immune, auditory, and endocrine system functioning (Bales, Zaleski et al., 2010; Prinzie et al., 2004; Vieira et al., 2015).

Learning impairment and comorbid psychiatric and behavioral problems are common in people with 22q11.2DS. For example, anxiety disorders are diagnosed in over 35 percent of children and adolescents with 22q11.2DS (Schneider et al. 2014) with rates of 54 percent reported (Stachon & DeSouza, 2011). Attention deficit hyperactivity disorder (ADHD) occurs in over 37 percent (Schneider et al., 2014) with some children having both (Stephenson et al. 2014). The syndrome is also associated with serious psychiatric illnesses including bipolar disorder and schizophrenia (Sphrintzen, 2000). Remarkably, 22q.11.2DS confers an increased risk 30-fold that of the general population for the development of schizophrenia in adolescence and young adulthood (Bassett et al., 2003). There appear to be higher than typical rates of autism spectrum disorder (ASD) as well but the prevalence ranges in the literature from 14 to 50 percent, suggesting that more study is needed (Bassett et al., 2003; Bish et al., 2005; Gothelf, Schaer & Eliez, 2008; Kates et al., 2007; Vorstman et al., 2006; Wenger et al., 2016).

The 22q11.2 deletion syndrome is a complex neurodevelopmental disorder with significant medical, intellectual, and socioemotional challenges that they and their families must cope with from infancy into an uncertain adulthood. Parental coping ability and access to capable medical and educational resources can vary greatly and depends on financial and community resources. Parents report elevated anxiety as a result of behavioral problems in their infants with 22q11.2DS (Briegel et al. 2007) and they worry that older children will develop a psychotic illness such as schizophrenia (Hercher & Bruenner, 2008). The aim of the present study is to measure factors that contribute to successful coping in parents and children affected by 22q11.2DS.

Behavioral Characteristics of 22q11.2 Deletion Syndrome

Many social deficits have been observed in 22q11.2DS children that may contribute to parental stress, anxiety, and depression. For example, children with 22q11.2DS are characterized as less social, shyer, less able to read facial cues, more withdrawn, less conscientious, more emotionally unstable, more irritable, more dependent, and show more internalizing symptoms than their typically developing (TD) peers (Shashi et al., 2012; Schonherz et al., 2014). Possible explanations for these temperamental characteristics include genetics (i.e. a neural basis for
personality traits) combined with the experiences of dealing with the medical issues that often accompany a 22q11.2DS diagnosis (Schonherz et al., 2014). Differences in brain development in regions associated with mood regulation and disinhibition may also be a factor (Green et al., 2009).

A study by Kiley-Brabeck and Sobin (2006) reports that social skills (e.g., prosocial behaviors such as sharing and helping, and using language to ask for help and engage with others; Elliot, Malecki & Demaray, 2001) and executive functioning (i.e., cognitive processes required for goal completion and problem solving; Welsh, Pennington, & Groisser, 1991) are negatively impacted in those with 22q11.2DS. Specifically, their findings suggest that cooperation, assertion, responsibility, and self-control in children with 22q11.2DS is significantly lower when compared to their paired typically developing sibling. These social skills are related to executive functioning and likely contribute to relational difficulties in school with peers and teachers and with parents and siblings at home.

Another phenotypic element of personality in 22q11.2DS children noted above is anxiety. Stachon and DeSouza (2011) report that as many as 54% of 22q11.2DS children experience symptoms and/or meet full criteria for an anxiety disorder. In a sample of 68 children with 22q11.2DS, Stephenson and colleagues (2015) found that 50% exhibited symptoms of anxiety and 63% exhibited symptoms of both anxiety and depression symptoms. Additionally, children who ranked the highest in anxiety and depression had significantly higher rates of dysfunctional communication and hyperactivity, and significantly lower scores of activities of daily living than their TD siblings, indicative of poorer adaptive functioning. These findings suggest that anxiety and depression are negatively impacting adaptive functioning in the 22q11.2DS population. These difficulties are also likely to contribute to parental stress. Taken together, findings indicate that the behavioral phenotypic profile of children with 22q11.2DS can be challenging for both the children who suffer from this syndrome, and their caregivers in terms of day-to-day functioning.

Adaptive Functioning, Emotional Intelligence and 22q11.2DS

The origins of anxiety in children with 22q11.2DS may arise from several sources. For example, poorer age-appropriate adaptive function, which refers to the age appropriate expectations for independent functioning and communication, likely contributes to anxiety via frustration in both the child and the caregiver. Capacity for emotional regulation and understanding other’s emotions contribute to stressful coping or can exacerbate negative stress in both parent and child. These interactions likely contribute to emotional development in children and impact stress, anxiety, and depression in caregivers of children with neurodevelopmental disorders (Cicchetti, Ackerman, & Izard, 1995; Hastings & Brown, 2002; Calkins & Hill, 2007).

Angkutsiri and colleagues (2012) found that anxiety but not full-scale intelligence related to adaptive functioning in children with 22q11.2DS. Specifically, poorer competence in skills such as communication, self-care, health and safety were associated with elevated levels of anxiety and not predicted by general intelligence. Thus, interventions designed to reduce anxiety may improve children’s adaptive functioning. Alternatively, improving these skills might reduce feelings of anxiety as the child builds confidence and autonomy. Furthermore, reducing caregiver burden and associated stress may also play a role in reducing anxiety in the child in a reciprocal way.

Optimal social and adaptive functioning is predicated on the presence of Emotional Intelligence (Brackett, Rivers, Shiffman, Lerner & Salovey, 2006; Koohsar & Bonab, 2011). Emotional Intelligence (EI) refers to the emotional, personal, and social dimensions of
intelligence, in that it is comprised of one's ability to adapt to the environment, manage emotions, and be able to relate to others via an understanding of both oneself and others (Bar-On & Parker, 2000). For example, the EI may be a protective factor against psychological distress due to the increased use of healthy coping mechanisms (e.g. planned exercise, relaxation time) implemented to manage stress (Tsouais & Nikolas, 2005). Research on those who rank higher on EI have been shown to have higher levels of self-control, humor, and internal locus of control (Schutte, Malouff, Thorsteinsen, Bhullar, & Rooke, 2007; Saklofske, Austin, Galloway & Davidson, 2007; Greven, Chamorro-Premuzic, Arteche, & Furnham, 2008).

Research on EI recognizes that its presence is associated with a wide assortment of outcomes. Lower EI, particularly in children, relates to a variety of behavioral symptoms, including externalizing behaviors such as delinquency (Koohsar & Bonab, 2011) and internalizing behaviors such as anxiety and depression (Schutte et al, 2007). Higher EI is associated with high academic achievement (Parker, Summerfeldt, Hogan & Majeski, 2004), work success (Dulewicz & Higgs, 1998), and leadership (Cooper & Sawaf, 1997). For example, Tsousis and Nikolaou (2005) found a positive correlation between levels of EI and both mental and physical health. In their examination of stress reactivity and EI, Salovey, Stroud Woolery and Epel (2002) found that those who rank higher in EI tend to have lower cortisol secretion in response to stressors. The authors suggest that this came about through the use of mood regulation when faced with stressors; participants with higher levels of EI interpreted stressors as less threatening and used less passive coping mechanisms.

A number of studies have focused on the impact of EI on relationships with family and peers. A 2002 study (Palmer, Donaldson & Strough) found evidence that the quality of interpersonal relationships is positively impacted by higher levels of EI. Similarly, Lopes, Salovey and Straus (2003) found that those scoring higher in EI reported higher levels of parental support, more positive relations with others, and fewer negative interactions with friends. Adolescents who are better able to infer what others are feeling (empathic accuracy) have better social relationships and are less likely to be victims of bullying which can be a significant stressor in this age group (Gleason, 2004). Salovey and colleagues (2002) report that those with higher perceived emotional intelligence also reported greater psychological and interpersonal functioning, used more active coping strategies, and interpreted repeated laboratory stressors as less threatening. They also showed a lower physiological stress response as indicated by lower levels of cortisol. Others have found that EI is negatively correlated with both acute and chronic stress (Singh & Sharma, 2012) likely as a result of more positive coping mechanisms those with higher EI appear to implement.

The implications of EI in relation to interpersonal relationships (Gleason, 2004) and as a potential buffer against stress (Salovey et al., 2002) can be especially useful in understanding factors affecting relationships in parent-child dyads. While the research on the role of EI in the context of parent-child relationships is sparse, Alegre (2010) discusses research that shows a connection between parenting practices and emotional intelligence in children; specifically, that parental warmth is positively correlated with adolescent emotional intelligence. Liau, Liau, Teoh and Liau (2003) found an inverse relationship between high EI in adolescents and rates of delinquency and parental monitoring. Further, parents who promote, train and reward emotionally intelligent behavior in their adolescents have children that demonstrate higher levels of EI (Martinez-Pons, 1999).

In light of the apparent benefits correlated with higher EI, including stronger interpersonal relationships, it stands to reason that higher levels of EI in children would make for
higher quality relationships with parents. It seems parents would have less emotional strife to manage in their children in the arena of social relationships with peers, and that their children would be less stressed (and by proxy, more agreeable) and that these strengths conferred by higher levels of EI would translate to the parent-child relationship as well. Parents themselves may have better EI and could, consequently, be better able to cope with difficult situations.

While a comprehensive literature review did not yield any studies to date examining EI in children with 22q11.2DS, it is hypothesized that higher levels of EI in children would positively impact parent-child dyads in the 22q11.2DS population, resulting in parents who are less stressed, anxious, and depressed.

**22q11.2DS and IQ**

It is important to address the relationship between EI to general intelligence or intelligence quotient (IQ) derived from standardized tests of intelligence, and as such, an analysis of the relationship between EI and IQ in children with 22q11.2DS is included in this study. To date, studies into the direction and strength of this relationship have yielded inconclusive results. To highlight the disagreement within the literature, a year 2000 study by Ciarrochi, Chan, and Caputi suggests that there was no relationship between EI and IQ in college students but found that IQ was related to mood-managing and preventing mood from biasing judgement. Another study conducted 12 years later in male college students also found no correlation between IQ and EI (Singh & Sharma, 2012). However, a study using functional neuroimaging to examine the relationship between and the neuro-correlates of IQ and EI in Vietnam War veterans found that IQ was positively related to EI. Further, this study found evidence that the same regions of the brain perform both emotional and cognitive tasks through a shared network of frontal, temporal and parietal brain regions (Barbey, Colom, & Grafman, 2013).

It appears that the relationship between EI and IQ is becoming conceptualized less as two distinct sets of abilities, and more as an interrelated system. To begin with, research is suggesting that EI may actually be comprised of two types of cognitive intelligence: fluid and crystalized (Webb et al., 2013). Additionally, EI may enhance mood to such an extent that it increases the ability to perform challenging cognitive tasks and enhance the ability to learn and problem solve (Schutte, Schuettpelz, & Malouff, 2001).

In people with 22q11.2DS, learning disabilities/intellectual disabilities are common with a prevalence rate of 97 percent (Firth, 2009). Using various editions of the Wechsler Intelligence Scale for Children (WISC) in a large number of studies, researchers reliably find that children and adolescents with 22q11.2DS have Full Scale IQ (FSIQ) scores within the range of 70 to 85 with variations of plus or minus 15 points. Notably, Verbal IQ (VIQ) scores are generally significantly higher than nonverbal scores and often approach age appropriate norms. One explanation for this is the deficits children with 22q11.2DS tend to have in their visuospatial-perceptual functioning (Swillen et al., 1997). As Swillen and colleagues (1999) point out, this pattern in scoring calls into question the relevance of the FSIQ score as a measure of intellectual capacity in 22q11.2DS because the VIQ is significantly higher than the Performance IQ.

Memory and spelling are often relative strengths for children and adolescents with 22q11.2DS. However, impairments in a variety of other cognitive and perceptual domains include visuospatial and temporospatial perception, attention, mathematics, and tasks that require both working memory and complex memory (Woodin et al., 2001). A longitudinal study of children with 22q11.2DS found that anxiety and lower and declining VIQ was associated with higher risk of developing a psychotic disorder in later years (Gothelf et al., 2007). Similar findings are reported in patients with schizophrenia but who do not have 22q11.2DS, with
language skills declining in the prodromal period before symptoms of schizophrenia emerge (Gothelf et al., 2005). One last important point to consider regarding IQ specific to this population is that the range in IQ scores have been found to be directly linked to 22q11.2DS, and not to any of the outward physical signs or symptoms of the chromosome deletion, such as cleft palate issues or cardiac problems. Instead, the range appears to be related to brain development in 22q11.2DS (Gerdes et al, 1999). Higher IQ and higher EI are likely to translate into better relationships for children with 22q11.2DS. Better quality relationships are likely to lead to less stress for both the child with 22q11.2DS and their parents. As such, addressing IQ in a study of parental well-being is necessary.

**22q11.2DS and Parental Stress**

By definition, stress is any uncomfortable "emotional experience accompanied by predictable biochemical, physiological and behavioral changes" (Baum, 1990). While not a formal diagnosis in and of itself, the experience of stress contributes to the etiology of a number of psychiatric disorders, including but not limited to post traumatic stress disorder and adjustment disorders, as outlined in the Diagnostic and Statistical Manual, Fifth Edition (American Psychiatric Association, 2013). Coping with the complex, serious, and unpredictable symptoms and conditions associated with 22q11.2DS is likely stressful for both the affected child but also for his or her family (Beaton & Simon, 2011).

Smith and colleagues’ 2001 study of developmentally delayed children identified that the greatest contributor to parental stress in families was a lack of family resources in the form of time and income. Their research showed that lack of time and income trumped lack of family support, which has been repeatedly identified as one of the biggest contributors. Spratt, Saylor and Macias’ 2007 study of families of children with developmental, behavioral and neurological problems had similar findings, adding that caregiver age also contributed to the amount of stress parents report. Resch and colleagues (2010) found that parents of children with developmental and physical disabilities cite lack of access to information and services in addition to the aforementioned stressors. Additionally, a lack of resources related to understanding the complexity of the diagnosis is implied in a study which determined the extent to which the internet is a source of information for parents of children with 22q11.2DS, identifying that the internet was the first source for parents regarding the comorbidity of various psychiatric illnesses associated with 22q11.2DS (van den Bree et al., 2013). This last study highlights the degree of uncertainty that surrounds the syndrome with parents worrying about their child’s future.

Studies of parental stress in other populations of children with neurodevelopmental disorders such as autism spectrum disorder (ASD), Down syndrome, or other major medical illnesses indicate that parents report higher levels of stress than their TD counterparts (Dumas, Wolf, Fisman & Culligan, 1991; Hayes & Watson, 2013). To date, research examining how the unique challenges faced by parents of 22q11.2DS children affects both the 22q11.2DS children and the parent-child relationship is sparse. Keller and Honig (2004) found that parents of children with 22q11.2DS and the children themselves reported elevated stress, anxiety, and depression.

For parents of children with 22q11.2DS, stress can be triggered by a variety of reasons, often first and foremost in the context of getting a diagnosis (Goodwin et al, 2017). The difficulty in getting an accurate diagnosis is due to the wide range of phenotypic expression present in 22q11.2DS, and the fact that the most accurate diagnosis comes via genetic testing (Firth, 2009). Additional sources of stressors for parents of children with 22q11.2DS include behavior problems that begin to emerge in infancy, such as sleep issues, language delays, and motor delays (Briegel et al., 2006) as well as concern for the increased risk for developing schizophrenia that having 22q11.2DS imparts (Beaton & Simon, 2011). Additional sources of
stress, as identified by Briègel and colleagues in their 2008 study of adolescents with 22q11.2DS and stress in parents concluded that social and attentional problems in their children were the most important behavior problems. Research on children with non-progressive neurodevelopmental disorders such as cerebral palsy found that child behavior was the best predictor of psychological well-being in caregivers (King, King, Rosenbaum & Goffin, 1999). Also, not to be overlooked are the health issues that a diagnosis of 22q11.2DS confers. These can include major heart and palate malformation necessitating multiple surgeries in infancy and childhood, hypocalcemia induced seizures, immunological abnormalities, and feeding difficulties (Kates, Tang, Antshel, & Fremont, 2015). While these stressors are identified in the context of child stress, these are, perhaps first and foremost, stressors for parents as well.

The role this stress has on the parent-child relationship within the context of 22q11.2DS has yet to be fully elucidated, but a study of mother-child dyads hints at the role bi-directionality may play in impacting this fundamental relationship (Weisman et al., 2015). Compared to their typically-developing (TD) counterparts, children with 22q11.2DS exhibited fewer positive emotions and their mothers’ interactions were more intrusive and less engaging. The mother-child dyad in the 22q11.2DS group showed less reciprocity overall than TD dyads.

Typically developing children have reduced natural killer T cell function in response to chronic but not short-term family stress (Wyman et al., 2007). Longitudinal studies of children of parents experiencing chronic stress indicate that rates of illness increase in those children by upwards of 40% and febrile illnesses by 77% (Caserta et al, 2008). The authors posit that the transmission of stress symptoms to children negatively impacts immune function leading to more illnesses. As children with 22q11.2DS often suffer from immune system deficiencies, mitigating stress in their parents may have especially important implications for reciprocal health and well-being.

Specific to the 22q11.2DS population is research by Prinzie and colleagues (2004) who found evidence that higher stress in parents was associated with higher levels of marital conflict and lower levels of parental consistency. Similarly, parents of children with other developmental disorders such as neural tube defects reported lower self-esteem, reduced parental satisfaction, and a lower internal locus of control. Parents’ perceived inadequacy in their roles as caregivers related to the severity of developmental and behavioral problems in their children (Spratt, Saylor & Macias, 2007). Research on parents of children with autism spectrum disorders have demonstrated that parental stress is a mediator of child behavior problems and poor parental self-efficacy, and that poor self-efficacy partially mediates the relationship between stress, depression, and anxiety in parents (Rezendes & Scarpa, 2011).

22q11.2DS and Parental Anxiety

Stress is the physiological and psychological response to changes in homeostasis, which can be endogenous or exogenous in origin. Anxiety is a psychological state that can accompany stress or a fear-inducing stimulus, but it can also remain even when the threatening stimulus is no longer present. Anxiety is the anticipation of possible future threats (Grupe & Nitschke, 2013). Anxiety is often associated with the physical manifestation of muscle tension and the psychological manifestation of attentional vigilance and this can elicit a powerful physiological stress response (APA, DSM-V, 2013). Coupled with avoidant behaviors, anxiety can exhaust coping resources and negatively affects function. When anxiety is excessive and persistent, it becomes pathological. Symptoms of anxiety disorders are wide-ranging, but can include panic attacks, refusal to speak in certain social situations, chest pain, irritability, sleep disturbances,
nightmares, restlessness, and fatigue that overall diminish quality of life (American Psychiatric Association, 2013).

Children with anxiety disorders are much more likely to have parents with anxiety disorders reflecting genetic and environmental contributions in the etiopathology of anxiety in families (Hettema, Neale & Kendler, 2001; Lieb et al., 2000; Fyer, Mannuzza, Chapman, Martin & Klein, 1995). Temperament styles including shyness, behavioral inhibition, and withdrawal are also associated with increased risk of developing an anxiety disorder. Very early environment appears to play a substantial role in the development of anxiety disorders with exposure to stressors such as lack of physical touch especially in the perinatal period being a key predictor (Steiner, 2002).

Parenting styles also fall under the umbrella of ‘environment’ and those styles that are highly controlling and/or highly critical are most closely associated with the development of anxiety disorders in TD children (Bögels & Brechman-Toussaint, 2006; McLeod, Wood & Weisz, 2007). Subtypes of anxiety disorders develop in response to environmental stressors. For example, separation anxiety disorder can develop after the loss of a loved one, and agoraphobia can develop following a traumatic event such as physical or sexual assault (American Psychiatric Association, 2013).

While parental behavior and circumstance can play a role in the development of anxiety in children, parental anxiety can also arise in the context of rearing a child with special needs. For example, parents of children with learning disabilities cope with sources of anxiety that predominantly focus on child development: concerns about adjustment in the context of academics; concerns about social functioning; fears about the child’s behavior; and worries about the future, especially when the child comes of age and how they will be able to function (Shetchman & Gilat, 2005). It is common for children with 22q11.2DS to have learning disabilities, low IQ, and socioemotional difficulties and thus it is likely that these same sources of anxiety apply to parents of children with 22q11.2DS as well (Swillen et al., 1999). Herscher and Brunner’s 2008 study of parents of children with 22q11.2DS supports this hypothesis with parents reporting anxiety concerning their children’s uncertain future as something they struggle with. Nearly 73% of parents participating in that study identified the current or potential presence of psychiatric illness in their children as the chief source of anxiety in raising a child with 22q11.2DS.

Anxiety is important to assess in parents for not only the health and well-being of the parent but also because an anxiety disorder in mothers is associated with higher rates of anxiety disorders in their children (McClure, Brennan, Hammen, & Le Brocque, 2001; Ginsburg & Schlossberg, 2002; Creswell, Schniering, & Rapee, 2005). Additionally, anxiety in multiple family members adds to the complexity of treatment. Parents with an anxiety disorder who have children with an anxiety disorder do not respond to treatment as well as patients without a child with anxiety (Reaven, Washington, Moody, Stern, Hepburn, & Blakeley-Smith, 2015). Thus, screening parents and children for anxiety and treating all affected family members is warranted.

**22q11.2DS and Parental Depression**

Depression can be defined as functional impairment due to feelings of sadness, emptiness, or irritability, often with somatic and cognitive changes such as weight fluctuations and negative appraisals. Children, adolescents, and adults appear to have the same core symptoms, but differ in their expression. Childhood symptoms of depression often include more oversleeping and more overeating, while psychomotor agitation is more common in adults (American Psychiatric Association, 2013).
As with anxiety, the etiopathology of depression is driven by a combination of factors that include genetics, temperament, disease processes, and experiences (American Psychiatric Association, 2013). Anxiety and depression are both associated with elevated metabolic activity of glucose in three large brain regions: the prefrontal, limbic and midbrain. Animal models of temperamental anxiety including rhesus monkeys have elevated metabolic brain activity as measured using positron emission tomography scans and functional magnetic resonance imaging in the prefrontal, limbic and midbrain regions associated with anxiety in humans. Anxious temperament was measured through a multigenerational pedigree process with findings suggesting that metabolic brain activity is the mediator between genes and the risk of developing anxiety and depression (Fox et al., 2015).

Further, the genetic predisposition for anxiety and mood disorders involve many of the same candidate genes. The clinical outcome appears to be epigenetic in nature and may rely on the developmental stage during which an individual is exposed to risk factors such as caregiver anxiety, abuse in childhood or experiencing a natural disaster (Kendler, Gardner, & Lichtenstein, 2008; Martin, Ressler, Binder, & Nemeroff, 2009). Similar to anxiety, a highly critical parenting style is implicated in the development of depression in children (Rapee, Schniering, & Hudson, 1997). Research on children whose parents suffer from depression exhibit high levels of self-doubt, self-blame and lack of hope for the future (Garber & Flynn, 2001). Additionally, the biggest risk factor for psychopathology, such as anxiety and depression in children is the presence of anxiety and depression in a parent (Connell & Goodman, 2002).

Parental depression often begins very early in the parenting role. Remarkably, birthing a typically developing child automatically puts a mother at a five-fold increased risk for developing depression (Dodge, 1990). Parental depression affects children in negative ways and one of the ways is poorer or less effective parenting practices (Garber & Flynn, 2001). For example, research shows that mothers suffering from depression are less emotionally responsive (Lombardo & Motta, 2008). Depression often presents in adults, whether parents or not, with symptoms ranging from low energy and poor concentration as seen in dysthymia to psychomotor retardation and loss of interest in all activities as can be observed in major depressive disorder (American Psychiatric Association, 2013). Furthermore, parents who are depressed often exhibit a parenting style that is more controlling and negative, and this is associated with higher rates of externalizing behaviors and poorer adjustment in their children compared to children of parents without depression (Fenning, Baker, Baker & Crnic, 2014). Research on children with borderline intellectual functioning shows that parents also report higher levels of depression than parents of TD children (Fenning et al., 2014). More complex psychopathology such as bipolar disorder in parents can also negatively affect emotional well-being and development of their children but this is not a forgone conclusion. Child characteristics such as temperament, developmental stage and sex of the child can moderate the risk of development of psychopathology in children. Levels of conflict and cohesion in the family further complicate the picture, exacerbating risk (Freed, Tompson, Wang, Otto, Hirschfeld-Becker, Nierenberg & Henin, 2015).

A diagnosis of 22q11.2DS carries with it a wide range of potential physical health problems and behavioral problems. As a result, parents of children with 22q11.2DS may be struggling with stress, anxiety, and depression coping with and worrying about their children, as evidenced by research on parental well-being in parents of children with other neurodevelopmental disorders.
**Bi-directionality**

As Bronfrenbrenner (1998) posits in the most recent version of his bioecological model, human development occurs through increasingly more complex reciprocal interactions (i.e. proximal processes) with the environment. These interactions are bi-directional in nature; the person and the environment influence each other. In the context of child rearing, parents are a significant aspect of the environmental experience of their children, and vice versa.

The presence of mental illness in children affects the well-being of their parents. In a study of children with a diagnosis of major depressive disorder (MDD), their mothers were more likely to parent in ways that are described as “overprotective,” with fathers of children with depression scoring lower on general functioning indices, which measure, for example, social and occupational functioning. Additionally, parent-child bonding and family functioning which includes providing affection and emotional support were also negatively impacted by the presence of MDD in children, as compared to controls (Stein et al., 2000).

Parents influence the well-being of their child through reciprocal interactions, through parental control of the child’s social environment, and through teaching and modeling behavior (Dodge, 1990). Studies connecting the reduction in oppositional defiant disorder and conduct disorder symptomology to decreases in poor parenting practices (inconsistent discipline, corporal punishment, etc.) as outlined in Shaffer, Lindheim, Kolko and Trentacosta’s 2013 study help support the bioecological model; when parenting practices improve, so too does child behavior. In examining the psychological components of the parent-child dyad, when parents are suffering from anxiety or depression, children often are experiencing shame, stigmatizing and bullying because of parent mental illness (Dam & Hall, 2016).

In terms of special populations of children, such as those suffering from neurodevelopmental disorders, one such study examined bi-directionality in borderline functioning children. As parents’ negative-controlling behavior increased, so too did negative behavior in their children, and across time, this increase in negative behavior was significantly greater than found in TD children. Mothers’ negative controlling behavior towards their five-year-old children was associated with an increase in negative child behavior by age six. Fathers’ negative-controlling behavior towards their children increased from age 5 to 6 in response to children’s negative behavior. Although the dynamic appears to differ in the order of effect with mothers’ parenting style increasing negative behavior in children and fathers in turn responding to this negative behavior, the findings provide evidence of bi-directionality with negative behavior increasing in a feed-forward fashion within the parent-child dyad (Fenning et al., 2014).

Bi-directionality may play out in the 22q11.2DS population due to the high emotionality that children with 22q11.2DS may display in interactions with parents as described by Schonherz, and colleagues (2014). Parents of children with 22q11.2DS may not respond as appropriately as they should when it comes to setting limits, thereby unintentionally depriving children with 22q11.2DS of an opportunity to practice self-regulation and social skills. Alternatively, parents’ perceived deficits in limit setting may be the result of past experiences with their children in which parents’ requests were either ignored or met with agitation by their child with 22q11.2DS. This bi-directional relationship can be more difficult to discern, in part due to the communication impairment that is so often a part of the symptomatology of 22q11.2DS: children may struggle to share that they are struggling.

Further research on bi-directional relationships in the context of 22q11.2DS is described by Prinzie and colleagues (2004) who identify some of the unique stressors in raising children with 22q11.2DS, such as the difficulty many of these children have in eating due to
velopharyngeal insufficiencies. The difficulty in feeding can be a profound stressor for parents, which can affect both the family context and the way in which parents then parent.

Bidirectionality is an important element for consideration in the 22q11.2DS population, in light of the variability in phenotypic expression of behavioral and emotional issues, and the role that parent-child relationships plays in child development (Prinzie et al., 2004). As Beaton and Simon suggest in their 2011 paper, studying anxiety in children with 22q11.2DS is important as it could potentially put children at risk for the development of psychotic disorders such as schizophrenia. Evidence in the literature indicates a bi-directional relationship between parents and children in terms of the transmission of stress, anxiety, and depression. Understanding this potential dynamic in children with 22q11.2DS and their parents is important for identifying ways to help parents, which in turn will help their children who are at risk for anxiety disorders and psychosis in adolescence and young adulthood.

The quality of early family experiences can have a profound impact on social emotional adjustment in adulthood. For example, anxiety is linked to a lack of affect, insensitive or unresponsive, hostile or rejecting parenting. Children with intellectual disabilities and neurological issues are often at higher risk for maltreatment, due to the amount of stress parents may be experiencing as a result of raising children with these problems (Overbeek et al., 2007; Spratt et al., 2007).

There appears to be a lack of research at least in terms of the presence of depression in the parents of children with 22q11.2 DS. This fact alone helps to drive the rationale for research in this area for a multitude of reasons, perhaps most importantly in terms of the effects parental well-being has on the parent-child dyad. Additionally, to date, no research has examined emotional intelligence in the 22q11.2DS population. As noted previously, higher levels of EI in children may offset parental stress, anxiety, and depression, and contribute to better quality parent-child relationships. The overarching aim of this study was to determine if there was a reciprocal relationship between stress, anxiety, and depression in children with 22q11.2DS and their parents and if emotional intelligence could serve as a buffer in this parent-child relationship.

**Hypotheses**

**Aim 1:** Examine whether differences exist between the experiences of stress, anxiety and depression in parents of children with 22q11.2DS and those of typically developing children.

**Hypothesis 1:** Parents of children with 22q11.2DS will report significantly greater symptoms of stress, anxiety, and depression than parents of TD children.

**Aim 2a:** Examine whether children with 22q11.2DS differ from their TD counterparts in levels of EI.

Hypothesis 2a: Children with 22q11.2DS will have lower levels of EI than their TD counterparts.

**Aim 2b:** To investigate the relationship between FSIQ scores on the WISC and scores of emotional intelligence in both populations.

**Hypothesis 2b:** There will be a positive correlation between FSIQ and EI scores in both populations of children.

**Aim 2c:** To investigate whether the presence or absence of 22q11.2DS will predict participants’ total EI scores on the BarOn EQi: YV.

**Hypothesis 2c:** Lower FSIQ and the presence of 22q11.2DS will result in lower scores of EI.

**Aim 3:** To investigate whether the diagnosis of 22q11.2DS will moderate the relationship between EI in children and stress, anxiety and depression in their parents.
**Hypothesis 3:** The presence of the 22q11.2DS diagnosis will result in lower EI in children, and higher stress, anxiety and depression in their parents.

**Methods**

**Participants**

Participants were parents and children currently enrolled in an ongoing study on behavioral, emotional, neurological and physiological correlates of 22q11.2DS. After approval was obtained through the University of New Orleans IRB committee, participants of the larger SCAN Laboratory were recruited from various organizations, including the Louisiana 22q Support Network and the 22q Texas/VCFS Texas, Inc., as well as through social media (Twitter, Facebook), flyers posted throughout the Greater New Orleans region and through word-of-mouth. Thus, families both with and without a 22q11.2DS child were recruited for participation.

For the purposes of this study, a total of 42 parents of 22q11.2DS children, along with 20 parents of TD children were included in the analyses. Child participants included 25 with 22q11.2DS (age range of 7.87 to 16.77 years old, \( M = 12.36, SD = 2.42 \)), and 17 who were TD (age range of 7.17 to 16.82 years old; \( M = 10.65, SD = 2.74 \)). All children with 22q11.2DS had been previously diagnosed with the chromosome deletion by independent medical professionals and provided documentation as such prior to the start of the study. Assessments were administered by advanced doctoral students who had completed graduate level coursework and had previous clinical experience working with children with 22q11.2DS.

**Measures**

**Stress:** Parental stress was measured through the Depression Anxiety Stress Scale-Long Form (DASS-42), a 42 item self-report questionnaire with numerical Likert-style response options (e.g., 0 = *did not apply to me at all,* 3 = *applied to me very much or most of the time*). The DASS-42 is comprised of three subscales of 14 items each, which correspond to depression, anxiety, and stress (Nieuwenhuijsen, de Boer, Verbeek, Blonk & van Dijk, 2003). Given that the current study includes more extensive measures of anxiety and depression (see below), only the Stress subscale (“I tended to over react to situations,” “I felt that I was rather touchy” (in the past week)) was used for analysis. Scores on the Stress subscale are divided into “normal” (scores of 0-14), “mild” (scores of 15-18), “moderate” (scores of 19-25), “severe” (scores of 26-33) and “extremely severe” (scores of 34 or greater) (Lovibond & Lovibond, 1995).

Past studies have indicated excellent reliability on the Stress subscale in both normative (Cronbach’s \( \alpha = 0.9 \); Lovibond & Lovibond, 1995) and clinical (Cronbach’s \( \alpha = 0.95 \)) samples (Antony, Bieling, Cox, Enns & Swinson, 1998). In the current study, internal consistency for parents of children with 22q11.2DS on the Stress subscale was \( \alpha = 0.939 \). For parents of TD children, internal consistency was \( \alpha = 0.916 \).

**Depression:**

Parental depression was measured using the Beck Depression Inventory-Second Edition (BDI-II; Beck, Steer & Brown, 1996), a 21 item self-report which uses Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text-Revised (DSM-IV-TR; APA, 2000) criteria to assess for the presence of clinical depression. Each of the 21 items is comprised of four statements that increase in severity (e.g., 0 = “I do not feel sad;” 1 = “I feel sad;” 2 = “I am sad all the time and I can't snap out of it;” 3 = “I am so sad and unhappy that I can't stand it.”) Participants are asked to select the statement which most accurately describes how they have felt in the last two weeks. Scores for each item range from 0 to 3. Items are summed to create a total from 0 (no depression) to 63 (severe depression) (Beck, Steer, & Brown, 1996).
The BDI-II has been normed on clinical samples ($\alpha = 0.92$), non-clinical samples ($\alpha = 0.90$) and medical samples ($\alpha = 0.93$) and are excellent (Wang & Gorenstein, 2013). In the current study, Cronbach’s alpha for parents of children with 22q11.2DS was $\alpha = 0.894$, and for parents of TD children, $\alpha = 0.916$.

**Anxiety:**

Parental anxiety was measured using the Beck Anxiety Inventory (BAI: Beck & Steer, 1993). Consisting of 21 items related to subjective, somatic or panic-related symptoms of anxiety, participants select the response which best describes past-month symptoms, ranging from 0 (“not at all”) to 3 (“Severely- it bothered me a lot”). Responses are summed to produce a single score, which can range from 0 to 63. Scores between 0 and 21 indicate little or no anxiety. Scores from 22 to 35 indicate moderate anxiety. Scores exceeding 36 indicate potentially concerning levels of anxiety. Internal consistency for the BAI in past studies have been good in non-clinical samples ($\alpha = 0.88$; Lovibond & Lovibond, 1995b) to excellent (e.g., $\alpha = 0.92$; Beck, Epstein, Brown, & Steer, 1988). Internal consistency for parents of children with 22q11.2DS on the BAI in the current study was $\alpha = 0.871$. For parents of TD children, internal consistency was $\alpha = 0.858$.

**Emotional Intelligence:**

Emotional Intelligence in both 22q11.2DS and TD children was measured using the BarOn Emotional Quotient Inventory: Youth Version (BarOn EQ i: YV; Bar-On & Parker, 2000a). The EQi:YV is a 60-item self-report questionnaire designed for use in children age 7 to 18 years, and includes 8 subscales with both gender and age-specific norms. The Intrapersonal Scale measures the extent to which an individual understands their own emotions, as well as how able they are to express and communicate these emotions to others (e.g.: “It is easy to tell people how I feel.”) The Interpersonal Scale measures the ability of an individual to understand and appreciate the feelings of others (e.g.: “I am good at understanding the way other people feel.”) The Adaptability Scale measures how flexible, realistic and effective an individual is at managing change (e.g.: “I try to use different ways of understanding hard questions.”) The Stress Management Scale measures how calmly and effectively an individual works under pressure (e.g.: “I can stay calm when I am upset.”) The Total EQ Scale measures an individual’s effectiveness of dealing with daily demands and is a measure of an individual’s overall EI. The General Mood Scale measures optimism (e.g.: “I enjoy having fun,”) (Bar-On & Parker, 2000b).

Questions are scored on a 4-point Likert-scale, with response options ranging from 0 (“Very Seldom True of Me”) to 3 (“Very Often True of Me”). Higher scores are representative of higher levels of EI. Scores are standardized, such that the mean score is 100, with a standard deviation of 15. To interpret EI holistically requires considering not just individual scale scores, but the pattern of scale scores. If an individual does not score below 90, there is no indication of problems with EQ. However, indicative of deficiencies in EI are multiple scale scores below 90. The BarOn EQ i: YV has been normed across cultures and is found to be both valid and reliable. Internal consistency on the scales in prior studies has ranged from fair ($\alpha = 0.65$; Intrapersonal scale, males ages 7-9 years (Bar-On & Parker, 2000b)) to excellent ($\alpha = 0.90$; General Mood scale, females, ages 16-18 years, (Bar-On & Parker, 2000b)). In this study, internal consistency for children with 22q11.2DS on the BarOn EQi: YV was $\alpha = 0.884$, and for TD children, $\alpha = 0.952$.

**Intelligence Quotient:**

The Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV) was administered to child participants by a trained laboratory research assistant in the doctoral
program at the University of New Orleans. Designed for use with children aged 6 through 16, the WISC-IV is comprised of 10 core subtests and 5 optional subtests. These subtest scores are converted into four index scores: Verbal Comprehension, Perceptual Reasoning, Working Memory, and Processing Speed. Additionally, the WISC-IV provides a Full-Scale Intelligence Quotient (FSIQ) score (Wechsler, 2003; Niolon, 2005).

The Verbal Comprehension Index is a measure of how well a child is able to listen, retrieve information, use reasoning, and express themselves. Verbal Comprehension scores are predictive of school readiness and achievement (Wechsler, 2004). The Perceptual Reasoning Index measures how well a child can examine a problem, how developed their visual-motor and spatial skills are, the ability to organize, and problem solve. It is a good assessment of fluid reasoning. The Working Memory Index assesses short-term memory and concentration. Finally, the Processing Speed Index, as its name suggests, measures the speed at which a child processes new information, as well as their ability to focus attention, discriminate and order visual information (Wechsler, 2004; Niolon, 2005).

Of particular interest for the current study, the WISC-IV was designed to link to emotional intelligence as measured by the BarOn EQi: YV. The reliability for the WISC-IV ranges from $\alpha = 0.88$ on Processing Speed to $\alpha = 0.97$ on FSIQ, respectively, across both general and clinical samples (Williams, Weiss, & Rolfhus, 2003). Internal consistency in the current study for children with 22q11.2DS was $\alpha = 0.848$ on the WISC-IV, and for TD children, internal consistency was $\alpha = 0.744$. 

Results

Preliminary Analyses

A series of bivariate correlations were computed to examine associations between main study variables. Means, standard deviations and correlations for the sample are presented in Table 1 while correlations are presented by diagnosis in Tables 2 and Table 3. In the 22q11.2DS population sample, higher scores on the Stress Scale of the DASS-42 were associated with statistically significantly higher BAI (anxiety) scores ($r = .52$, $p < .001$). Higher scores on the Stress Scale were associated with statistically significantly higher scores on the BDI (depression) ($r = .79$, $p < .001$). Lastly, higher scores on the BAI were associated with statistically significantly higher scores on the BDI ($r = .46$, $p < .001$). In the typically developing population sample, higher scores on the Stress Scale of the DASS-42 were associated with statistically significantly higher scores on the BDI ($r = .73$, $p < .05$)

Diagnostic Status and Intellectual Functioning

T-tests were used to compare intellectual functioning of children with 22q11.2DS with TD children. Consistent with existing research, results revealed that the presence of 22q11.2DS resulted in significantly lower scores on all measures: on average, children with 22q11.2DS scored lower on the Verbal Comprehension Composite of the WISC-IV ($M = 82.68$, $SE = 2.698$), than their TD counterparts ($M = 107.76$, $SE = 3.129$). This difference, $-25.085$, 95% CI $[-33.508, -16.662]$ was significant $t(40) = -6.019$, $p < .001$, $d = -1.387$.

Additionally, children with 22q11.2DS scored lower on the Working Memory Composite of the WISC-IV ($M = 76.04$, $SE = 3.129$) than their TD counterparts ($M = 102.06$, $SE = 2.879$). This difference, $-26.019$, 95% CI $[-35.169, -16.869]$ was significant $t(40) = -5.747$, $p = .001$, $d = -1.353$. On the Processing Speed Composite of the WISC-IV, 22q11.2DS children scored lower ($M = 72.36$, $SE = 2.224$) than their TD counterparts ($M = 101.88$, $SE = 3.475$). This difference, $-29.522$, 95% CI $[-37.465, -21.580]$ was significant $t(40) = -7.512$, $p = .001$, $d = -1.539$. On average, children with 22q11.2DS scored lower on the FSIQ of the WISC-IV ($M = 71.56$, $SE = 2.717$) than their TD counterparts ($M = 105.88$, $SE = 3.035$). This difference, $-34.322$, 95% CI $[-42.689, -25.956]$ was significant $t(40) = -8.291$, $p = .001$, $d = -1.60$. 
Table 1. Means and standard deviations of main study variables by diagnostic group

<table>
<thead>
<tr>
<th>Dx Group</th>
<th>22q11.2DS</th>
<th>TD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>M (SD)</td>
</tr>
<tr>
<td>DASS-SS</td>
<td>40</td>
<td>9.98 (8.02)</td>
</tr>
<tr>
<td>BDI</td>
<td>40</td>
<td>8.40 (7.44)</td>
</tr>
<tr>
<td>BAI</td>
<td>40</td>
<td>6.38 (6.90)</td>
</tr>
<tr>
<td>BarOn Intra</td>
<td>29</td>
<td>14.10 (2.96)</td>
</tr>
<tr>
<td>BarOn Inter</td>
<td>29</td>
<td>36.79 (6.54)</td>
</tr>
<tr>
<td>BarOn Stress Man</td>
<td>29</td>
<td>31.41 (5.72)</td>
</tr>
<tr>
<td>BarOn Adapt</td>
<td>29</td>
<td>25.62 (6.27)</td>
</tr>
<tr>
<td>BarOn Mood</td>
<td>29</td>
<td>45.86 (7.92)</td>
</tr>
<tr>
<td>Total EQ</td>
<td>25</td>
<td>91.00 (11.11)</td>
</tr>
<tr>
<td>WISC Verbal</td>
<td>25</td>
<td>82.68 (13.49)</td>
</tr>
<tr>
<td>WISC Percept.</td>
<td>25</td>
<td>75.68 (13.52)</td>
</tr>
<tr>
<td>WISC WM</td>
<td>25</td>
<td>76.04 (15.87)</td>
</tr>
<tr>
<td>WISC ProSpeed</td>
<td>25</td>
<td>72.36 (11.12)</td>
</tr>
<tr>
<td>FSIQ</td>
<td>25</td>
<td>71.56 (13.59)</td>
</tr>
</tbody>
</table>

Note: †p < .10; *p < .05; **p < .01; ***p < .001. DASS-SS = Depression Anxiety Stress Subscale; BDI = Beck Depression Inventory; BAI = Beck Anxiety Inventory; Bar-On Intra = Intrapersonal subscale; Bar-On Inter = Interpersonal subscale; Bar-On Stress Man = Stress Management subscale; Bar-On Adapt = Adaptiveness subscale; Bar-On Mood = General Mood subscale; Total EQ = Bar-On Emotional IQ total; WISC Percept = Perceptual Reasoning; WISC WM = Working Memory; WISC ProSpeed = Processing speed; FSIQ = WISC full-scale IQ.

Table 2. Correlations of Main Study Variables by Diagnosis: 22q

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. DASS-SS</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. BDI</td>
<td>.79**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. BAI</td>
<td>.52**</td>
<td>.46**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Bar-On Intra</td>
<td>.11</td>
<td>.13</td>
<td>.31</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Bar-On Inter.</td>
<td>.18</td>
<td>.01</td>
<td>.17</td>
<td>.19</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Bar-On StressMan</td>
<td>-.17</td>
<td>.04</td>
<td>-.17</td>
<td>.02</td>
<td>.02</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Bar-On Adapt</td>
<td>.38*</td>
<td>.24</td>
<td>.25</td>
<td>.40*</td>
<td>.56**</td>
<td>-.01</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Bar-On Mood</td>
<td>-.06</td>
<td>-.07</td>
<td>-.03</td>
<td>.30</td>
<td>.65**</td>
<td>.15</td>
<td>.43*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Total EQ</td>
<td>.22</td>
<td>.17</td>
<td>.23</td>
<td>.62**</td>
<td>.72**</td>
<td>.35</td>
<td>.82**</td>
<td>.61**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. WISC Verbal</td>
<td>.13</td>
<td>.19</td>
<td>.12</td>
<td>-.10</td>
<td>.16</td>
<td>.06</td>
<td>.17</td>
<td>.32</td>
<td>.14</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. WISC Percept.</td>
<td>.35</td>
<td>.38</td>
<td>.27</td>
<td>.12</td>
<td>.08</td>
<td>-.07</td>
<td>.48*</td>
<td>.05</td>
<td>.27</td>
<td>.48*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. WISC WM</td>
<td>-.07</td>
<td>-.07</td>
<td>.01</td>
<td>.16</td>
<td>.11</td>
<td>.06</td>
<td>.40</td>
<td>.13</td>
<td>.29</td>
<td>.66**</td>
<td>.66**</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. WISC ProSpeed</td>
<td>.37</td>
<td>.37</td>
<td>.23</td>
<td>.10</td>
<td>.14</td>
<td>-.01</td>
<td>.27</td>
<td>.36</td>
<td>.21</td>
<td>.63**</td>
<td>.62**</td>
<td>.57**</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>14. FSIQ</td>
<td>.20</td>
<td>.24</td>
<td>.17</td>
<td>.06</td>
<td>.13</td>
<td>.01</td>
<td>.40</td>
<td>.23</td>
<td>.25</td>
<td>.85**</td>
<td>.82**</td>
<td>.88**</td>
<td>.78**</td>
<td>1</td>
</tr>
</tbody>
</table>

Note: †p < .10; *p < .05; **p < .01; ***p < .001. DASS-SS = Depression Anxiety Stress Subscale; BDI = Beck Depression Inventory; BAI = Beck Anxiety Inventory; Bar-On Intra = Intrapersonal subscale; Bar-On Inter = Interpersonal subscale; Bar-On Stress Man = Stress Management subscale; Bar-On Adapt = Adaptiveness subscale; Bar-On Mood = General Mood subscale; Total EQ = Bar-On Emotional IQ total; WISC Percept = Perceptual Reasoning; WISC WM = Working Memory; WISC ProSpeed = Processing speed; FSIQ = WISC full-scale IQ.
Table 3. Correlations of Main Study Variables by Diagnosis: TD

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. DASS-SS</td>
<td>.73**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. BDI</td>
<td>-.32</td>
<td>-.22</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. BAI</td>
<td>.03</td>
<td>.05</td>
<td>-.06</td>
<td>.1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Bar-On Inter.</td>
<td>-.10</td>
<td>-.01</td>
<td>-.40</td>
<td>.44</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Bar-On Stress Man</td>
<td>.28</td>
<td>-.06</td>
<td>-.20</td>
<td>.53*</td>
<td>.42</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Bar-On Adapt</td>
<td>.38</td>
<td>.74</td>
<td>-.15</td>
<td>.69**</td>
<td>.81**</td>
<td>.53*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Bar-On Mood</td>
<td>.03</td>
<td>.77</td>
<td>-.38</td>
<td>.29</td>
<td>.69*</td>
<td>.62**</td>
<td>.52*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Total EQ</td>
<td>.07</td>
<td>.03</td>
<td>-.22</td>
<td>.87**</td>
<td>.77**</td>
<td>.74**</td>
<td>.91**</td>
<td>.60*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. WISC Verbal</td>
<td>-.00</td>
<td>-.12</td>
<td>-.20</td>
<td>.28</td>
<td>.24</td>
<td>.25</td>
<td>.27</td>
<td>.46</td>
<td>.58*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. WISC Percept</td>
<td>-.08</td>
<td>-.26</td>
<td>-.30</td>
<td>.49*</td>
<td>.28</td>
<td>.44</td>
<td>.25</td>
<td>.27</td>
<td>.46</td>
<td>.58*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. WISC WM</td>
<td>-.06</td>
<td>-.43</td>
<td>.08</td>
<td>.34</td>
<td>.01</td>
<td>.40</td>
<td>-.01</td>
<td>.00</td>
<td>.25</td>
<td>.46</td>
<td>.60*</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. WISC ProSpeed</td>
<td>.34</td>
<td>.08</td>
<td>-.11</td>
<td>.45</td>
<td>.06</td>
<td>.19</td>
<td>.34</td>
<td>.02</td>
<td>.34</td>
<td>.30</td>
<td>.55*</td>
<td>.11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>13. FSIQ</td>
<td>.05</td>
<td>-.22</td>
<td>-.19</td>
<td>.51*</td>
<td>.22</td>
<td>.38</td>
<td>.30</td>
<td>.16</td>
<td>.45</td>
<td>.81**</td>
<td>.89**</td>
<td>.68**</td>
<td>.64**</td>
<td>1</td>
</tr>
</tbody>
</table>

Note: †p < .10; *p < .05; **p < .01; ***p < .001. DASS-SS = Depression Anxiety Stress Subscale; BDI = Beck Depression Inventory; BAI = Beck Anxiety Inventory; Bar-On Intra = Intrapersonal subscale; Bar-On Inter = Interpersonal subscale; Bar-On Stress Man = Stress Management subscale; Bar-On Adapt = Adaptiveness subscale; Bar-On Mood = General Mood subscale; Total EQ = Bar-On Emotional IQ total; WISC Percept = Perceptual Reasoning; WISC WM = Working Memory; WISC ProSpeed = Processing speed; FSIQ = WISC full-scale IQ

**Hypothesis 1**

Hypothesis 1 stated that parents of children with 22q11.2DS would report significantly greater symptoms of stress, anxiety, and depression than parents of typically developing children. To test this hypothesis, multivariate testing and a series of independent t-tests were conducted.

Multivariate testing using Pillai’s Trace revealed that there was no significant effect of diagnosis on the reports of stress, anxiety, and depression reported by parents: $V = .027, F(3,54) = .504, p = .681$.

On average, stress levels did not differ between parents of children with 22q11.2DS ($M = 9.98, SE = 1.268$) and parents of typically developing children ($M = 7.58, SE = 1.55$), $t(57) = 1.125$, difference of 2.396, 95% CI [-1.868, 6.660], $d = .312, p = .265$. Parents of 22q11.2DS ($M = 6.38, SE = 1.04$) did not report significantly higher levels of anxiety than parents of typically developing children ($M = 5.15, SE = 1.24$), 1.23, 95% CI [-2.24, 4.70], $t(60) = .710, p = .480, d = .193$. When examining depression, the difference between parents of 22q11.2DS ($M = 5.80, SE = 1.41$), and parents of TD children ($M = 7.53, SE = 1.85$) was again not significant at .88, 95% CI [-3.33, 5.09], $t(59) = .417, p = .678, d = .116$.

**Hypothesis 2a**

Hypothesis 2a predicted that children with 22q11.2DS would have lower scores of EI as measured by the BarOn EQi: YV than their typically developing counterparts. This hypothesis was tested through the use of multivariate testing and independent t-tests (see Table 1).
Using Pillai’s Trace, there was a significant effect of diagnosis on the scales of the BarOn EQi: YV: \( V = 0.305, F(5,42) = 3.685, p = .007. \)

On average, participants with 22q11.2DS scored significantly lower (\( M = 91.00, SE = 1.28 \)) on the Total EQ Scale of the BarOn EQi: YV than their TD counterparts (\( M = 104.82, SE = 2.31 \)), \( t(46) = -3.270, .879, 95\% CI [-12.92, -3.10], p = .002, d = .879 \). Significantly lower scores were also found for 22q11.2DS participants (\( M = 36.80, SE = 1.22 \)) on the Interpersonal Scale of the BarOn EQi: YV than their TD counterparts (\( M = 40.37, SE = 1.30 \), difference of -3.58, 95\% CI [-7.16, 0.11], \( t(46) = -1.95, p = .057, d = .559 \)). Significantly lower scores were also found on the Adaptability Scale for 22q11.2DS children over their TD counterparts (\( M = 25.62, SE = 1.16 \); \( M = 31.84, SE = 1.29 \). This difference, -6.221, 95\% CI [-9.81, -2.64], \( t(46) = -3.50, p = .001 \), represented a large effect size, \( d = .926 \). The last significant difference was found on the Stress Management Scale, where participants with 22q11.2DS scored lower (\( M = 31.41, SE = 1.06 \) than their TD counterparts (\( M = 36.11, SE = 1.70 \)). This difference, -4.691, 95\% CI [-8.51, -0.88], \( t(46) = -2.48, p = .02 \), represented a medium effect size, \( d = .694 \).

There was no statistically significant difference between 22q11.2DS children (\( M = 14.10, SE = .55 \)) and their TD counterparts (\( M = 15.84, SE = 1.15 \)) on the Intrapersonal Scale of the BarOn EQi: YV: \( t(46) = -1.36, p = .185, d = .439 \). Additionally, there was no significant difference between participants with 22q11.2DS (\( M = 45.86, SE = 1.36 \)) and their TD counterparts (\( M = 46.89, SE = 2.04 \)) on the General Mood Scale, \( t(46) = -0.44, p = .66, d = .130 \).

**Hypothesis 2b**

To test the hypothesis that FSIQ scores from the WISC-IV would be positively associated with EI scores from the BarOn EQi: YV, Pearson correlations were conducted. Results indicated that there was a significant positive correlation between FSIQ and EI in the 22q11.2DS population: \( r = .415, p = .03 \). However, there was no significant correlation between FSIQ and EI in the TD population, \( r = .45, p = .07 \).

**Hypothesis 2c**

A multiple regression was carried out to investigate whether FSIQ and the presence or absence of 22q11.2DS could significantly predict participants’ total EI scores from the BarOn EQi: YV. The results of the regression indicated that the model explained 21.4\% of the variance and that the model was a significant predictor of EI, \( F(2,39) = 5.298, p = .009 \). However, on their own, neither FSIQ (unstandardized b = .249, 95\% CI [-.114, .612], SE b = .179, \( \beta = .325, p = .173 \)) nor diagnosis (unstandardized b = -5.274, 95\% CI [-20.933, 10.385], SE b = -.160, \( \beta = -.160, p = .500 \)) uniquely predicted EI.

**Hypothesis 3**

Finally, hypothesis 3 stated that the diagnosis of 22q11.2DS in children would moderate the relationship between EI in children and stress, anxiety, and depression in their parents. Specifically, it was hypothesized that the presence of the 22q11.2DS diagnosis would result in lower EI in children, and higher stress, anxiety, and depression in parents. Three moderation models were conducted using the PROCESS (Hayes, 2012) plug-in for SPSS. Prior to analysis, EI was mean-centered, and an EI x 22q11.2DS interaction term was created.

The first moderation model examined the 22q11.2DS diagnosis, children’s EI, and their interaction as predictors of parents’ stress scores. Specifically, it was predicated that the diagnosis of 22q11.2DS and lower EI would predict higher levels of stress in parents. Results indicated that the overall model did not significantly predict variance in measures of stress in parents, \( F(3, 41) = .672, p = .5741 \). Neither EI nor the presence of 22q11.2DS in children predicted stress in parents. The interaction term was also non-significant, indicating that there
was no moderating relationship of 22q11.2DS on the relationship between parental stress and EI. (see Table 4).

Table 4: Regression analyses predicting parental stress from 22q11.2DS diagnosis and EI

<table>
<thead>
<tr>
<th></th>
<th>B (SE)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>22q11.2DS Presence</td>
<td>-.14 (.265)</td>
<td>-.054</td>
<td>.958</td>
</tr>
<tr>
<td>EI (centered)</td>
<td>.22 (.18)</td>
<td>1.212</td>
<td>.233</td>
</tr>
<tr>
<td>22q11.2DS x EI (centered)</td>
<td>-.27 (.37)</td>
<td>-.726</td>
<td>.472</td>
</tr>
</tbody>
</table>

Note: The overall regression model did not predict a significant amount of variance, full-model $R^2=.112$; $F(3, 41)=.672, p<.001$. *$p<.05$, **$p<.001$.

The second model examined whether the 22q11.2DS diagnosis moderated the relationship between EI in children and parental anxiety. Specifically, it was predicted that the presence of the diagnosis would result in lower EI, and higher parental anxiety. Results indicated that EI, 22q11.2DS, and their interaction explained 25.9% of the variance in the BAI. The overall model was significant as a predictor of variance in the BAI (anxiety), ($F(3,44) = 3.110, p = .036$) and higher EI was associated with higher parental anxiety ($p = .007$). However, the presence of 22q11.2DS in children did not predict anxiety in parents, nor did it moderate the effect of EI on anxiety (see Table 5).

Table 5. Regression analysis predicting parental anxiety from 22q11.2DS diagnosis and EI

<table>
<thead>
<tr>
<th></th>
<th>B (SE)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>22q11.2DS Presence</td>
<td>.50 (2.03)</td>
<td>.246</td>
<td>.807</td>
</tr>
<tr>
<td>EI (centered)</td>
<td>.37 (.13)</td>
<td>2.811</td>
<td>.007</td>
</tr>
<tr>
<td>22q11.2DS x EI (centered)</td>
<td>-.089 (.27)</td>
<td>-.330</td>
<td>.743</td>
</tr>
</tbody>
</table>

Note: The overall regression model predicted a significant amount of variance, full-model $R^2=.259$; $F(3, 44) =3.110, p<.001$. *$p<.05$, **$p<.001$.

The third and final moderation model examined 22q11.2DS as a moderator of the relationship between EI in children and parental depression. Specifically, it was predicted that the presence of 22q11.2DS would result in lower EI levels and higher parental depression. Results indicated that the overall model did not significantly predict higher levels of depression in parents, $F(3,43) =2.98, p =.827$. Additionally, neither the 22q11.2DS diagnosis, EI, nor the interaction between the two were significant predictors of parental depression (Table 6). Thus, the third hypothesis was not supported in the current sample.
Table 6: *Regression analysis predicting parental depression from 22q11.2DS diagnosis and EI*

<table>
<thead>
<tr>
<th></th>
<th>B (SE)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>22q11.2DS Presence</td>
<td>.60 (2.40)</td>
<td>.251</td>
<td>.803</td>
</tr>
<tr>
<td>EI (centered)</td>
<td>.12 (.20)</td>
<td>.569</td>
<td>.572</td>
</tr>
<tr>
<td>22q11.2DS x EI (centered)</td>
<td>-.34 (.42)</td>
<td>-.807</td>
<td>.424</td>
</tr>
</tbody>
</table>

*Note: The overall regression model predicted a significant amount of variance, full-model $R^2=.068$; $F(3, 43) =.298$, $p<.001$. *$p<.05$, **$p<.001$.***
Discussion

The overarching goal of this study was to explore the possible role EI in children with 22q11.2DS may play in the context of the parent-child relationship, as measured by self-reports of stress, anxiety, and depression in parents. No study to date has examined EI in children with 22q11.2DS.

The first hypothesis, that parents of children with 22q11.2DS would report significantly higher levels of stress, anxiety, and depression was not supported by the data. The lack of significant levels of stress in parents of 22q11.2DS in this study mirrors the findings of Briegel, Schneider & Schwab’s 2006 examination of stress in parents of infants with 22q11.2DS who also found no significant difference in stress levels between these parents and parents of TD children. They followed-up their research with a study examining anxiety of parents in adolescents with 22q11.2DS in 2008 and concluded that these parents did have higher levels of stress than the general population, but that, interestingly, parents of children with 22q11.2DS reported normal levels of life satisfaction, as measured by a subscale of the Freiburger Personality Inventory. Future research in this area of parental stress, anxiety, and depression should examine whether these levels remain the same or change over time especially as demands of child rearing shift across developmental stages and as children encounter constantly evolving social and academic expectations and norms.

Another possible explanation for the lack of significance in reports of stress, anxiety, and depression may be due to adaptation. It is possible that parents have adapted to the diagnosis of 22q11.2DS to such an extent that results are an accurate barometer of psychological functioning. Future studies should consider controlling for the amount of time parents have been aware of the diagnosis, although the diagnosis itself may be lowering stress, anxiety, and depression, in light of the research previously discussed surrounding the process of diagnosis and the role that “not knowing” can play in increasing stress (Goodwin, McCormack & Campbell, 2017). For example, one of the families in the present study had received a diagnosis only the month before participating, when this child was already ten years of age. These parents also reported non-significant levels of stress, anxiety, and depression.

No data reflecting severity of the symptoms of 22q11.2DS was included in this study, nor were comorbid medical conditions controlled for in the statistical models. These would all theoretically impact parents to varying degrees. Having a child with high levels of ADHD, low IQ and severe heart troubles would arguably create more opportunity for stress, anxiety, and depression in parents than a child who is behaviorally nearly asymptomatic and may have medical issues that do not require extensive surgery or follow-up care. In examining the levels of functioning of the children who participated in this study as a source of bias in the results, this must be considered as a possible limitation. Many of the children with 22q11.2DS traveled to the University of New Orleans from across the United States. As a result, there may be limited generalizability of results due to this sample being well enough (both physically and psychologically) to travel as far as they did to participate, and this may also help explain the lack of findings in the parent results.

Additional explanations for parent results include social desirability and contextual factors. Social desirability in this study may have skewed parent responses to their reported levels of stress, anxiety, and depression due to parents’ knowledge that their responses would be reviewed and connected to their child’s data. Contextual factors would include, but are not limited to, the novelty of traveling to a new city for testing, meeting new people (specifically, the graduate students and principal investigator of the larger SCAN Lab study), and/or the possibility
of getting a break from some of the reminders of the day-to-day stressors that might have impacted parents.

Self-report measures may not be the optimal metric of stress even if the person is experiencing stress. For example, Barker, Greenberg, Seltzer & Almeida (2012) found that parents of children with serious mental illnesses had higher stress levels as measured by cortisol. Future research in the area of stress in parents of children with 22q11.2DS should include this physiological measurement. Psychological measures of stress associated with anxiety and depression, combined with diagnostic interviews of parents might be more useful in getting a clearer picture of psychological functioning. Further studies might look at how self-report measures are phrased, as the DASS 42 looked at symptoms over the past week, and the BAI inquired about symptoms over the last month, while the BDI asked about how participants were currently feeling.

Lastly, it is possible that the parent data is an accurate representation of how parents are coping. No questions were asked pertaining to social support networks; parents may be very well supported by family and friends, thereby reducing the stress, anxiety, or depression that could potentially be elicited initially by the 22q11.2DS diagnosis. Additionally, parents may have previously or even currently been in treatment for stress, anxiety, or depression at the time of enrollment in this study, and that may be what the data is reflecting.

Hypothesis 2a, which stated that the presence of 22q11.2DS would result in lower component scores on the scales of the BarOn EQi: YV was supported by the analyses. Children with 22q11.2DS scored significantly lower than TD children on several subscales of the BarOn EQi: YV, including the Total EQ scale, as well as the Interpersonal, Adaptability and Stress Management scales. However, there was no significant difference in the two child populations on the Intrapersonal or General Mood Scales of the BarOn EQi: YV.

There are many possible explanations for this study’s results that children with 22q11.2DS scored significantly lower on subscales of the BarOn EQi: YV. The BarOn EQi: YV perceives EI as an ability to handle relationships, and in terms of traits (such as optimism) and is measured via self-reports (Brackett, Rivers, Shiffman, Lerner, and Salovey, 2006). Meanwhile, EI as defined by Mayer, Salovey and Caruso in the Mayer Salovey Caruso Emotional Intelligence Test (MSCEIT) is considered in terms of abilities, as a set of mental skills that are measured by performance tests (Mayer, Salovey & Caruso, 2000). The association between these two measures of EI are low (rs ≤ .22) and as such, a different measure of EI in this study may have yielded different results (Brackett & Mayer, 2003). Additionally, a comparison of the results of these two different measures on this population could potentially yield deeper insight into explaining why functioning is at the level it is, as these measures are assessing different skills.

The finding that EI and Adaptability scores were both lower in the children with 22q11.2DS echoes the hypothesis that this population is more vulnerable to stress (Beaton & Simon, 2011) and have difficulty adapting to change or day-to-day demands (Angkustsiri et al, 2012). This finding also supports the theory that EI is a contributor to adaptive functioning. These findings regarding lower EI and Stress Management also provide support for previous research suggesting that higher levels of EI are related to lower stress levels (Singh & Sharma, 2012; Salovey et al., 2002).

As for explanations for the lower scores on the other scales of the BarOn EQi: YV, future research might include honing in on skill sets associated with each and measuring them more specifically (i.e. testing for Stress or Interpersonal skills with separate measures designed
specifically for these constructs). However, in light of the huge behavioral phenotypic range of children with 22q11.2DS, this finding in variation of scores on the scales may not be that surprising.

Hypothesis 2b predicted that higher Full-Scale IQ scores on the WISC-IV would be related to higher scores of EI, and this was supported in the TD children. This lends more evidence to the proposal that EI and IQ are related. Tables 1 and 2 offer more insight into the relationship between the IQ scores and the components of EI, as conceptualized and measured by the BarOn EQi: YV. Taking longitudinal measures of EI would also be of value, especially in light of the decline reported by Gothelf and colleagues in Verbal IQ which appear to be connected to prodromal schizophrenia (2007). If EI is related to intelligence, as these results suggested, it is interesting to consider what other factors EI may be related to and should be considered for measurement in future studies. For example, identifying whether the number of friends increases with either higher EI or IQ would be interesting to explore.

Hypothesis 2c predicted that FSIQ and the presence or absence of 22q11.2DS could significantly predict participants’ total EI scores from the BarOn EQi: YV. However, the results did not fully support this hypothesis. While children with 22q11.2DS scored significantly lower than TD children on both FSIQ and EI, further analyses indicated that neither FSIQ nor the presence of 22q11.2DS significantly predicated EI.

The final hypothesis was that the presence of the 22q11.2DS diagnosis would result in lower EI in children, and higher stress, anxiety, and depression in parents. Results did not support this hypothesis. However, it should again be noted that results from this study represent a “snap shot” in time, one series of measures at one time point. In light of all of the possible factors that could be influencing these measurements, as well as the small sample size of this study, the lack of significant findings with this final hypothesis must be considered in context and are addressed in depth in the next section.

Limitations

A limitation related to the parental well-being component of this study was the absence of information on family resources, and intactness of families. Maternal mental health is poorer in single mothers and those who are experiencing greater child-related stress. This is most true in raising children with intellectual disabilities, which, as already discussed, have a high comorbidity with 22q11.2DS (McConkey, Truesdale-Kennedy, Chang, Jarrah, & Shukri, 2008). Another weakness was the exclusion of comorbid diagnoses in 22q11.2DS children; disorders such as attention deficit hyperactivity disorder and anxiety disorders were not controlled for, and as Shashi and colleagues point out in their 2012 study, these could be impacting measures of EI. Extrapolating on that point, comorbid diagnoses could also impact FSIQ scores. Lastly, in terms of parent data, neither parental IQ nor socioeconomic status measurements were collected. As these relate to child IQ, they should be addressed in future research.

Parent-child interactions were not measured directly. This study sought to capture bi-directional effects but did not look directly at the actual relationships between parents and children, relying instead on inferences. The bi-directional component brings with it a whole other set of considerations, including what is environmental and what is genetic. Thus, future studies might consider adding in measures of parent-child interaction quality.

Another oversight was not controlling for the varying ethnicities and cultures of the families who participated in this study. There is a wide body of literature that looks at how Caucasian parents cope with disabilities as compared to, for example, their Asian counterparts. In a study by Lai, Goh, Oei, & Sung (2015), research suggests that Caucasian parents use more
emotion-focused coping than Asian parents, who use more problem-focused coping strategies. This information could have potential impact on parent reports of stress, anxiety, and depression.

For those parents who did report higher levels of stress, anxiety, and depression, the sources of these were unclear. Within the autism spectrum population, for example, it has been widely documented that caregivers report more mental distress than caregivers of other populations (including TD populations) but as Faithhorne and colleagues point out in their 2015 study, it has not been clear whether that distress is the result of the caregiver role, or whether there were preexisting sources of stress and/or mental disorders.

In examining the parent data, this study also failed to look at sex differences in reports of stress, anxiety, and depression. This was due largely to the small sample size but was also guided by previous research that found no significant sex differences in parent stress (Briegel et al., 2008). However, in their 2018 study of parents of children with neurodevelopmental disorders, Durukan, Kara, Almabaideen, Karaman, & Gül, found higher scores in mothers of depression and anxiety than in fathers. Along those same lines, considering sex of children in the analyses may also yield important insights. In Briegel, Schneider and Schwab’s 2007 study of infants, they examined how sex differences in children may differentially influence parental stress and found that girls induced more stress in parents than boys did.

Implications

This thesis and the surrounding literature raise a number of implications for the 22q11.2DS population. Chief among them is the need for early detection of 22q11.2DS, considering the impact not having a diagnosis may have as a possible contributor to stress, anxiety, and depression in parents. A need for psycho-educational groups for parents with children with 22q11.2DS has been identified in research in Ireland conducted by Alugo and colleagues (2017), and part of the barrier to treatment may be stigma, as their research also suggests from a series of focus groups with parents of 22q11.2DS.

Why the need to focus on parents? Stress is an intrinsic part of the diagnosis of 22q11.2DS, both for parents and children (Herscher & Brunner, 2008) and its presence may modify the parent-child relationship and differentially affect both parents and children. The parent-child relationship is crucial in the development of EI, and EI is crucial in terms of adaptive functioning.

In future research with parents of children with 22q11.2DS, it may make sense to measure stress, anxiety, and depression at various time-points, such as during times of stress (birth of child, medical emergencies, etc.) so as to better understand parent stress-responses. In looking closer at the EI component of this study, it would also make sense to measure parent EI to be able to consider and/or control for the possible role of heritability.

Lastly, children with 22q11.2DS are already at higher risk for developing psychotic disorders such as schizophrenia, so understanding any possible contributors to their development, in keeping with the diathesis-stress model of mental illness is crucial (Beaton & Simon, 2011). If parents are stressed, anxious, or depressed, and if this is affecting the parent-child relationship, there is the potential to deleteriously affect children’s EI and adaptive functioning. Likewise, if a child’s EI and adaptive functioning are compromised by 22q11.2DS, this affects parents, which can affect their interactions with their children. Consequently, detection and treatment of stress, anxiety, and depression in parents can have an influence on the development of the 22q11.2DS child.

The parents of both populations did not differ in terms of reported stress, anxiety, and depression. The EI in both populations of children did not have any measurable relationship to
parental stress, anxiety, or depression in parents. The parents in this sample may be particularly resilient, or perhaps the experience of having a child with 22q11.2DS has made them more so. The literature on the experience of parenting a child with 22q11.2DS underscores the importance of actively maintaining an awareness of stress, anxiety, and depression, considering the potential ramifications these may have on parental well-being and subsequently, parenting. Additionally, working with children to foster EI is a recommendation which may have a long-term impact on the developmental process. The children included in this study have many more developmental challenges ahead of them, and this study assessed one brief period which may not be reflective of the previous or future trajectories through the developmental stages.
References


VITA

The author was born in Chicago, Illinois. She obtained her Bachelor of Science degree in psychology from Tulane University in 2003 and her Master of Arts in Community Counseling from John Carroll University in Cleveland, Ohio in 2009. She joined the University of New Orleans psychology graduate program to pursue a PhD in applied developmental psychology and became a member of Professor Elliott Beaton’s research lab in 2016.