PARENTAL PERCEPTIONS OF THE CAUSE OF THEIR CHILD'S AUTISM SPECTRUM DISORDER: INFLUENCE ON TREATMENT

A Dissertation Presented to the
Faculty of the College of Education
University of Houston

In Partial Fulfillment
of the Requirements for the Degree

Doctor of Philosophy

by

Christie M. Brewton

April 2017

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Abstract

Background: Parents are largely responsible for selecting treatments for their children with autism spectrum disorder (ASD) and may choose treatments that lack research support. Recent research has focused on how parental perceptions about the cause of their child's ASD may influence subsequent treatment choices, although further investigation is needed. Additionally, some research has demonstrated an association between (a) age and (b) onset type (i.e., presence of developmental regression) of an individual with ASD, and treatments pursued by parents. **Purpose:** The current study used a large sample of parents of children with ASD (n = 326) to examine whether parental perceptions of the *cause* of their child's ASD influences frequency of current treatment choices overall and within created categories, with child age and onset type examined as potential moderators. **Methods:** A principal components analysis (PCA) was run on the Cause subscale of the IPQ-RA. A focus group was conducted to methodically determine how to group together numerous ASD treatments used in the current study. Poisson Regressions were run to determine relationships between causal factors and treatment selection. **Results:** Results from regression analyses revealed that several parental beliefs about cause of ASD (e.g., environmental risk factors, metaphysical factors) predicted an increase (or decrease) in the frequency of current parental treatment choices overall and within the evidence-based categories. Onset type was found to moderate these relationships in several instances, while child age had a lesser impact. **Conclusion:** Professionals who work with children with ASD and their families should collaborate and strive to understand the factors that drive parental treatment selection. Through understanding, professionals can approach the task of treatment planning with parents in

a more informed manner and promote treatments that will have a positive and meaningful impact in the functioning of children with ASD.

Keywords: Autism Spectrum Disorder, parental perceptions, etiology, IPQ-RA, treatment, onset type

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Chapter I

Introduction

Although there has been a rise in diagnostic prevalence and a surge in research conducted over the past several decades, the cause(s) of Autism Spectrum Disorder (ASD) remains unknown (Fombonne, 1999; Hill, Zuckerman, & Fombonne, 2014). ASD is a multifaceted diagnosis that widely impacts the individual and family across the lifespan and in multiple settings (e.g., school, home) (Cidav, Marcus, & Mandell, 2012; Leyfer et al., 2006; Seltzer et al., 2003). Currently, the lack of understanding regarding cause and the complex symptomatology inherent in ASD is further complicated by a plethora of treatment options, which parents are largely responsible for navigating when developing a treatment plan for their children (Green et al., 2006). Various factors impact parental treatment decisions (e.g., recommendations from professionals, parental desire to have an impact on symptoms, media), which often result in parents selecting and cycling through multiple treatments simultaneously based on decisions that are often not guided by current evidence-based practices. (Goin-Kochel, Mackintosh, & Myers, 2009; Levy & Hyman, 2005; Shyu, Tsai, Tsai, 2010). Although identifying the actual cause(s) of ASD remains elusive, emerging research investigating parental perceptions of cause of their child's ASD provides an opportunity to gain a better understanding of *why* parents are making certain treatment choices. In addition to providing insight into parental treatment decisions, understanding parental perceptions of cause of their child's disorder may elucidate factors that affect whether a parent will continue with treatment recommendations made by professionals (Hebert & Koulouglioti, 2010), impact public health decisions (e.g., vaccinations; Yudell, Tabor, Dawson, Rossi, & Newschaffer,

2013), and help clinicians engage parents in more meaningful conversations about treatment (Harrington, Rosen, Garnecho, & Patrick, 2006). The current study aimed to investigate parental perception of cause of their child's ASD and how these perceptions may impact treatment choices. The literature has indicated that child age may play a role in parental treatment selection, as younger children oftentimes receive a greater number and different types of treatments compared to older children (Green et al., 2006; Goin-Kochel, Myers, & Mackintosh, 2007). Furthermore, type of symptom onset (i.e., involving a regression in skills, early onset) has been linked to parental perceptions of cause, which may impact treatment decisions (Goin-Kochel, Mire, & Dempsey, 2014; Shumway et al., 2011). Therefore, current age and type of symptom onset were examined to consider their role in the relationship between parental perceptions of cause and treatment choices. The following review of the literature will outline: (a) current diagnostic criteria for ASD; (b) prevalence of ASD; (c) causal hypotheses surrounding ASD; (d) impact of ASD on the individual and family; (e) available treatments for ASD; (f) parental perceptions about treatments and causes for ASD; (g) a theoretical model for understanding how illness is cognitively represented; and (h) measuring parental perceptions.

Chapter II

Review of Literature

The DSM-5 (American Psychiatric Association [APA], 2013) characterizes the diagnostic classification of ASD by deficits in social communication across contexts (e.g., issues with social-emotional reciprocity, understanding relationships) and restricted interests/repetitive behaviors ([RRBs]; e.g., insistence on sameness, preoccupation with unusual objects). These symptoms manifest in the early developmental period and cause impairment in various areas of functioning (APA, 2013). Per the DSM-5, a diagnosis of ASD includes a series of specifiers intended to provide additional descriptive information about an individual's clinical presentation (APA, 2013; Volkmar & McPartland, 2014). Specifiers include indication of the: (a) presence of a known etiological factor (i.e., genetic, medical, or environmental factor), (b) presence of an intellectual impairment, (c) presence of expressive and/or receptive language impairment, (d) presence of catatonia, and (e) severity specifiers of the aforementioned symptom domains (i.e., social communication/interaction and restricted interests/repetitive behaviors). The severity specifiers can range from Level 1 (i.e., "Requiring support) to Level 3 ("Requiring very substantial support) (APA, 2013; Volkmar & McPartland, 2014).

Creation of the DSM-5 criteria for ASD was meant to address limitations present within the DSM-IV-TR (APA, 2004; Volkmar & McPartland, 2014), which will be discussed briefly here, as much of the available research cited within the current study relied on the DSM-IV-TR classification scheme. The DSM-IV-TR used the umbrella term Pervasive Developmental Disorders (PDDs), which identified distinct diagnostic categories (i.e., subtypes), including Autistic Disorder (AD), Asperger's Syndrome (AS),

Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS), Childhood Disintegrative Disorder (CDD), and Rett's Disorder (RD). In the DSM-IV-TR, AD, AS, and PDD-NOS diagnoses were referred to collectively as ASDs, while CDD and RD were indicated as more rare diagnoses under the PDD umbrella (APA, 2004). Also, the three core-symptom dimensions in the DSM-IV-TR (i.e., social deficits, communication deficits, and RRBs) were collapsed into two dimensions in the DSM-5 (i.e., social communication deficits and RRBs). This change was made largely because: (a) research demonstrated that differences between the communication and social criteria were subjective (Lord & Bishop, 2014) and (b) the DSM-5 aimed to utilize a dimensional approach (as opposed to a categorical approach used within the DSM-IV-TR), which allows for variability within symptom dimensions to be captured (Grzadzinski, Huerta, & Lord, 2013).

Rather than using the aforementioned distinct diagnostic categories, the DSM-5 captures all individuals under a single diagnosis (i.e., ASD). This modification was made for numerous reasons. One reason was to align the diagnostic criteria to reflect the current understanding of ASD as a purely behavioral diagnosis, which meant removing those diagnostic categories with a clear genetic cause (i.e., RD) (Lord & Bishop, 2014). However, under the DSM-5, those individuals who have a known genetic diagnosis (e.g., Fragile X Syndrome) and who also meet diagnostic criteria for ASD, may still receive an ASD diagnosis (representing behavioral symptoms), with the genetic diagnosis recorded as an associated feature of ASD. One advantage to this method is it allows for the recording of additional information concerning genetic and biological findings, which may aid in illuminating causal factors (Lord & Jones, 2013). Another reason for

modifying DSM-IV-TR diagnostic categories was that categorization *within* subtypes was not reliable across clinicians or time (Lord et al., 2012; Lord & Bishop, 2014). Other changes made to the DSM-IV-TR criteria during the creation of the DSM-5 included slight changes within the symptom dimensions (e.g., used broader principles to describe domain criteria allowing for more flexible interpretation), the inclusion of neurobiological specifiers (e.g., risk factors, biological conditions), and the addition of severity indicators (Lord & Bishop, 2014). Throughout the remainder of this document, the terms "ASD" and "autism" will be used interchangeably.

Prevalence of ASD

The diagnostic prevalence rate (i.e., proportion of cases in a population) of ASD over the past several decades has consistently increased (Hill et al., 2014). Currently, the Centers for Disease Control and Prevention (CDC) estimated that ASD affects 1 in 68 individuals (CDC, 2014; 2016). These rates include an approximate 3:1 to 4:1 male to female ratio (Fombonne, 1999; Werling & Geschwind, 2013). While there is not a definitive explanation regarding the increase of diagnostic rate, possibilities include: (a) the broadening of diagnostic criteria over time (Fombonne, 2003; Rutter, 2005; Wazana, Bresnahan, & Kline, 2007; Wing & Potter, 2002); (b) methodological differences across studies (Fombonne, 2003; Wazana et al., 2007; Wing & Potter, 2002); (c) increased awareness of symptoms among the professional community and parents (Wing & Potter, 2002; Rutter, 2005); (d) diagnostic substitution (i.e., shifting referral and diagnostic practices are resulting in identifying an individual with ASD who may have received a different diagnosis in the past; Shattuck, 2006); (e) the development and increased use of ASD-specific screening/diagnostic tools and services (Fombonne, 2003; Wing & Potter,

2002); and (f) a possible true rise in the incidence rate (i.e., number of new cases in a population) of the disorder (Fombonne, 2003; Rutter, 2005).

The prevalence rate of autism has continually increased over the past several decades, which has resulted in a surge of ASD research. One area of ASD research that requires more attention is understanding causal factors, which remains largely unknown. Understanding causality, particularly parental perceptions of these factors, is important as these perceptions may influence treatment choices made by parents. The next section will outline hypothesized causes of ASD within the literature.

Hypotheses about Causes of ASD

As aforementioned, there remains a lack of understanding concerning casual factors of ASD. In general, researchers believe that there is no single cause, but that development of the disorder is based on the interplay of genetic, epigenetic (i.e., heritable changes in gene expression that does not involve changes to the DNA sequence), and environmental factors (Chaste & Leboyer, 2012; Dawson, 2013; Perseco & Bourgeron, 2006). Since the first description of autism by Kanner (1943), there have been many hypotheses regarding potential causes of the disorder. Historically, what is now recognized as ASD was previously referred to as infantile psychosis and/or early infantile autism (EIA), and these diagnoses were thought to be a precursor to the development of childhood schizophrenia (Kanner, 1943). This section will start by reviewing some of the earlier causal hypotheses (e.g., related to parent characteristics and relationships) and progress through the more current hypotheses (e.g., related to perinatal/prenatal factors, genetic factors).

Parent-related characteristics and relationships. One of the first hypotheses regarding the cause of autism was related to parent personality characteristics (e.g., obsessiveness, intellectual, asocial) and/or patterns of parent-child relations (e.g., "cold-hearted" mothers, "mechanical" fathers) (Kanner, 1943, 1949). In fact, in an early paper, Kanner (1943) described a group of children suspected of having autism and stated that within the group "there [were] very few warmhearted mothers and fathers" and he characterized the marriages as "cold and formal affairs" (pp. 250). In a later paper, Kanner went on to describe the mothers of these children as lacking in outward displays of affection, while the fathers were described as displaying "unemotional objectivity" towards their children (Kanner, 1949).

In opposition to Kanner's perspective that autism develops as a result of emotionally deprived parent-child interactions, other researchers posited that symptoms of autism were maintained by over-responsive mothers who were acting in an attempt to alleviate their own feelings of anxiety and guilt over their child's condition (Green & Schecter, 1957). Additionally, some theorists posited that the development of verbal, social, and behavioral deficits of a child with autism might stem from intermittent reinforcement from the parents resulting in extinction of adaptive behaviors from the child (Ferster, 1961). More specifically, researchers ventured that various conditions may affect the parental response to a child (e.g., parental mental illness, parental engagement in other activities besides childcare [e.g., cleaning house, answering the phone]), which contributed to faulty reinforcement and conditioning of child behaviors (Ferster, 1961). Furthermore, Harper and Williams (1974) hypothesized that a child experiencing sensory deprivation (e.g., hearing loss, visual impairment) combined with environmental deficits

(e.g., traumatic events, having a parent with a mental illness) creates a level of stress that produces symptoms associated with autism. These early causal hypotheses are not currently thought to contribute to the development of ASD.

Organic brain damage. Another early theory was that autism was largely the result of brain damage (Boucher, 1977; Colby & Parkinson, 1977; Hier, LeMay, & Rosenberger, 1979; Rutter, 1967). Several early studies attempted to link autism with left-handedness, which was once thought to reflect brain damage to the left hemisphere that manifested in language impairment (Boucher, 1977; Colby & Parkinson, 1977; Hier et al., 1979); however, discrepant results were found between these early studies and no conclusive evidence was uncovered. Also, researchers hypothesized that the poor performance of individuals with autism on intelligence subtests with higher verbal loadings (compared to performance subtests) (Rutter, 1968) was suggestive of a left hemisphere abnormality (McCann, 1982). Rutter (1968) argued that there were no instances of brain abnormalities found in over half of the subjects studied in the literature (up to 1968), only that some brain dysfunctions (e.g., encephalitis) had been found in infants who later developed autism, making it difficult to attribute causality solely to the existence of brain abnormalities.

Language and perceptual abnormalities. Another early hypothesis was that language impairment was the primary reason for the development of autism. Rutter (1968) noted that research highlighted the difficulties inherent in children with ASD in responding to sounds (e.g., no startle response, difficult to distract with verbal cues). Furthermore, it was suggested that certain speech abnormalities characteristic of individuals with autism (e.g., echolalia, reversal of pronouns) was the result of a failure to

comprehend speech (Rutter, 1968). This failure in comprehension was posited to result in social withdrawal in young children with ASD, thereby leading to social interaction deficits (Rutter, 1968).

While the aforementioned hypotheses have largely either evolved or disappeared from the current literature, researchers are still uncertain about what causes ASD in the vast majority of cases. Since these earlier hypotheses, the body of research investigating potential causes of ASD has grown substantially and numerous other hypotheses have surfaced.

Differences in brain structure. Since the initial emergence of causal hypotheses related to organic brain damage, technological advances (e.g., positron emission tomography, magnetic resonance imaging) have allowed researchers to launch more in-depth investigations into brain differences in individuals with ASD compared to other populations. This area of research has resulted in several theories regarding potential causal factors. Numerous studies have found increased cerebral volume and size in children with ASD compared to typically developing individuals (Piven et al., 1995; Sparks et al., 2002). Results from one study found an increase in total brain volume for children with ASD (i.e., ages 8-12 years) compared to a control sample, which decreased in late adolescence and adulthood; however, head circumference was found to be increased in the adolescents and adults, which suggests these individuals had an increased brain volume as children compared to a typically developing sample (Aylward, Minshaw, Field, Sparks, & Singh, 2002). Researchers theorize that the abnormal patterns of brain growth (i.e., resulting in an increased brain volume) during early childhood and adolescence may contribute to interference in the development of several abilities (e.g.,

language, social skills, play skills, frontal lobe functioning), thus resulting in ASD (Aylward et al., 2002; Courchesne et al., 2001).

A recent meta-analysis of 16 studies involving voxel-wise whole-brain analyses on individuals with ASD compared to a typically developing sample reported the following results: (a) abnormalities in the lateral occipital lobe (V5 region), which may result in differences in motor-processing abilities; (b) abnormalities in the postcentral somatosensory cortex (area 3b) and a subsection of the human motor cortex (area 4p) that may affect the sensorimotor functioning; (c) abnormalities in the Basal Ganglia region, which may lead to motor difficulties, repetitive or stereotyped behaviors, and difficulties interpreting emotional body language; and (d) anomalies associated with the Medial Temporal Lobe, which has been linked with impaired facial recognition and processing of emotional facial expressions (Nickl-Jockschat et al., 2012). The authors noted that longitudinal studies incorporating better-defined samples were important future directions for this body of research, which ultimately aims to discover causal treatments (Nickl-Jockschat et al., 2012).

Exposure to environmental toxins. Another widely discussed potential causal factor is exposure to toxins, particularly in connection to vaccinations (Flaherty, 2011; Plotkin, Gerber, & Offit, 2009; Landrigan, 2010). Proponents of the "vaccination hypothesis" argue that the measles-mumps-rubella (MMR) vaccination causes intestinal damage that leads to the release of proteins in the bloodstream, which subsequently affects neural development. This now-discredited theory was first introduced by Andrew Wakefield and has significantly influenced parental perceptions about the cause of ASD (Plotkin et al., 2009). Wakefield's original 1998 publication on the topic has since been

retracted; furthermore, numerous studies have been conducted that demonstrate *no link* between autism and the MMR vaccination, although many parents continue to endorse an ASD-vaccine connection (Dales, Hammer, & Smith, 2001; Fombonne & Chakrabarti, 2001; Jain et al., 2015; Kaye, del Mar Molero-Montes & Jick, 2001; Richler et al., 2006; Taylor et al., 1999).

A second causal hypothesis related to vaccinations suggests that the use of Thimerosal (i.e., a compound that contains mercury), which is used in some vaccines, is neurotoxic and may cause inflammation of the brain (Plotkin et al., 2009; Ratajczak, 2011). In 1997, the Food and Drug Administration Modernization Act (FDAMA) was released, which intended to serve as a widespread regulation of food, medical products, and cosmetics in the United States; as part of this Act, the Food and Drug Administration (FDA) required that amounts of mercury be identified in all foods and drugs, including vaccinations that contained Thimerosal (FDAMA, 1997). Based on vaccine manufacturer reports, it was determined that some infants may have experienced levels of mercury exposure that exceeded recommended federal guidelines; despite a lack of evidence about the consequences of potential mercury exposure at the reported levels, several governmental bodies reacted (CDC, 1999). Thus, in 1999, the American Academy of Pediatrics and the Public Health Service recommended the removal of Thimerosal from all vaccines (CDC, 1999), which took place in 2001 in the United States. While removal of Thimerosal from vaccinations was mandated largely as a *precautionary measure*, this directive—coupled with the rising concern about the MMR vaccination—provoked concern among members of the public, leading to the emergence of several anti-mercury groups (Plotkin et al., 2009). Since the removal of Thimerosal, numerous studies have

been conducted to investigate the potential relationship between Thimerosal exposure and the development of ASD and *no supporting evidence* has been found (Heron & Golding, 2004; Stehr-Green, Tull, Stellfeld, Mortenson, & Simpson, 2003; Madsen et al., 2003; Taylor, Swerdfeger, & Eslick, 2014).

Other studies have investigated potential relationships between prenatal and perinatal exposure to heavy metals (e.g., arsenic, lead, aluminum, mercury, manganese) and autism risk and severity. These studies were largely conducted on the premise that developing children are less able to rid their bodies of heavy metals and that exposure to these chemicals may damage the developing brain, although these claims have not been substantiated in the literature (Adams et al., 2009; Landrigan, 2010). This body of research also includes investigation of hazardous air pollutants (HAPs), which were defined by the Environmental Protection Agency (EPA) as chemicals that may be associated with various adverse health problems (e.g., cancer, neurologic problems) (EPA, 1994). Some studies have found associations between heavy metal exposure (DeSoto & Hitlan, 2010; Palmer, Blanchard, Stein, Mandell, & Miller, 2006; Priya & Geetha, 2011), HAPs (Windham, Zhang, Gunier, Croen, & Grether, 2006) and an increased risk for ASD diagnosis; however, other studies have produced contradictory findings with mixed or no association between metals, HAPs and ASD (Abdullah et al., 2012; Kalkbrenner et al., 2010). A recent review on heavy metal pollution and ASD conducted by Gorini, Muratori, and Morales (2014) indicated that the mixed findings among these studies may be related to: (a) varying sample sizes, (b) assessing exposure at different time periods, and (c) comparison of metal analyses from different samples. These authors suggested that more research needs to be done in this area, particularly on

the effects of mixed-metal exposures and the development of advanced assessments for environmental neurotoxicity in individuals with ASD, in order to determine continued investigation of this hypothesis.

Prenatal factors. The contribution of prenatal factors to the increased risk of ASD has been studied extensively over the past several decades. Gardener, Spiegelman, and Buka (2009) conducted the first comprehensive meta-analysis that included 40 articles (published up to March, 2007) investigating the association between prenatal factors and an increased risk for ASD. All studies encompassed in the review included a comparison group and multimodal data (e.g., parent report, medical record review). Only risk factors that had been examined in two or more studies were included in the review. Across studies, the authors found a significant increased risk for ASD when considering several factors, including maternal gestational diabetes, maternal bleeding during pregnancy, maternal medication use during pregnancy, and the mother being born in another country (Gardener et al., 2009). However, the authors noted that few of these very early factors have been investigated across multiple, well-designed studies and the findings are largely inconsistent, which is likely the result of wide variability in study design characteristics (Gardener et al., 2009).

Studies released after this meta-analysis have further identified high pregnancy weight (i.e., 198.4 pounds or more) substantial weight gain during pregnancy (i.e., 39.7 pounds or more) (Dodds et al., 2011); viral infection during the 1st trimester; bacterial infection during the 2nd trimester (Atladottir et al., 2010); maternal fever without the use of fever-reducing medications (Zerbo et al., 2013); frequent exposure of the pregnant mother to second-hand smoke; chronic and acute medical conditions unrelated to

pregnancy (i.e., including thyroid gland related conditions, epilepsy, mental illness, diabetes, heart disease, hypertension, viral influenza, urticarial convulsions, serious anemia, and type-B hepatitis; Zhang et al., 2010); and maternal unhappy emotional state (i.e., feeling unhappy most of the time during pregnancy; Zhang et al., 2010), all which may be associated with an increased risk for ASD. However, it is unclear whether the increased risk of ASD associated with unhappy maternal state during pregnancy and chronic/acute medical conditions is related more so to ingestion of medications taken to alleviate these conditions. Additionally, maternal use of selective serotonin reuptake inhibitors (SSRIs) during the year before pregnancy and during the first trimester may increase the risk of ASD (Croen, Grether, Yoshida, Odouli & Hendrick, 2011; Rai et al., 2013); furthermore, maternal use of antiepileptic drugs (e.g., valproate) was found to be associated with increased risk for ASD (Christensen et al., 2013; Rasalam et al., 2005).

Research also has identified advanced maternal (i.e., ages 30-35 years and older) and paternal age (i.e., age 40 years and older) as risk factors for ASD (Croen, Najjar, Fireman, & Grether, 2007; Grether, Anderson, Croen, Smith, & Windham, 2009; Shelton, Tancredi, & Hertz-Picciotto, 2010). While over 40 studies have examined this association, results are inconsistent regarding whether advanced paternal age only, maternal age only, or advanced age in both parents contributes to a greater risk of ASD (Lee & McGrath, 2015). For instance, one large-scale study found that ASD risk was associated independently with advanced maternal age, advanced paternal age, and advanced paternal and maternal age (Parner et al., 2012). While more research is needed to determine the underlying mechanism(s) through which advanced parental age increases risk for ASD, current hypotheses suggest that increased occurrence of

spontaneous genomic alternations, increased cumulative exposure to environmental toxins, and increased incidences of chronic conditions (which may require medications). Furthermore, certain parenting techniques (e.g., seeking services sooner than younger parents) may play a role as some parents may be more sensitive to recognizing developmental differences in their children (Parner et al., 2012).

Perinatal and neonatal factors. In addition to prenatal factors, potential associations between perinatal and neonatal factors and increased risk for ASD have been widely studied. In a large-scale meta-analysis conducted by Gardener, Speigelman, and Buka (2011), factors associated with an increased ASD risk included abnormal birth presentation in general, breech presentation, complications with the umbilical cord (e.g., wrapped around neck at birth), fetal distress, birth injury/trauma, maternal hemorrhage, low birth weight, congenital malformations, low 5-minute Apgar score, meconium aspiration, feeding difficulties, neonatal anemia, complications with incompatibility of blood type, and hyperbilirubenemia (i.e., elevated serum bilirubin concentration). However, the authors of this review noted that very few of these factors have been investigated in multiple well-designed studies and those that have provided inconsistent and largely insignificant results (Gardener et al., 2011).

Studies conducted after Gardener et al., 2011 (i.e., review only included studies published through March 2007) found an increased risk for ASD when the child had a breech presentation (Bilder, Pinborough-Zimmerman, Miller, & McMahon, 2009); birth through cesarean section (Bilder, et al., 2009; Zhang et al., 2010); abnormal gestational age (i.e. preterm and/or postterm birth) (Zhang et al., 2010); delayed crying (Zhang et al., 2010); experienced apnea (i.e., pause in breathing for longer than 20 seconds after birth;

Zhang et al., 2010); and jaundice (Zhang et al., 2010). While many studies have examined the association between environmental risk factors (e.g., prenatal and perinatal) and the development of ASD, some researchers have posited that these risk factors may be associated with larger underlying risk factors (e.g., dysfunction in brain development; Tordjman et al., 2014).

Genetic factors. Genetic influences have been widely investigated and thought to be a primary contributing factor to the development of ASD. Twin studies have indicated that ASD has a monozygotic twin concordance rate of 73-95% compared to a dizygotic concordance rate of 1-10% (Caglayan, 2010; Folstein & Rosen-Sheidley, 2001; Persico & Bourgeron, 2006; Muhle, Trentacoste, & Rapin, 2004; Veenstra-Vanderweele & Cook, 2004). The higher rate of monozygotic concordance found in numerous studies lends support to the hypothesis that ASD is a highly heritable disorder (Muhle et al., 2004). Heritability is a concept that refers to the proportion of phenotypic variation (i.e., observable characteristics) that is attributable to variation in genetic factors (Lichtenstein, Carlstrom, Rastam, Gillberg, & Anckarsatar, 2010). Furthermore, recent research has indicated that approximately 19% of infants with at least one older sibling with ASD will later develop ASD; the same study showed an additional twofold increase in risk (32.2%) if an infant has more than one older sibling with ASD (Ozonoff et al., 2011). Additionally, first-degree relatives of affected individuals have demonstrated broader autism phenotype (BAP) characteristics (i.e., sub-clinical features associated with ASD in unaffected individuals) (Bernier, Gerdts, Munson, Dawson, & Estes, 2012; Hasegawa et al., 2014; Piven, Palmer, Jacobi, Childress, & Arndt, 1997), lending additional support to a potential genetic link.

Many individuals who have a known genetic syndrome are also diagnosed with what some researchers refer to as "secondary autism" (i.e., syndromic autism; identified with ASD when there is a known genetic cause). Known genetic syndromes that may lead to a diagnosis of secondary autism include Fragile X Syndrome, Rett Syndrome, Prader-Willi, Angelman Syndrome, Down Syndrome, Neurofibromatosis Type 1, Timothy Syndrome, Turner Syndrome, Tuberous Sclerosis, Klinefelter Syndrome, and Phenylketonuria (Caglayan, 2010; Folstein & Rosen-Sheidley, 2001; Geschwind, 2011).

For individuals with ASD without a known genetic syndrome (i.e., non-syndromic) several chromosomal structural variations (i.e., copy number variations [CNV] including deletions and duplications) have been identified that may contribute to the development of the disorder (Caglayan, 2010; Marshall et al., 2008); in fact, chromosomal abnormalities are thought to be present in approximately 3-8% of cases (Xu, Zwaigenbaum, Szatmari, & Scherer, 2004). For instance, Marshall et al. (2008) investigated the presence of chromosomal abnormalities in a well-categorized sample of simplex (i.e., one individual with ASD) and multiplex (i.e., more than one individual with ASD) families (N = 427). Results revealed that approximately 7% of randomly selected individuals from the sample carried one *de novo* (i.e., gene abnormality present for the first time in a family) CNV. Additionally, the authors noted that an abnormality in the 16p11.2 chromosomal region was found in 1% of the study sample, which was not found in the control sample (Marshall et al., 2008). More recent research has identified over 230 CNV regions thought to be associated with ASD, with particular evidence supporting the association between ASD and rare *de novo* events at 7q11.23, 15q11.2-13.1, 16p11.2, and *Nuerexin 1* (Sanders et al., 2011). Over the past decade, numerous large-scale studies

(e.g., Autism Genetic Resource Exchange [AGRE], Autism Genome Project, Simons Simplex Collection [SSC], Simons Variation in Individuals Project [SVIP]) have been developing genomic data repositories that will provide greater access for researchers to study the genetic influences of ASD (Simons VIP Consortium, 2012).

Although various hypotheses exist regarding the potential cause(s) of ASD, there is no definitive cause, or set of causes, consistently attributable to the development of the disorder. However, regardless of what causes ASD, the disorder has a profound influence on the affected individual and his or her family, which is discussed in the next section.

Impact of ASD on the Individual and Family

ASD has a pervasive impact on the affected individual. Symptoms of the disorder are typically recognized within the first 24 months of a child's life and persist throughout development, although the nature of impairment may fluctuate over time (APA, 2013; Seltzer et al., 2003). Individuals with ASD experience issues with communicating and relating to others (e.g., limited social engagement, language deficits, limited affective displays; Volkmar, Chawarska, & Klin, 2005), which may lead to various problems, including lack of friendships and victimization by peers (Van Roekel, Sholte, & Didden, 2010). Additionally, individuals with ASD may experience a myriad of physical symptoms including: (a) sleep problems (Krakowiak, Goodlin-Jones, Hertz-Picciotto, Croen, & Hanson, 2008), (b) gastrointestinal problems (e.g., chronic diarrhea, constipation, acid reflux; Molloy & Manning-Courtney, 2003), (c) incontinence (e.g., bed wetting; Geier, Kern, & Geier, 2012), and (d) sensory processing issues (e.g., sound sensitivity, pain sensitivity; Geier et al., 2012).

Furthermore, individuals with autism are likely to be diagnosed with one or more co-morbid mental health conditions. These associated conditions may affect the level of ASD severity, which has implications for associated diagnostic specifiers and necessary care (Leyfer et al., 2006; Matson & Nebel-Schwalm, 2007). The most common co-occurring condition in ASD is intellectual disability (ID) (Baio, 2012), with an estimated 26% (Chakrabarti & Fombonne, 2001) to 50% (Yeargin-Allsopp et al., 2003) of affected individuals meeting the criteria for ID. Additionally, many individuals with ASD demonstrate problematic externalizing behaviors (e.g., hyperactivity, conduct problems, aggression), which may warrant associated behavioral diagnoses such as Attention Deficit/Hyperactivity Disorder (ADHD) or Oppositional Defiant Disorder (ODD) (Leyfer et al., 2006; Mahan & Matson, 2011; Simonoff et al., 2008). Furthermore, co-occurring internalizing symptoms (e.g., depression, somatization) may result in co-morbid mood disorders (e.g., Major Depressive Disorder [MDD]), as well as anxiety disorders (e.g., social anxiety, specific phobias, Obsessive Compulsive Disorder [OCD]) (Leyfer et al., 2006; Mahan & Matson, 2011; Simonoff et al., 2008). Although the symptom profile and presence of co-morbid conditions varies by individual and throughout the lifespan, symptoms have an enduring effect on individual functioning and quality of life (Kuhlthau et al., 2010).

The impact ASD has on an individual is evident across settings. In the school context, individuals with ASD may be identified under the Individuals with Disabilities Education Improvement Act (IDEIA) under the special education category of Autism (AU) or another category (e.g., Intellectual Disability [ID]), depending on which category most appropriately represents a child's need (IDEIA, 2004). Qualifying for a special

education category does not require a DSM diagnosis; however, in order to qualify for special education services, an individual must demonstrate an educational need (i.e., symptoms must adversely affect school performance) (IDEIA, 2004). Educational placement and services provided in the school will vary based on individual need and the unique symptom profile (e.g., cognitive ability, behavior problems, verbal ability), which will likely affect the level of support and placement decisions (e.g., time in general education, special education) a student receives (White, Scahill, Klin, Koenig, & Volkmar, 2007).

In addition to the impact of ASD on the diagnosed individual, which is evidenced across settings, having a child with ASD also impacts the family. Individuals with ASD often require additional medical and mental health services; thus, the family may have increased health care expenditures (Gurney, McPheeters, & Davis, 2006; Leslie & Martin, 2007). This increase in cost is especially concerning given that mothers of children with ASD are less likely to work, may work fewer hours per week, and may make less money than mothers of typically developing children (Cidav et al., 2012). Furthermore, research has indicated that parents of children with ASD experience higher levels of stress compared to parents of typically developing children and parents of children with other disabilities (Baker-Ericzen, Brookman-Frazee, & Stahmer, 2005; Estes et al., 2009; Karst & Vaughn Van Hecke, 2012). Higher levels of parental stress among parents of children with ASD is not surprising considering that persons with ASD may exhibit numerous behavior problems (Lecavelier, Leone, & Wiltz, 2006), issues with social and emotional functioning (Estes et al., 2009), and require increased caretaking

demands relative to typically developing children (e.g., time spent obtaining services, providing long-term care; Baker-Ericzen et al., 2005).

In summary, ASD has a pervasive impact on the affected individual and the family that is evident across various settings. These issues contribute to the complexity and difficulty of the selection of treatments, decisions that are made largely by parents. The following section will outline some of the various treatment options that currently exist for ASD.

Treatments for ASD

ASD varies in clinical presentation and symptomatology, which results in a fluctuation of service needs that continues to change throughout the lifespan of affected individuals. Through a large web-based survey of parents with children with ASD, Green et al. (2006) queried parents (n = 522) on 116 treatments to investigate which treatments parents reported to be currently using or had used in the past. Parents reported currently using an average of seven treatments and had tried an average of eight treatments overall (Green et al., 2006). Another study found that children were currently receiving 4-6 different treatments, trying 7-9 overall (Goin-Kochel et al., 2007). Still other researchers reported that infants at-risk for ASD were receiving a range of 0-7 treatments, while their older siblings with ASD were receiving a range of 3-12 different treatments (Regehr & Feldman, 2009). Results from a large-scale study with children with ASD (n = 2758) found that frequency of use of various treatments (e.g., school and private speech therapy, school and private occupational therapy, intensive behavioral treatments), as well as number of treatment types endorsed, was highest among the youngest age group (i.e., 6-year-olds) and decreased among the older cohorts (i.e., 11- and 16-year-olds);

however, use of psychotropic medications was highest among the older children (i.e., 16-year-olds) and decreased among the younger cohorts (Mire, Raff, Brewton, & Goin-Kochel, 2015), which was consistent with other studies (e.g., Jain, 2015). A study conducted by Mire, Gealy, Kubiszyn, Burridge, and Goin-Kochel (2015) highlighted the importance of parental perceptions (i.e., number of symptoms attributed to ASD, amount of perceived control over treatment, and whether the parent perceived their child's ASD to be a chronic illness) on treatment decisions; however, more research on the influence of parental perceptions on treatment decisions, particularly the influence of perceived *cause* of ASD, needs to be conducted to further illuminate the importance of perceptions. The aforementioned research highlights that many parents are using a variety of treatment options simultaneously, that current child age may have an impact on the treatment selection and implementation by parents, and that parental perceptions of ASD plays a role in the parental selection of various ASD treatments.

This remainder of this section will outline categories of available treatment options for ASD. It is important to note that there is variability in terms of targeted treatment domains (e.g., social functioning, adaptive functioning), and often treatments affect more than one domain. There also is wide variability in terms of strength of evidence to support the treatments. Moreover, the criteria through which interventions are deemed "effective" can vary, and the large number of studies available can make it difficult to synthesize and present information in a meaningful way. One way to report the most accurate representation of evidence-based data is to utilize large-scale systematic reviews (Mulrow, 1994).

The following sections will present various treatment options available for individuals with ASD. Because of the large number of treatment options available, the following sections are not exhaustive, but are arranged by category and include specific examples within designated categories that aim to provide an overview of the kinds of treatments available. It is also important to note that treatment strategies (e.g., behaviorally-based reinforcement techniques) may be used across different categories of treatments; furthermore, evidence supporting use of treatments will differ across systematic reviews, such that findings from one review may support use of a treatment within a particular age-range and for targeted domains (e.g., academic functioning, interpersonal skills), while another review may differ in these aspects.

Behavioral treatments. Behavioral treatments are largely grounded in the principles of applied behavior analysis (ABA) and are the most studied and well established form of treatment for ASD (Odom, Collet-Klingenberg, Rogers, & Hatton, 2010; Warren et al., 2011; Young, Corea, Kimani, & Mandell, 2010). ABA principles focus on how environmental events influence the behavior of an individual (Vismara & Rogers, 2010) and techniques include teaching new skills, promoting generalization of skills learned, and using principles of reinforcement to decrease problem behaviors (Warren et al., 2011). Oftentimes, structured behavior programs utilize ABA principles and involve a combination of behavioral strategies delivered across a variety of settings at high volumes (e.g., over 20 hours per week) for multiple years (Lovaas, 2002). Behaviorally based treatments typically target core symptoms of ASD and generally aim to decrease problem behaviors (e.g., aggression, repetitive behavior) and increase adaptive behaviors (e.g., social communication) (Lord & Jones, 2013).

First, several foundational behavioral strategies (e.g., reinforcement, differential reinforcement, extinction, task analysis and chaining; Lovaas, 1981, 2002) will be reviewed as many of the behaviorally based treatments utilize numerous strategies. Following this, a selection of common, behaviorally based treatment approaches, including discrete trial training (DTT) and functional communication training (FCT; Carr & Durand, 1985), will be reviewed, followed by functional behavior assessment (FBA), which is a behaviorally based systematic approach in which data are collected to identify events that predict and maintain behaviors (Glasberg, 2005). Finally, this section will review comprehensive treatment models (CTMs), which target core ASD symptoms by including several focused treatments over an intensive time period (i.e., numerous hours a week spanning months or years) (Odom et al., 2010).

Reinforcement is a behavioral strategy that includes presenting or withdrawing a stimulus following a behavior in order to increase the frequency of that behavior (Cooper, Heron, & Heward, 2007). Supported for use with individuals with ASD (studies reviewed research for individuals from birth – 21 years), the literature maintains that this strategy is effective in improving functioning in communication, academics, behavior, and play skills, among other areas (National Autism Center, 2009; Odom et al., 2010; Young et al., 2010). Differential reinforcement aims to replace interfering or problem behaviors through reinforcing incompatible or alternative behaviors (Cooper et al., 2007). This behavioral strategy is supported by research to use with children with ASD (ages 4-12 years) mainly for improving communication skills, behaviors (National Autism Center, 2009; Odom et al., 2010), academic functioning, interpersonal skills, motor skills, play

skills, self-regulation (National Autism Center, 2009), and in promoting self-management of behavior (Young et al., 2010).

Extinction is another behavioral strategy that involves withholding reinforcement after an undesirable behavior in order to gradually eliminate occurrence of the behavior (Cooper et al., 2007). This strategy is supported by research for use with individuals with ASD (ages birth – 21 years) and has demonstrated positive outcomes in the areas of communication, behavior (National Autism Center, 2009; Odom et al., 2010; Young et al., 2010), social development (Young et al., 2010), interpersonal skills, motor skills, play behaviors, and self-regulation of behaviors (National Autism Center, 2009). Task analysis and chaining identifies the individual steps of a skill (e.g., brushing teeth) and breaks them down into manageable steps that are linked together over time (Cooper et al., 2007). This strategy is considered useful for individuals (ages 3-21 years) and has demonstrated effectiveness in the areas of academics, communication, play behaviors (National Autism Center, 2009; Odom et al., 2010), interpersonal skills, motor skills, and self-regulation of behaviors in individuals with ASD (National Autism Center, 2009).

DTT is a systematic instructional method that uses one-on-one instruction to teach a variety of skills using small discrete steps (Lovaas, 1981, 2002). The literature supports using DTT for individuals with ASD (ages 2-9 years) for improving communication skills, behavior (National Autism Center, 2009; Odom et al., 2010), academic functioning, higher cognitive functions, interpersonal skills, motor skills, play behaviors and self-regulation (National Autism Center, 2009). FCT is a behaviorally based treatment that aims to replace inappropriate behaviors with appropriate behaviors that serve the same function (Carr & Durand, 1985; Mancil, Conroy, Nakao, & Alter, 2006).

This method has shown to be effective with individuals (ages birth – 21 years) in the areas of communication, decreasing problem behaviors (National Autism Center, 2009; Odom et al., 2010; Young et al., 2010), academics, play skills, and self-regulation (National Autism Center, 2009). FBA is an evaluation that aims to determine the underlying function of a behavior so that it can be modified (Glasberg, 2005). The literature endorses use of FBA's with individuals with ASD (ages birth – 21 years) in the areas of behavior, communication (National Autism Center, 2009; Odom et al., 2010; Young et al., 2010), play skills, self-regulation (National Autism Center, 2009), social development, and sensory and motor development (Young et al., 2010).

Comprehensive treatment models (CTM), which target the core deficits of ASD through a set of practices that are applied over an extended period of time (i.e., several hours a week over months or years) (National Research Council, 2001; Odom, Boyd, Hall, & Hume, 2010; Rogers & Vismara, 2008), may be behaviorally based. For instance, pivotal response training (PRT; Koegel & Koegel, 2006) incorporates naturalistic methods that integrate teaching strategies into naturally occurring activities (e.g., play, bath time) and has been shown to improve communication, behavior, play skills (National Autism Center, 2009; Odom et al., 2010), and social skills (Odom et al., 2010) in individuals with ASD (ages 3-9 years). Other naturalistic behavioral interventions include incidental teaching (Charlop-Christy, 2008), and milieu teaching (Kaiser, Hendrickson, & Alpert, 1991), which have shown to be effective in the areas of communication, social skills (National Autism Center, 2009; Odom et al., 2010; Young et al., 2010), learning readiness, and play behaviors (National Autism Center, 2009) in children with ASD (birth – 9 years).

Developmental treatments. Developmentally focused CTMs address core symptoms of ASD through teaching goals that are tailored to the child's developmental level and skills (Vismara & Rogers, 2010). Developmental models include the Denver Model and the Early Start Denver Model (ESDM; i.e., for toddlers; Dawson et al., 2010), which combines ABA, developmental, and relationship-based approaches (Warren et al., 2011). ESDM has demonstrated effectiveness in randomized control trials that increased IQ, language abilities, and adaptive behaviors in toddlers with ASD in one-to-one (i.e., one therapist per child; Dawson et al., 2010) and group settings (Vivanti et al., 2014). Also, the Developmental Individual-Difference, Relationship-Based model (DIR; i.e., Floortime) uses an understanding of a child's functional development, individual differences (i.e., in processing, sensory reactivity, and motor planning), and interactive patterns to positively affect relational functioning of children with ASD (Greenspan & Weider, 1999). Although this treatment approach has some support, the systematic review conducted by the National Autism Center (2009) labeled this treatment as "emerging" meaning that more high quality studies are needed to determine effectiveness with individuals with ASD.

Communication and speech treatments. Various other treatments for ASD focus on improving communication skills and speech of individuals with varying degrees of verbal or nonverbal abilities (Vismara & Rogers, 2010). The Picture Exchange Communication System (PECS) uses pictures to teach functional communication to individuals with limited language capacity; the intervention takes place in natural settings to promote the use of speech in the social environment (Bondy & Frost, 2001). Researchers have demonstrated that PECS is considered an effective treatment for

individuals with ASD (ages 3-12 years) in improving communication skills, promoting social development (Odom et al., 2010; Young et al., 2010), and positively impacting behavior (Odom et al., 2010), while other systematic reviews have indicated that PECS requires additional research to demonstrate effectiveness with this population (National Autism Center, 2009; Warren et al., 2011). Joint-attention treatments promote responding to nonverbal bids made by others (e.g., following eye gaze, pointing to objects) (Charman et al., 1997); these treatments have demonstrated effectiveness with individuals with ASD (ages birth – 5 years) and have facilitated improvements in the areas of communication, interpersonal skills, and social development (National Autism Center, 2009; Young et al., 2010). Speech therapy (ST) is one of the most common treatment types used by families across multiple settings (e.g., school and community) (Bitterman, Daley, Mirsa, Carson, & Markowitz, 2008; Green et al., 2006); speech therapists generally target the pragmatic, language, and social communication needs of an individual with ASD through utilizing a variety of strategies and treatments.

Also, facilitated communication devices have been implemented in recent decades. This intervention involves a facilitator assisting an individual (e.g., by guiding arm or hand) in communicating through spelling out words or touching symbols (e.g., for bathroom) on a letter pad (National Autism Center, 2009). The American Psychological Association (1994) issued a statement about the potential for abuse in situations involving facilitated communication devices (e.g., unfounded accusations of maltreatment, communication used inappropriately for therapy and treatment decisions). The statement concluded that the use of these devices is controversial and not is supported by scientific evidence (APA, 1994). Furthermore, the National Autism Center (2009) review

concluded that the use of facilitated communication devices with individuals with ASD is currently unsupported by research.

Sensory and motor-deficit treatments. Many individuals with ASD experience sensory and/or motor difficulties. Sensory Integration (SI) therapy focuses on processing sensory information for learning motor or academic skills (Baranek, 2002); effectiveness research on the use of SI with individuals with ASD is mixed, and more research is needed (Baranek, 2002; Case-Smith & Arbesman, 2008). Recent reviews also concluded that SI therapy does not currently have enough evidence to support positive outcomes in individuals with ASD (Lang et al., 2012; National Autism Center, 2009; Warren et al., 2011).

Occupational therapy (OT) aims to improve various areas of functioning in individuals with ASD, such as adaptive functioning, motor coordination, and communication. Occupational therapists often use interventions that are perceived to be particularly engaging with individuals with ASD and may individualize interventions to fit a person's unique set of needs (Watling & Dietz, 2007). Furthermore, therapists may use a variety of techniques related to sensory and motor functioning such an SI therapy and ABA to improve performance and functioning (Case-Smith & Arbesman, 2008). However, occupational therapy may not be considered a specific treatment, but rather a treatment modality.

Social skills treatments. One of the core deficits for individuals with ASD is difficulty with social communication and interaction. There are various interventions that focus on the development of social skills, and these training programs are often implemented across school and outpatient settings (Barry et al., 2003). The use of social

narratives (i.e., using social stories to describe social situations to facilitate understanding) and social skills training groups (i.e., group instruction on building social skills that includes components such as modeling and feedback) have demonstrated effectiveness with children and adolescents with autism in various areas of functioning. These include improving communication and social skills in individuals with ASD (ages 3 – 18 years) for social skills groups and with individuals (ages 6-14 years) for social narratives (National Autism Center, 2009; Odom et al., 2010; Young et al., 2010).

Other/miscellaneous treatments. There are a variety of other treatments being used with individuals with ASD that are difficult to categorize. These therapies often purport to affect a variety of abilities (e.g., behavioral, social, sensory-motor) in individuals with ASD. For instance, music therapy seeks to teach individual skills or goals through the use of music (e.g., interactive instrument playing and singing). Massage/touch therapy aims to reduce stereotypic behavior and increase on-task responding through deep tissue stimulation (Young et al., 2010),

Pharmaceutical treatments. Parents may also choose pharmaceutical interventions to treat children with ASD. Currently, the FDA has approved two atypical antipsychotic medications for use with individuals with ASD. Aripirozole (i.e., Abilify) was approved for use with children (ages 6-17 years) and risperidone (i.e., Risperdal) was approved for use with children to treat irritability in persons with ASD (Warren et al., 2011). Aripiprozole has strong empirical support in decreasing problem behaviors, such as emotional distress, aggression, self-injury, and repetitive behaviors; however, a large number of side effects are also attributed to the use of this drug, such as weight gain and drowsiness (Warren et al., 2011). Risperidone has a moderate amount of scientific

support in reducing various problem behaviors (e.g., hyperactivity, irritability, tantrums) but also may result in serious side effects, such as weight gain, drowsiness, and extrapyramidal symptoms (e.g., tremors) (Warren et al., 2011).

Children and adolescents with ASD are often prescribed other medications "off label" (i.e., for purposes other than those approved by the FDA) to treat symptoms despite insufficient evidence to support use with the population. Medications that are commonly prescribed "off-label" include first generation atypical antipsychotics (i.e., haloperidol), serotonin reuptake inhibitors (i.e., fluoxetine, citalopram), stimulants (i.e., methylphenidate), anti-hypertensive (i.e.,guanfacine) and anti-depressant (i.e, norepinephrine reuptake inhibitor; atomoxetine). While there is some support for use of these medications with individuals with ASD, there is currently insufficient evidence available; also, these medications have not been approved by the FDA for use with individuals with ASD to treat these particular symptom areas (Warren et al., 2011). Nevertheless, use of psychotropic medications is a popular treatment choice. For example, results from a national sample (n = 1605) found that 31.3% of parents reported currently using one or more psychotropic medication(s) to treat their child's ASD symptoms, and 41.7% of parents reported having used a psychotropic medication at some point in their child's treatment; older children were more likely than younger children to be currently taking a medication or to have used a medication in the past (Mire, Nowell, Kubiszyn, & Goin-Kochel, 2014).

Complementary and alternative medical treatments. Many of the aforementioned treatments aim to improve functioning in a variety of areas directly associated with ASD. However, numerous complementary and alternative medical

(CAM) therapies are available that purport to address the *cause* of symptoms (Levy & Hyman, 2005). The National Center for Complementary and Integrative Health (NCCIH; 2015) defines "complementary" as the use of a non-mainstream practice *in combination* with conventional medicine and "alternative" as use of a practice *in the place of* conventional medicine. The American Academy of Pediatrics (2001) defines CAM treatments as "strategies that have not met the standards of clinical effectiveness, either through randomized controlled clinical trials or through the consensus of the biomedical community". The number of available CAM treatments has increased over recent years and parents, who may search for treatment options through a variety of sources (e.g., Internet), are very likely to encounter numerous CAM treatments, many of which claim to be effective in eradicating symptoms (Wong & Smith, 2006).

Many CAM treatments propose to treat ASD by targeting the immune system (e.g., treatments include antiviral agents, intravenous immunoglobins, chelation), targeting the gastrointestinal system (e.g., digestive enzymes, gluten-free/casein-free diet), modulating the central neurotransmitters and neuropeptides (e.g., Vitamins, folic acid, nutritional supplements [DMG]), and providing non-biological interventions (e.g., Auditory Integration Training, massages, acupuncture, craniosacral manipulation, hippotherapy, dolphin therapy; Levy & Hyman, 2005; Young et al., 2010). While one systematic review indicated that the use of proteins/amino acids has marginal evidence (i.e., requires further research to determine effectiveness) for use with individuals with ASD in improving social interactions, other CAM treatments currently have insufficient or no evidence to support their use with this population (Huffman, Sutcliffe, Tanner, & Feldman, 2011). Regardless of research support, various studies demonstrated that 71%

of parents have elected to use CAM treatments in the past; furthermore, approximately 30 - 50% of parents reported current use of a CAM treatment (Christon, Mackintosh, & Myers, 2010; Levy, Mandell, Merhar, Ittenbach, & Pinto-Martin, 2003). However, many parents neglect to inform their child's treating physician that they are using a CAM treatment; one study found this figure to be as high as 62% (Wong & Smith, 2006).

As evidenced in this overview, there are various types of treatments available for individuals with ASD. The large number of treatments combined with differences in theoretical basis, functionality, targeted domains, and empirical evidence makes categorizing treatments a very challenging task. The treatment categories included in this review highlight the variations in the targeted domains across treatments, and treatments that target one domain (e.g., communication, repetitive behaviors) may also affect changes across other areas of functioning. However, it is not clear that parents choose treatments based on the targeted domains and may choose treatments based on a variety of factors, which is discussed in detail in the following sections. Furthermore, while some treatments have strong empirical support (e.g., behavioral treatments), others have little or no research support (e.g., CAM treatments). Again, whether or not a treatment has evidence to support its use may be less of a factor in treatment selection, as many parents endorse use of treatments that have little or no empirical support (Green et al., 2006). This suggests that parents may place more value on *other* factors when making decisions about which treatments to pursue. The following section will discuss various factors that may influence parental decision-making when selecting treatments for their children with ASD.

Factors Affecting Parental Treatment Choices

Parents of individuals with ASD are largely responsible for selecting treatments for their children; therefore, understanding *why* parents make certain choices regarding treatment is important. Parents are faced with a myriad of treatment options that vary based on numerous factors (e.g., accessibility, cost), which can make developing and maintaining a treatment plan overwhelming (Goin-Kochel et al., 2009). Additionally, literature regarding the effectiveness of numerous treatments is mixed, which means that parents and professionals must make decisions regarding treatment choices based on a combination of factors, which is described further below.

In examining factors that pertain to treatment selection, Green (2007) found that parents often consider other parents' experiences with different treatments and the Internet to obtain information about treatment. Other research has demonstrated that professional referrals impact parents' decisions about treatment (Deyro et al., 2016; Green, 2007); this is complicated by the fact that different types of professionals make different treatment suggestions. More specifically, research has shown that psychologists and behavior analysts were more likely to recommend empirically supported treatments, while medical professionals were more likely to recommend treatments with mixed or no support (Miller, Schreck, Mulick, & Butter, 2012). As mentioned previously, certain practical factors (e.g., accessibility, cost) may also play a large role in the selection of treatments. A qualitative study conducted by Mackintosh, Goin-Kochel, and Myers (2012) indicated that parents were concerned about waitlists, access to specialists, limitations (i.e., intensity), and accessibility within geographic location of many available treatments. Furthermore, regarding cost in relation to available treatment options, parents expressed concern about the monetary cost of treatments, lack of reimbursement by

insurance companies, and the time and effort necessary to promote success of some treatments (e.g., maintaining a gluten-free diet) (Mackintosh et al., 2012). Numerous other factors may also influence parental decision-making, including parental desire to find a treatment that may have a quick and significant impact (Metz, Mulick, & Butter, 2005), parenting style, access to services and treatment, media influence (Levy & Hyman, 2005; Shyu et al., 2010), effect of the selected treatment strategy, and fit of the child and/or parent with the therapist (Shyu et al., 2010).

Child-specific characteristics. Child-specific characteristics (e.g., current age, type of symptom onset) may also play a role in parental treatment decisions. In general, research has indicated that younger children receive a greater number of treatments than older children (Green et al., 2006), which may be related to the consideration that early intervention is linked to more favorable developmental outcomes (Koegel, Koegel, Ashbaugh, & Bradshaw, 2014). Goin-Kochel and colleagues (2007) found that younger children were receiving more diet, behavioral (e.g., ABA, floor time), educational, and alternative (e.g., chelation) treatments than older children; also, older children were more likely to receive pharmacological treatments compared to younger children—findings that have been corroborated in subsequent research (e.g., Mire et al., 2015). It was also observed that the likelihood of parents using biomedical treatments (e.g., special diets, chelation) decreased as a child's age increased (Mire et al., 2015).

A second child-specific characteristic that may influence parental treatment options is type of symptom onset, specifically, regressive and early onset. *Regressive onset* of ASD refers to an observable regression (i.e., loss) in mastered skills after a period of normal development; children with *early onset* do not experience a regression

in skills, but demonstrate symptoms of ASD within the first year of life (Goin-Kochel et al., 2014; Shumway et al., 2011). Recent research has identified four types of symptom onset within the regressive and early categories and demonstrate links between type of onset with parental perceptions of cause of ASD, which may in turn influence treatment decisions made by parents (Goin-Kochel et al., 2014). However, more research is needed in order to further investigate the importance that child-specific characteristics, such as current age and type of ASD onset, may have on parental perceptions of cause and treatment decisions.

Parental Perceptions and Treatment Choices

Perhaps one of the most important factors that may affect the selection of treatments is how parents perceive their child's autism—how they think about it, feel about it, and understand it. Researchers have stated that, "[t]he way in which people adapt to and seek treatment for an illness is influenced by how they perceive and explain that illness" (Shyu, et al. 2010, pp. 1323). Cognitive theory posits that human behavior may be explained by understanding thought processes, or mental representations (Thagard, 2010). One of the first psychologists to study cognitive functions (e.g., awareness, reaction, perceptions) was William Wundt, whose work formed the basis for cognitive research (Grider, 1993). Perceptions are cognitive processes that mediate a person's ability to cope with an illness threat (Leventhal, Nerenz, & Steele, 1984). Therefore, parental perceptions about their child's ASD are likely to affect behaviors (e.g., choosing treatments) (Dardennes et al., 2011; Mire et al., 2015).

Regarding *how* parental perceptions influence treatment decisions, Mandell and Novak (2005) posited that parents who believe that they can cure their child's autism may

choose treatments that claim to "cure" ASD; in fact, many CAM treatments have claimed to be effective in treating ASD, but there is no empirical evidence to substantiate such claims (e.g., vitamin supplements, secretin) (Metz et al., 2005). Also, parental perceptions regarding the efficacy of certain treatments may be a determining factor in whether they continue or discontinue a course of treatment (Goin-Kochel et al., 2009). A more recent study found that the likelihood of parents selecting psychotropic medications for their child decreased when parents attributed *more* symptoms to their child's ASD, which may suggest parents' believe medications are more useful in tackling secondary symptoms rather than core ASD symptoms (Mire et al., 2015). Within the same study, results indicated that parental perception of control over their child's treatment was associated with higher likelihood of private occupational therapy, other intensive treatments (e.g., Floortime), and the use of psychotropic medications; also, parents were less likely to pursue private speech therapy when they believed their child's ASD to be more chronic in nature (Mire et al., 2015).

Parental perceptions of cause and impact on treatment. When studying parental perceptions, perceptions about *causes* of their child's ASD may play a particularly important role in understanding parental treatment decisions. Understanding parental perceptions about cause is important, as these perceptions may impact treatment choices; public-health related decisions (e.g., whether or not to vaccinate a child; Yudell et al., 2013); may help professionals determine what psychoeducational resources or additional information to provide; and may offer insight into factors that contribute to parental stress (Dale, Jahoda, & Knott, 2006).

Although various hypotheses about potential causes of ASD exist, confirmed causal factors remain unknown; it is not surprising then that research depicting parental perceptions regarding etiological factors are mixed. A study by Mercer, Creighton, Holden, and Lewis (2006) found that 90.2% of parents (n = 41) believed that there were multiple causes that contributed to their child's ASD; furthermore, 90.2% endorsed that genetic causes contributed to their child's ASD (e.g., family history), 68.3% endorsed perinatal factors, 51.2% believed in diet-related causes, 43.9% endorsed prenatal factors (e.g., maternal vaccination, substance abuse, maternal illness, advanced maternal age), and 40% reported perceptions that vaccinations contributed to their child's autism.

A study conducted by Al-Anbar, Dardennes, Prado-Netto, Kaye, and Contejean (2010) investigated parental perceptions about causes and examined how these perceptions may influence treatment decisions. Results indicated that those endorsing external/environmental causes were more likely to report selection of metabolic treatments (e.g., special diets, vitamin supplements; Anbar et al., 2010). A follow-up study that investigated the same factors suggested that parents who perceived that early traumatic experiences played a causal role were less likely to use behavioral interventions and PECS; furthermore, parents who attributed cause to illness during pregnancy were more likely to choose medication treatments (Dardennes, et al., 2011).

Research has also attempted to look at the relationship between parental perceptions about cause and the type of symptom onset (Goin-Kochel et al., 2014). A recent study found that many parent's endorsed both internal causes (e.g., 75.8% endorsed genetics; 59.7% brain structure) and external causes (e.g., 46.3% endorsed the will of God, 41.8% vaccines, 37.3% environmental pollution). Furthermore, parents were

more likely to attribute the cause of their child's ASD to external/environmental causes if they had witnessed a regression in skills (e.g., language, social) in their child (Goin-Kochel et al., 2014). While some research investigating child characteristics (e.g., current age, type of symptom onset) and parental treatment decisions is available in the literature, this author has not found any research examining these child-specific factors as potentially influencing the relationship between parental perceptions of cause and treatment decisions.

Based on this review, it is evident that there are various factors that may affect treatment choices made by parents for their children with ASD; furthermore, parental perceptions about their child's ASD, particularly about cause, has also been found to impact treatment decisions. However, information about how parental perceptions of cause may potentially impact treatment decisions is lacking and more research needs to be conducted in this area. The next section will discuss a model that may be used to conceptualize parental perceptions of their child's ASD.

Leventhal's Model of Illness Representation

The concept of "illness" is culturally-dependent and shaped by how members of a culture perceive and manage an illness (Mandell & Novak, 2005). Some have described ASD as a lifelong condition, which may also be conceptualized as a chronic illness (Avdi, Griffin, & Brough, 2000). Leventhal and colleagues described the self-regulatory process that individuals engage in when representing an illness and coping with illness (Leventhal, Meyer, & Nerenz, 1980). An individual's "representation" of illness is composed of attributions, which are a person's attempts to understand, predict, and control a perceived threat (Leventhal, Leventhal, & Contrada, 1998; Taylor, Lictman, &

Wood, 1984; Wong & Weiner, 1981). In one of the most widely used models of illness representation—Leventhal's Model of Illness Representation-- the major attributions that are thought to contribute to an individual's ability to cope with an illness are *identity, cause, consequences,* and *duration* of the illness (Leventhal et al., 1984). This model was developed to help reduce stress and improve an individual's understanding, attitudes (i.e., perceptions), and behavior surrounding health practices (Leventhal et al., 1984).

Regarding the *identity* attribution of illness representation, a series of studies on patients with hypertension (Meyer, 1981) solidified the "commonsense notion" that individuals with illness symptoms will seek a label or a diagnosis (i.e., identity) for their symptoms (Leventhal et al., 1984). These concrete cognitive attributions (i.e., identifying illness through labels and symptoms) were found to influence treatment adherence and continuation of treatment (i.e., treatment behaviors) in patients with hypertension (Leventhal et al, 1984). More specifically, patients who believed that a treatment affected their *symptoms* (not necessarily the illness itself) were more likely to adhere to a treatment plan compared to those patients who did *not* believe that the treatment impacted symptoms (Meyer, Leventhal, & Gutmann, 1985). The same series of studies found that the *duration* attribute of illness representation also contributed to treatment behaviors; for example, patients who perceived their illness to be a chronic condition were more likely to adhere to treatment compared to those who perceived their illness as an acute condition (Leventhal et al, 1984; Meyer et al., 1985).

In addition to the *identity* and *duration* attributes of illness representation, attributes pertaining to both the *causes* and *consequences* of an illness are also thought to contribute to coping and appraisals in this model (e.g., responses to illness, resources;

Leventhal, Leventhal, & Cameron, 2001). The *cause* of the illness may be perceived in numerous ways, including attributions to an external cause (e.g., virus), internal (e.g., genetics), and behavioral (e.g., smoking). Perceptions about the *consequences* of an illness may include physical changes, emotional experiences, and economic impact. Furthermore, in the later conceptualizations of Leventhal's Model of Illness Representation, Leventhal and colleagues included an additional illness representation attribute (i.e., *controllability*), which considered the response of an illness to treatment (Leventhal et al., 2001).

These illness representations (i.e., *identity, duration, cause, consequences,* and *controllability*) have complex effects on emotions, which together may affect behaviors (e.g., treatment decisions and adherence). For example, having a chronic disease may lead to feelings of depression or despair as the illness representations are negative (e.g., long duration, severe consequences [death]), which may influence treatment adherence (Leventhal et al., 1984, 1998).

Leventhal's model presents a framework to contextualize how illness representations may impact health perceptions and behaviors. However, the literature examining how these representations associate with parental beliefs about their child with ASD, particularly regarding perceptions about cause, is sparse. The next section will provide a review on how Leventhal's model has evolved to provide the basis for the development of a measure about parents' perceptions of their child's ASD.

Measuring Parental Perceptions

Weinman, Petrie, Moss-Morris, and Horne (1996) developed the Illness Perception Questionnaire (IPQ), which was designed to measure an individual's

perceptions about their own illness. The IPQ is theoretically based on Leventhal's Model of Illness Representation (Leventhal et al., 1998) and measures the five components of *identity* (i.e., patients' ideas about the illness label), *cause* (i.e., ideas about the cause of illness), *duration* (i.e., perception of how long the illness will last and prognosis), *consequences* (i.e., perceptions of illness severity and impact on functioning), and *controllability* (i.e., perceptions about power over the illness; Weinman et al., 1996). The creators of the IPQ aimed to construct a measure that had flexibility so that it could be adapted to other illness populations; also, they created a separate version to use in gathering data about others' (e.g., significant others, family members) perceptions of an illness (Moss-Morris et al., 2002; Weinman et al., 1996).

The IPQ was later revised (i.e., became the IPQ-R) in order to improve the measurement properties and widen the scope by including an additional *emotional* component to the cognitive representation of illness (Moss-Morris, et al., 2002). The validation process of the IPQ-R included the collection of data from multiple illness groups (i.e., asthma, diabetes, rheumatoid arthritis, chronic pain, acute pain, myocardial infarction, multiple sclerosis, and HIV), which speaks to the aforementioned adaptability of the scale to various populations (Moss-Morris, et al., 2002). As part of the revision, the authors extended the number of items that measure perceptions about *cause* from 10 to 18 (Moss-Morris et al., 2002).

The IPQ-R was modified for use with parents of children with ASD (i.e., became the IPQ-RA; Al Anbar et al., 2010) in order to investigate potential relationships with parental perceptions and treatment choices, including an exploratory analysis of the relationship between parental perceptions of cause and treatment. The authors of the IPQ-

RA adapted the IPQ-R by: (a) replacing the term “illness” with “disorder”, (b) including a 14-item list of common ASD symptoms, and (c) modifying the wording of the items to make them appropriate for parents who would be completing the measure about their children (e.g., replaced “my illness” with “his (or her) disorder”; Al Anbar et al., 2010). A principle components analysis (PCA) was conducted on the 18 items that compose the Cause subscale, and results revealed a three-factor solution that represented parental perceptions about cause; these factors were labeled personal attributions (e.g., “alcohol intake”, “personal injury”), environment (e.g., “pollution”, “germs or virus”), and heredity attribution (i.e., contained two items of “heredity” or “chance or bad luck”; Al Anbar et al., 2010). However, limitations to this study included a small sample size (*n* =89), inclusion of non-confirmed cases of ASD, a short list of treatment options, and non ASD-specific perceptions about cause.

A more recent study using the IPQ-RA (Mire, et al., 2015) further modified the wording from “his/her disorder” to “your child’s ASD”, changed the causal belief item of “heredity” to “genetics”, and added three additional causes including: (1) “in utero stress or accident”, (2) “my child’s brain structure”, and (4) “stress at birth” (Mire, et al., 2015). These modifications were made in order to make the measure more applicable to parents of children with ASD and to provide a more representative range of causal perceptions that have been reviewed in the literature. Given that the number of items included in the IPQ-RA casual perceptions subscale increased and that there has been a preponderance of ASD research since the Al Anbar et al., (2010) study was published, there is currently a need for a re-analysis of the factor structure of the Cause subscale of the IPQ-RA, as well as further investigation of the relationship between parental perceptions of cause and

treatment.

Current Research Questions

There are numerous treatment options for children with ASD, and parents are largely responsible for choosing which treatments to pursue for their children. Despite the number of treatment options available for ASD, it is well documented that some treatments have more evidence in support of effective outcomes for persons with ASD (e.g., behavioral therapy) compared to other treatments (e.g., CAM treatments) (Simpson, 2005; Rogers & Vismara, 2008). It is important that parents are informed about the differences between various treatment options and, therefore, more capable of making educated decisions about treatment. Understanding the factors that may influence parental-treatment choices is important, as such decisions ultimately bear on the developmental outcomes of affected children, as well as the quality of family functioning, as a whole. Recent research suggests a potential link between parental perceptions of cause of ASD and treatment choices, although this relationship is not entirely understood. However, research does suggest that perceptions are malleable (Diefenbach & Leventhal, 1996); thus, understanding how parental perceptions (e.g., cause of ASD) influence treatment choices for their children may allow clinicians to lead a more engaging and informed conversation about treatment with parents, including which treatments (i.e., evidence-based) to pursue over others.

The primary goal of the current study was to examine whether parental perceptions of cause of their child's ASD predicts treatment choices. More specifically, the first research aim investigated the factor structure of the Cause subscale of the Illness Perception Questionnaire – Revised for Autism (IPQ-RA; Al Anbar et al., 2010).

Secondly, the current study used a formal consensus coding approach to investigate the different categories of treatments that parents of children with ASD are *currently* using, which aligns with current reports of parental perceptions of cause. A third research aim combined the results from the first two research aims to investigate reported parental perceptions of cause of their child's ASD and whether these perceptions predict frequency of current treatment use within several treatment categories that were conceptualized by researchers and clinicians who work with children and families with ASD. The third research question also investigated whether two child characteristics discussed within the literature (i.e., current age, type of symptom onset) moderated the relationship between parental perceptions of cause of their child's ASD and frequency of treatment use within created categories. Although there is some association between (a) current age of the child and treatment use, and (b) type of symptom onset and perceptions of cause, the author has not found any research suggesting that these factors will specifically moderate the relationship between parental perceptions of cause and treatment choice; therefore, the inclusion of these moderators served as an initial investigation into this potential relationship.

Understanding parental perceptions of cause is important because they may influence treatment choices a parent makes and may also affect whether a parent continues with treatment recommendations made by professionals (Hebert & Koulouglioti, 2010). Furthermore, a clinician who is knowledgeable about how parental perceptions of cause may impact treatment selections will be better equipped to engage a parent in a meaningful conversation about treatment; once engaged, a clinician would

have the opportunity to educate parents about evidence-based treatments and warn parents about potentially harmful treatment options (Harrington et al., 2006).

Chapter III

Method

Participants

The final number of participants (i.e., parents of children with ASD) for the current study was 326. Participants were drawn from a project at the University of Houston titled Parental Perceptions and Family Stress: Implications for Treatment-Seeking for Children with Autism Spectrum Disorder (PeP; Principle Investigator: Dr. Sarah Mire). Data collection for the PeP study started in November 2014 and was completed in July 2015. Overall, the PeP study collected data from 362 participants. During data review, it was discovered that in 28 instances data were collected on the same child with ASD from both the mother and the father. Research suggests that within families who have children with ASD, the mother typically is more heavily involved with care for the child or is often referred to as the primary caregiver (Benson, Karlof, & Siperstein, 2008; Dardas & Ahmad, 2014). Therefore, the 28 duplicate fathers were deleted from the participant pool for the current study. Furthermore, eight participants were deleted due to missing data (see Results section for further details).

All participants for the PeP study were drawn from families who had previously participated in a multi-site, national study called the Simons Simplex Collection (SSC). Data collection for the SSC was completed in March 2011, and the entire sample contained 2,737 simplex families. As part of the SSC, participants were given the option to consent to be re-contacted for future research studies. Approximately 1,325 families consented to be re-contacted for future studies. The SSC was a collaborative study funded by the Simons Foundation Autism Research Initiative (SFARI). The goal of the SSC was

to identify de novo genetic variants within simplex families (i.e., only one individual with ASD in the family) that contribute to development or risk of ASD. In addition to the genetic data, a wealth of clinical data was also collected in order to better characterize the sample.

For the SSC, data were collected from participants across 12 university-sites which included: (1) Baylor College of Medicine (Houston, Texas); (2) Children's Hospital Boston (Boston, MA); (3) Columbia University (New York, New York); (4) Emory University (Atlanta, Georgia); (5) McGill University (Montreal, Quebec), (6) University of California (Los Angeles, California); (7) University of Illinois (Chicago, Illinois); (8) University of Michigan (Ann Arbor, Michigan); (9) University of Missouri (Columbia, Missouri); (10) University of Washington (Seattle, Washington); (11) Vanderbilt University (Nashville, Tennessee); and (12) Yale University (New Haven, Connecticut).

The SSC had extensive inclusion and exclusion criteria. In order to be included in the SSC, individuals with ASD (i.e., also referred to as probands) had to be between the ages 4-17 years, 11 months and meet criteria for an ASD diagnosis per the DSM-IV-TR. Diagnoses were made by teams of psychologists and physicians through the research-reliable administrations of the *Autism Diagnostic Interview - Revised* (ADI-R; Le Couteur, Lord, & Rutter, 2003) and the *Autism Diagnostic Observation Schedule* (ADOS; Lord, Rutter, DiLavore, & Risi, 2002) in conjunction with clinical opinion, as well as medical- and developmental-history review. Also, probands who were between ages 4-6 years, 11 months had to demonstrate a nonverbal mental age of 24 months or greater; probands age 7 years and older must have demonstrated a nonverbal mental age of 30

months or more (i.e., per the *Mullen Scales of Early Learning* [Mullen, 1995], the *Differential Ability Scales-II* [Elliott, 2007], the *Wechsler Intelligence Scale for Children-IV* [Wechsler, 2003] or the *Wechsler Abbreviated Scale of Intelligence* [Wechsler, 1999]). Enrolled families were also required to have an unaffected sibling (i.e., did not qualify for an ASD diagnosis) of the proband and both biological parents available and willing to submit a blood sample for DNA.

Families were excluded from the SSC if the proband was born preterm *and* had a low birth weight (i.e., fewer than 36 weeks gestation *and* weighed less than 4 lbs 6.5 oz), extensive prenatal and/or perinatal complications (e.g., stayed in the neonatal intensive care unit after birth, experienced extensive oxygen deprivation), a genetic disorder (e.g., Fragile X Syndrome), was non-English speaking, or had other complicating factors that might hinder participation (e.g., extensive sensory or motor difficulties). Furthermore, families in which the immediate and/or extended family (i.e., up to third-degree relatives) were suspected of having ASD were excluded. For a more detailed description of the SSC database and the procedures used for data collection, see http://sfari.org and Fischbach and Lord (2010). Through a series of identifiers (i.e., numerical codes), the participant data for the PeP study was linked to the SSC data.

The demographic data for the PeP participants used in the current study may be seen in Table 1.

Table 1.

Demographic Characteristics of PeP Participants (percentages in parentheses)

Characteristic	PeP (n = 326)
Mothers	287 (88.0)
Race	

	White	278 (85.3)
	Black/African American	5 (1.5)
	Asian American	8 (2.5)
	More than One	22 (6.7)
	Other	12 (3.7)
	Not Specified	1 (.3)
Ethnicity[a]		
	Non-Hispanic	302 (92.6)
	Hispanic	23 (7.1)
Income[b]		
	Less than 20K	4 (1.2)
	21-35K	16 (4.9)
	36-50K	19 (5.8)
	51-65K	32 (9.8)
	66-80K	34 (10.4)
	81-100K	63 (19.3)
	101-130K	33 (10.1)
	131-160K	42 (12.9)
	More than 161K	65 (19.9)
Parental Education		
	Less than High School	1 (.3)
	High School Diploma	50 (15.3)
	Associate's Degree	35 (10.7)
	Bachelor's Degree	127 (39.0)
	Master's Degree	85 (26.1)
	Doctoral Degree	28 (8.6)

[a] Missing one value. Participants were given the choice to not answer.
[b] Missing 18 values. Participants were given the choice to not answer.

A majority of participants were mothers (88.0%), Caucasian (85.3%), and Non-Hispanic (92.6%). While the racial categories of Native American and Native Hawaiian were options within the survey, no participants selected these categories. A large proportion of families reported an income level higher than $81,000 (62.2%). Many of the participants reported their education level was Bachelor's Degree (39.0%) or Master's Degree (26.1%). The mean age of participant's was 46.06 years (*SD* = 5.81).

With the exception of child age, additional child-specific characteristics are not

utilized in the current study. However, these variables can help illustrate the functioning of each child with ASD across various domains. These additional child-specific characteristics are presented in Table 2.

Table 2.

Child-Specific Characteristics of the PeP Study

Characteristic	Mean (*SD*)	Range
Child Age in Years	13.56 (3.45)	7-23
Adaptive Behavior Composite	73.52 (11.94)	32-103
Full Scale IQ Scores	83.25 (28.23)	16-155
Nonverbal IQ Score	86.02 (26.46)	21-144
Verbal IQ Score	80.12 (31.59)	7-153

The average age in years of children with ASD was 13.56 (*SD* = 3.45). Furthermore, per the *Vineland Adaptive Behavior Scales, Second Edition (*VABS-2*)* given as part of the SSC*,* the average adaptive functioning of participants was in the moderately low range (*M* = 73.52; *SD* = 11.94). Cognitive scores were obtained as part of the SSC using the *Mullen Scales of Early Learning* (Mullen, 1995), the *Differential Ability Scales-II* (Elliott, 2007), the *Wechsler Intelligence Scale for Children-IV* (Wechsler, 2003), or the *Wechsler Abbreviated Scale of Intelligence* (Wechsler, 1999). Scores for Full Scale (*M* = 83.25; *SD* = 28.23), Verbal (*M* = 80.12; *SD* = 31.59), and Nonverbal (*M* = 86.02; *SD* = 26.46) IQ all fell in the Below Average range.

Measures

Data originated primarily from the PeP project (i.e., new data collection) and a small portion from the SSC database (i.e., extant data). Data from the SSC database are made available to researchers through an application process and unique identifiers allow a link between participant data from both studies. Data extracted from the SSC included a portion of the demographic information and data regarding type of symptom onset (i.e., moderating variable). The remainder of the data for the current study was extracted from the PeP study data.

Demographic Information. Demographic variables were drawn from both the SSC and PeP data. Within the SSC study, demographic data were collected using a Background History Form that was completed through a phone interview between a parent and a member of the research staff across the 12 data collection sites. Variables from the SSC that were used included information regarding race/ethnicity, which was captured using the following categories: (a) African American, (b) Asian American, (c) Caucasian, (d) Native American/Alaskan Native, (e) Native Hawaiian/ Other Pacific Islander, (f) More than One Race, (g) Other, and (h) Not Specified. For the current study, there were no participants who selected their race/ethnicity as Native American/Alaskan Native or Native Hawaiian/Other Pacific Islander. Furthermore, adaptive functioning and cognitive scores (i.e., full-scale IQ, verbal IQ, and nonverbal IQ) were collected as part of the SSC and reported on an interval level in the current study. These child-specific characteristics are not used in the main analyses of the study, but were provided to present a depiction of child functioning across several areas.

In addition to the demographic variables from the SSC database, certain demographic variables were drawn from the PeP study data. This is because the PeP

study data contained some demographic information that was not collected as part of the SSC, and some of the variables were captured differently across studies (e.g., interval versus categorical data). From the PeP study data, current age of child with ASD is reported in years. Parental level of education was collected and included the following categories: (a) Less than high school, (b) High School Diploma, (c) Associate's Degree, (d) Bachelor's Degree, (e) Master's Degree, and (f) Doctoral Degree (e.g., PhD, MD, DDS, OD, etc.). Current family household income was also reported and was captured on an interval level (e.g., $51,500). The income data were collapsed and presented in levels (e.g., Less than $20,000). All demographic information may be seen in Tables 1 and 2.

Parental perceptions of cause of ASD. Parental perceptions of cause of their child's ASD were measured using the Illness Perception Questionnaire – Revised for Autism (IPQ-RA; Al Anbar et al., 2010), which is a modified version of the IPQ-R (Moss-Morris et al, 2002) for use with parents of children with ASD. The IPQ-RA (like the IPQ-R and the original IPQ [Weinman et al., 1996]) is based on Leventhal's Model of Illness Representation and includes five components of illness representations (i.e., identity, cause, consequences, timeline, and control; Leventhal et al., 1980, 1998).

The IPQ-RA contains 76 items that compose nine subscales (which quantitatively represent the five components of illness representation). Seven of the subscales were identified using a principle-components analysis (PCA) and are: (a) Timeline (i.e., both acute and chronic) subscale; (b) Timeline-cyclical subscale; (c) Consequences subscale; (d) Personal Control subscale; (e) Treatment Control subscale; (f) Illness Coherence subscale; and (g) Emotional Representations subscale. A PCA was also conducted on the 18 causal items, which constituted the Cause subscale. The analysis identified three

factors (i.e., labeled personal attributions, environment, and heredity attributions) with Cronbach Alpha coefficients ranging from .67 to .86. The IPQ-RA also contains an Identity subscale, which demonstrated acceptable internal consistency reliability a coefficient of .75 (Al Anbar et al., 2010).

The current study included only the items from the IPQ-RA that comprise the Cause subscale. Additional modifications to this subscale were made (Mire et al., 2015) that were sustained in this study. As aforementioned, those changes included: (a) modifying the wording from "his/her disorder" to "your child's ASD", (b) changing the causal belief item of "heredity" to "genetics", and (c) the addition of causal items (i.e., "in utero stress or accident", "my child's brain structure", and "stress at birth") (Mire, et al., 2015). In total, the Cause subscale is comprised of 21-items on a five-point Likert scale ranging from 1 (*Strongly Disagree)* to 5 (*Strongly Agree*). The 21 Cause subscale items may be seen in Appendix A. Although Al Anbar et al. (2010) conducted a factor analysis on the Cause subscale of the IPQ-RA, there were several reasons why a second EFA was warranted. For instance, the Al Anbar et al. (2010) study had 18 items on the Cause subscale (compared to 21-items for the current study) and used a low sample size (n = 89). The current study had a larger sample size (n = 326) compared to the Al Anbar et al. (2010) study, which is more aligned with acceptable guidelines when conducting an EFA (Floyd & Widaman, 1995).

Treatments. Data on treatments that parents used with their children with ASD were collected as part of the PeP study and were included in the current study. For the PeP study, parents were presented with a comprehensive list of 116 different treatments used to treat ASD; this list of treatments was based on the treatments used in the Green

(2006) study. For each treatment used, the parent was asked to indicate at what ages (i.e., starting at age 1 through 18 years and older) the specific treatment was used. Parents were asked to select the age their child started the treatment through the age at which they stopped. For example, if a parent started a child on a treatment when he/she was age 8.5 years and stopped the treatment when the child was age 9 years, the parent would select ages 8-9 years for that treatment. Also, parents were asked to select the age at which a treatment was used even if it was only used for a short time (e.g., months or weeks). However, for the current study, ***only*** currently endorsed treatments were used. The complete list of treatments may be seen in Appendix B. For this study, a formal consensus coding approach was employed, with the goal of separating the treatments into categories for analysis (more information provided in the Data Analysis section).

Type of symptom onset and child age. Type of symptom onset was examined as a moderating variable between parental perceptions of cause of their child's ASD and categories of treatments endorsed by parents. Shumway et al. (2011) identified four patterns of onset (i.e., early onset, delay plus regression [loss in skills], plateau, regression) as a more inclusive conceptualization of the emergence of ASD symptoms (i.e., compared to regression versus early onset); also, this onset categorization scheme has been used in subsequent research to investigate the relationship between ASD-onset type and parental perceptions of cause (Goin-Kochel et al., 2014). Onset type was categorized through the aforementioned scheme using data from the ADI-R (Le Couteur et al., 2003), which was drawn from the SSC dataset. As stated earlier, the data from the SSC and the PeP study were linked through unique identifiers. The ADI-R was administered as part of the SSC study and is an in-depth, semi-structured parent interview

that is widely used as part of an evaluation to identify individuals with ASD. In order to administer the ADI-R for the SSC, examiners were required to establish inter-rater reliability with expert clinicians (i.e., at .90 or above); once established, reliability status was maintained through consultants' rigorous scrutiny of randomly selected ADI-R administrations, which had all been videotaped as a study requirement.

Using the categorization scheme created by Shumway et al. (2011), the following three ADI-R items were used: onset of symptoms within first 12 months, per hindsight (Item 4), loss of language skills (Item 11), and/or loss of social engagement/responsiveness (Item 25). Based on codes for these three items, the four onset patterns will be conceptualized as: (a) Early onset (i.e., symptoms present in the first 12 months, no losses; Item 4 = 0, Item 11= 0, and Item 25 = 0); (b) Delay plus Regression (i.e., some delays before loss; Item 4 = 0, Item 11 = 1, and/or Item 25 $\geq$ 1); (c) Plateau (i.e., no early delays, no loss; Item 4 $\geq$ 1, Item 11 = 0, and Item 25 = 0); and (d) Regression (i.e., no delays before clear loss; Item 4 $\geq$ 1, Item 11 = 1, and/or Item 25 $\geq$ 1).

In addition to type of symptom onset, current age of the child with ASD was examined as a potential moderating variable between parental perceptions of cause of ASD and treatment choice in the current study. As mentioned previously, data on age of child with ASD was collected in years (i.e., ages 5-25 years and older) and was drawn from the PeP study.

Procedures

Only families who had previously participated in the SSC and had consented to be recontacted for future studies about ASD (i.e., 1,325 families) were invited to participate

in the PeP study. The Simons Foundation Autism Research Initiative (SFARI) works in collaboration with the Interactive Autism Network (i.e., SSC@IAN) in the collection of additional data from these participants for approved studies. Before former SSC participants could be recontacted, SFARI required proof of institutional approval; the PI (Dr. Sarah Mire) was granted approval from the Committee for the Protection of Human Subjects (CPHS) at the University of Houston (UH) to complete data collection for the PeP study (protocols: 14217-01 and 14009-EX - [3718]). Furthermore, approval to conduct the current study was also obtained (protocol 16355-01). The first part in this section (i.e., PeP study) outlines the procedures for conducting the PeP study. The second part (i.e., Consensus coding of presented treatments) outlines the procedures for the focus group, which was conducted as part of research question two of the current study.

PeP study. Once approval was processed, the SSC@IAN team sent an email to each SSC participant who had consented to be recontacted with information about the PeP study. From that email, interested participants were able to opt in to receiving the link (i.e., via email) to complete the PeP survey. The initial email that participants received from PeP study staff included information about the study (e.g., study aims, brief description), a link to the survey, a unique eight-digit ID (i.e., used to protect anonymity), and the consent form (see Appendix C for the recruitment email; Appendix D for the consent). The PeP-survey link directed participants to the electronic survey, which was created using the Qualtrics survey platform. Qualtrics is a program created for professional researchers to use to collect web-based survey data; the program uses sophisticated firewall systems and implements other security features that protect data and identifying participant information. When participants clicked on the PeP survey

link, they were immediately instructed to enter their unique eight-digit ID (which was sent to them via email); for security reasons, a participant was locked out of the survey following five incorrect ID entry attempts. If locked out, participants were instructed to contact the PI who collaborated with the current author to reissue a new ID and survey link. Once the participant successfully accessed the survey, they were able to complete the measures that constituted the PeP study. Participants were informed that completion of the entire survey would take approximately one hour and 20 minutes.

The complete PeP survey included: (a) a Participation Questionnaire, which contained another copy of the consent, demographic questions, and queries regarding parent perceptions of ASD severity; (b) the IPQ-RA, which asked participants to relay their cognitive experiences of caregiving for a child with ASD across several dimensions (i.e., identity, timeline, cause, consequences, and cure/control); (c) the Family Adjustment Measure (FAM), which gathered information about family support, social support, coping, and distress related to raising a child with a disability; and (d) the treatment questionnaire, which asked participants to provide information about different treatments used and the ages treatments were used. In addition to these measures, participants also completed one of two additional measures, depending on the child's age. Participants who indicated that his/her child with ASD was age 11 or younger completed the Parenting Stress Index – Fourth Edition- Short Form (PSI-4-SF), a 32-item measure that aims to identify parent-child problem areas. If a parent indicated that his/her child was age 12 years or older, he/she completed the Stress Index for Parents of Adolescents (SIPA), a 112-item measure that identifies stressful areas in parent-adolescent interactions. Although the PeP study contained numerous measures, only the Cause

subscale from the IPQ-RA and the treatment questionnaire were used in the current study.

After participants completed the survey, they were redirected to a page where they had the opportunity to enter a random drawing for one of five mini iPads. This page asked participants to enter identifying information (i.e., name, email, address, phone number) in order to facilitate delivery of the prize. Participants were informed that the identifying information was independent of their survey responses and that entering into the drawing was completely voluntary.

Consensus coding focus group. The current study collected data on 116 different ASD treatments. As previously mentioned, participants were asked to indicate which treatment options they used across their child's lifespan. In order to separate treatments into meaningful and useful categories, the current study used a *formal consensus coding approach* to make a decision regarding the *structure* of the treatment categories (e.g., by empirical support, by function, combination). A formal consensus coding approach uses a set of procedures to engage a group of professionals in making a decision regarding a specified goal. Formal consensus coding approaches have been used since the 1950s within a variety of disciplines (i.e., medicine, social sciences, education) and operate largely on the following assumptions: (a) a group of individuals is more likely to make informed decisions than an individual person; (b) decisions made from a group of individuals have more authority; (c) group decision-making promotes discussion of ideas and encourages individual members to justify their perspectives; (d) use of formal consensus methods are more controlled and structured; and (e) use of formal consensus methods are more aligned with the scientific method (Black et al., 1999).

There are various formal consensus methods outlined in the literature (Black et al, 1999; Fink, Kosecoff, & Chassin, 1984; Nair, Aggarwal, & Khanna, 2012). The current study utilized a Nominal Group Technique (NGT), which involves a single structured group meeting where a panel independently generates ideas to specific questions in order to establish a prioritization of ideas (Delbecq, Van de Van, & Gustadfen, 1975; Fink, Kosecoff, & Chassin, 1984; Nair, Aggarwal, & Khanna, 2012). NGT was chosen among the available formal consensus approaches because it has several advantages, including that panel participants meet in person and have equal opportunity for participants to voice their opinions (Nair et al., 2012).

A list of potential panel participants was generated and included professionals and graduate students from local agencies who were involved in the treatment and/or research of children and families with ASD. Twelve graduate students and professionals were approached (i.e., by email, see Appendix E) about participating in the panel. Out of those approached, six professionals from three different local agencies (i.e., Texas Children's Hospital/Baylor College of Medicine, Harris Center for Mental Health and IDD, University of Houston) and three graduate students from the University of Houston (UH) participated in the focus group, which was conducted in May 2016.

The focus group duration was approximately two hours and took place at UH. Upon arrival, focus group participants were presented with several documents including a consent form (see Appendix F), a brief background survey (see Appendix G), and a meeting agenda (see Appendix H). Participants completed consent forms and background surveys at the beginning of the meeting. The author of the current study acted as the

facilitator of the meeting and a separate individual (who was not a focus group participant) recorded meeting notes.

The procedures for the focus group followed those outlined for a NGT meeting (Dunham, 1998). The first step was a brief introduction to the topic (i.e., ASD treatments) and an explanation of the goal of the meeting (i.e., "What is the best categorization scheme for ASD treatments?"). The second step involved each focus group participant independently generating ideas on categorization schemes for ASD treatments. Each participant was provided with a lined sheet of paper to record his or her ideas. The third step was a feedback session in which each focus group participant voiced one of his or her ideas. Each idea was written on a whiteboard that was visible to all participants. Participants voiced ideas (one at a time) around the table until all ideas had been recorded. The fourth step included discussion of recorded ideas. During this step, focus group participants were encouraged to raise any questions about a recorded idea with the goal of clarifying each idea. Once all ideas were clarified, the fifth step involved each focus group participant ranking each idea in order of what he or she considered the best categorization scheme for ASD treatments. Each participant received a follow-up email containing the results of the rank-ordered list. The categorization scheme that was ranked the highest was used in the current study as the basis for defining categories of treatment for analysis in the current study (see Results section).

Analytic Method

Research question one: Exploratory factor analysis. Using IBM's Statistical Package for the Social Sciences (SPSS) – Version 24 (IBM Corp., 2012), an exploratory factor analysis (EFA) of the IPQ-RA Cause subscale was conducted with the goal of

identifying the underlying factors. The current study used a Principal Components Analysis (PCA) as the factor extraction method, which is deemed an acceptable extraction method for factor analysis (Schonemann, 1990; Velicer & Jackson, 1990). A multi-method approach was used to determine how many factors to retain for rotation, which included a combination of visual inspection of a scree plot and inspection of the Eigenvalues (i.e., factors with Eigenvalues greater than 1 were retained). Furthermore, given that there was no reason within the literature to believe the factors would be correlated with one another, an orthogonal varimax rotation was used as the rotation method. Following rotation, factor patterns were examined. Research suggests using a cut-off score to determine whether an item loads on a factor (i.e., general rule of thumb is .32 or greater; Tabachnick and Fidell, 2001).

Several assumptions must be met in order to ensure that results from the EFA are generalizable to the population. One assumption is linearity between all variables. This assumption was tested by checking all variables using the correlation matrix table. Any variable that does not have at least one correlation with another variable (i.e., $\geq .2$) may be measuring something different from all the other variables. Variables that are not correlated with any other variable should be either removed from analysis or examined further and noted. A second assumption is sampling adequacy. This was tested using the Kaiser-Meyer-Olkin (KMO) statistic for the overall data set (should be above .6), the KMO measure for each individual variable (at minimum above .5), and the Bartlett's test of Sphericity, which should be statistically significant ($p < .05$). A final assumption is that the data are normally distributed (i.e., absence of substantial skewness or kurtosis of variables). This assumption was checked by an examination of the skewness and kurtosis

values of each variable (see Results section). Criteria for what is considered acceptable cutoffs for skewness and kurtosis vary with some researchers indicating variables should be within +/- 3 for skewness and +/- 8 for kurtosis (Kline, 2005).

Research question two: Consensus coding focus group and categorization of treatments. The outcome of the focus group (see Results section) provided the categorization scheme by which treatments were then categorized. Before treatments could be categorized, the data were screened for any treatments for which *all* participants did not endorse *any* current use. These treatments were removed from consideration within the current study (see Results section). The next step was to categorize the remaining treatments. As previously mentioned, one way to report the most accurate representation of evidence-based data is to utilize large-scale systematic reviews (Missouri Autism Guidelines Initiative, 2012; Mulrow, 1994). Therefore, several large-scale reviews were identified and used to categorize the treatments.

In 2012, the Missouri Autism Guidelines Initiative (MAGI) released a report that combined information from several nationally recognized systematic research reviews. Each review contained within the MAGI report focused on providing research evidence for the effectiveness of various treatments for ASD. The MAGI (2012) report included systematic reviews from: (a) The Centers for Medicaid and Medicare Services (CMS), which compiled 271 research articles from 1998-2008 and focused on providing information about the effectiveness of behavioral and psychosocial interventions for ASD (Young et al., 2010); (b) The National Standards Project (NSP1), Phase One, that integrated research from 775 studies from 1957-2007 to provide information on the strength of evidence supporting educational and behavioral interventions for ASD

(National Autism Center, 2009); (c) The Agency for Healthcare Research and Quality (AHRQ), which reviewed 159 studies from 2000-2010 and focused on behavioral, educational, medical, allied health, and CAM interventions (Warren et al., 2011); (d) the Stanford Autism Research Team (StART), which included 115 articles from 1994-2007 and focused on current pharmacological and CAM treatments (Huffman et al., 2011); and (e) a private review that investigated Comprehensive Treatment Models (CTM), which included an evaluation of 30 CTMs using data from published literature, procedural information (e.g., curriculum), and information gathered from the program developers (Odom, Boyd, Hall, & Hume, 2010).

In addition to the systematic reviews covered in the MAGI report, other reviews were identified and used to categorize treatments in the current study. The National Standards Project (NSP2) conducted one additional review, which was a second phase review that built on the first NSP review mentioned in the MAGI report. This report reviewed 389 articles published between 2007 and 2012 (National Autism Center, 2015). Another identified review was conducted by the National Professional Development Center on Autism Spectrum Disorders (NPDC) and reviewed 465 articles published between 1990 to 2011 (Wong et al., 2014).

Once all treatments were categorized using the identified systematic reviews, the overall frequency of treatments and the categories of treatments were used as outcome variables in the current study.

Research question three: Poisson Regressions. The third research question was examined using SPSS – Version 24.0 (IBM Corp., 2012). In order to examine the third research question, a non-linear regression called the Poisson regression was used, which

is a type of generalized linear model analysis. A Poisson regression provides the ability to investigate dependent variables that consist of "count data" (i.e., frequency counts of treatments). The first analysis was a single Poisson regression, which investigated the potential of the independent variables (i.e., IPQ-RA Cause subscale factors) and moderators (i.e., current child age and onset type) to predict the *overall* frequency of current treatment use reported by participants (i.e., outcome variable). The second analysis consisted of four separate Poisson regressions. These series of analyses investigated whether the independent variables and moderators predicts frequency of treatments within each outcome category (see Results section for description of categories). Moderators used in the current study included current child age (i.e., continuous variable) and onset type (i.e., categorical variable), which investigated the interaction between the parental perceptions of cause and frequency of current treatment use. Interaction terms were created for current child age and type of symptom onset (i.e., Early Onset, Delay plus Regression, Plateau, and Regression). In order to make the intercept variable more easily interpretable, two modifications were made to the data. First, the Likert scale of the predictor variables (i.e., IPQ-RA Cause subscale factors) was transformed so that "0" equals "Strongly Disagree" and "4" equals "Strongly Agree". Secondly, the continuous moderator variable of age was centered to the mean.

Several assumptions must be met in order to ensure that these results are generalizable to the population. The first assumption is that the dependent or outcome variable must consist of count data. This assumption was met as the outcome variable consisted of the number of current treatments endorsed by participants. The second assumption is that there must be one or more independent or predictor variables,

measured on a continuous, ordinal, or nominal scale. This assumption was also met as there were six predictor variables (i.e., factors of the Cause subscale), which are represented as the mean of the Likert scale responses. For the current study, these variables were treated as continuous. A third assumption is independence of observations, which means that observations were not subject to an outside influence common to several of the observations. This assumption was met for the current study. A final assumption is that the means and variance of the model is similar, which is unique to the Poisson regression. This assumption was tested using the Pearson Chi-Square statistic; this statistic should be close or equal to one. Statistics over one represent an overdispersion of the data (i.e., observed variance is higher than the variance in the theoretical model) and statistics under one represent an underdispersion of the data (i.e., observed variance is lower than the variance in the theoretical model; see Results section).

Chapter IV

Results

Preliminary Data Analysis. Prior to data analysis, data were cleaned and checked for missing data and outliers. Missing data may affect the results of a study and the generalizability of the results. Frequency tables were generated to examine the number of missing cases for the variables used in analyses (i.e., IPQ-RA Cause subscale items, type of symptom onset, child age). For the IPQ-RA Cause subscale (i.e., 21 items), one participant did not complete 13 items (61.9 % of the subscale), a second participant did not complete 21 items (100% of the subscale), a third participant did not complete two items (9.5% of the subscale), and an additional two participants did not complete one item (4.7% of the subscale). Furthermore, an additional three participants were missing data for the type of symptom onset variable. There were no missing data for the child age variable.

These eight participants were visually examined on a number of demographic characteristics and did not demonstrate any systematic differences from the overall PeP sample. More specifically, these eight participants were primarily mothers (87.5%), Caucasian (75%), had incomes higher than $81,000 (66.6%), and had an education level of college graduate or higher (75%). The lack of systematic differences suggests that these missing data are missing completely at random (MCAR) as the participants had the option to not complete any item on the survey. In situations where data are suspected to be MCAR and the participants with missing data do not exceed 5% of the entire sample, a listwise deletion method (i.e., deleting an entire record if a single value is missing) has been suggested (Parent, 2012). Together, the eight participants consist of 2.5% of the

entire sample. Therefore, the decision was made to delete these eight participants from analyses. For the treatment data, participants indicated whether or not their child had received a certain treatment (i.e., 116 possible treatments) across child age (i.e., age one through 18 and over). While it is possible that participants failed to identify certain treatments at any age, it is not possible to determine whether these data are truly missing.

It is also important to identify outliers, as they can affect the results of the data analysis. Outliers of the IPQ-RA Cause subscale items were assessed using a visual inspection of histograms. It should be noted that the Cause subscale items are Likert scale data (ranging from 1 to 5) that are being treated as continuous variables for the current study. After examining the histograms for each item, it was determined that most of the items are highly skewed. More specifically, items 1, 3, 4, 5, 6, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 19, 20, and 21 were skewed right. Items 2 and 18 were skewed left, while item 7 followed a bell-shaped curve. For the items that were skewed right, many of the participants selected "Strongly Disagree" or "Disagree" for the item representing what they felt was a cause for their child's ASD. For items 2 (i.e., "genetics") and 18 (i.e., "my child's brain structure"), which were skewed left, many of the participants selected "Strongly Agree" or "Agree" for those items representing a cause of their child's ASD. For item 7 (i.e., "environmental pollution"), which followed a bell-shaped curve, many of the participants selected "Neither Disagree or Agree" or "Agree" as being a potential cause of their child's ASD.

Research Question One: Exploratory Factor Analysis

Descriptive statistics. A principal components analysis (PCA; i.e., type of EFA) was conducted on the 21-items of the Cause subscale of the IPQ-RA in order to reveal the

underlying factor structure. See Appendix A for the complete list of the 21-items on this subscale. The 21-items were reported on a 5-point Likert scale ranging from 1 ("Strongly Disagree") to 5 ("Strongly Agree"). The means, standard deviations, skewness/kurtosis for each item on the Cause subscale may be seen in Table 3.

Table 3.

Means, Standard Deviations, and Skewness/Kurtosis of each Item on the Cause Subscale

Cause Item	*M*	*SD*	Skewness	Kurtosis
General life stress	2.19	1.20	.706	-.663
Genetics	4.21	0.83	-1.251	2.125
A germ or virus	2.26	1.12	.565	-.686
Diet or eating habits	2.25	1.14	.501	-.939
Chance or bad luck	2.41	1.30	.349	-1.236
Poor medical care in the past	1.61	0.78	1.271	1.226
Environmental pollution	3.20	1.21	-.436	-.756
My own behavior or decisions	1.87	0.97	.885	-.073
In utero stress or accident	2.50	1.23	.238	-1.124
Mental attitude/negative views	1.55	0.80	1.578	2.501
Family worries about ASD	1.50	0.76	1.622	2.571
Will of God	2.21	1.34	.645	-9.27
My own emotional state	1.77	0.97	1.149	.476
My or my partner's age	2.13	1.20	.668	-.792
My own alcohol consumption	1.38	0.68	1.956	3.730
My own tobacco consumption	1.36	0.65	2.061	4.866
Accident or injury	1.56	0.83	1.448	1.517
My child's brain structure	3.70	1.12	-.950	.321
Deterioration of my child's immunity	2.25	1.21	.645	-.628
Toxins found in vaccines	2.41	1.34	.510	-.950
Stress at birth	2.50	1.29	.269	-1.173

A majority of the means (85%) are under 3, indicating participants had a tendency to "Strongly Disagree" or "Disagree" with many of the items. A majority of the skewness/kurtosis statistics were within acceptable ranges, with the exception of the

kurtosis statistic for Cause items 15 and 16, which are slightly higher although some researchers have indicated acceptable ranges being between +/- 8 (Kline, 2005). Additionally, Table 4 presents the frequencies and percentages of endorsement of each participant by each item on the Cause subscale.

Table 4.

Percentages of Participant Endorsement of Causes by Item (N = 326)

	Strongly Disagree	Disagree	Neither Disagree nor Agree	Agree	Strongly Agree
Cause Subscale Items	%	%	%	%	%
1. General life stress	**36.8**	**30.7**	12.6	16.3	3.7
2. Genetics	1.2	3.1	9.5	**46.0**	**40.2**
3. A germ or virus	**30.1**	**34.4**	17.5	15.6	2.5
4. Diet or eating habits	**32.2**	**31.9**	16.0	18.4	1.5
5. Chance or bad luck	**35.9**	**19.3**	17.8	22.1	4.9
6. Poor medical care in the past	**54.0**	**34.7**	7.7	3.7	0.0
7. Environmental pollution	13.2	13.5	25.2	36.2	12.0
8. My own behavior or decisions	**45.4**	**30.4**	16.9	6.7	0.6
9. In utero stress or accident	28.5	23.0	23.0	21.2	4.3
10. Mental attitude/negative views	**60.1**	**28.5**	8.3	2.5	0.6
11. Family worries about ASD	**62.6**	**27.6**	7.1	2.5	0.3
12. Will of God	46.6	13.2	19.3	14.1	6.7
13. My own emotional state (e.g., depression, anxiety)	**50.9**	**29.8**	11.0	7.7	0.6
14. My or my partner's age	**42.6**	**22.4**	17.2	15.0	2.8
15. My own alcohol consumption	**71.2**	**22.1**	4.6	2.1	0.0
16. My own tobacco consumption	**72.1**	**21.5**	5.2	0.9	0.3
17. Accident or injury	**61.3**	**24.8**	10.1	3.4	0.3
18. My child’s brain structure	7.1	7.7	16.3	**46.3**	**22.7**
19. Deterioration of my child's immunity	**35.3**	**27.6**	19.0	13.2	4.9

20. Toxins found in vaccines/immunizations	35.9	19.6	21.8	13.2	9.5
21. Stress at birth	31.6	19.6	22.1	20.9	5.8

For items 1, 3, 4, 5, 6, 8, 10, 11, 13, 14, 15, 16, 17, and 19, 60% or more participants endorsed either "Strongly Disagree" or "Disagree". For Item 2 (i.e., "Genetics"), over 80% of participants endorsed "Strongly Agree" or "Agree" for this cause being a perceived cause of their child's ASD. Similarly, over 60% of participants endorsed "Strongly Agree" or "Agree" for item 18 (i.e., "My child's brain structure"). For items 7, 9, 12, 20, and 21, participant's responses were more evenly distributed.

Principal components analysis. Before conducting the principle components analysis (PCA), several assumptions were tested. Linearity between variables was tested by examining the correlation matrix table; it was discovered that all variables had at least one correlation with another variable (at $\geq .2$), which indicated there were no variables measuring something different than all the other variables. Sampling adequacy was tested through several methods to ensure that PCA was an appropriate analysis for the current sample. The Kaiser-Meyer-Olkin (KMO) measure was .834, which verifies that the sample size is more than acceptable, or meritorious, for the analysis (Field, 2009; Kaiser, 1974). All KMO measures for individual items were at .57 or above, which is above the acceptable limit of .5 (Field, 2009). Furthermore, Bartlett's test of sphericity was significant $\chi^2 (326) = 2233.31$, $p < .001$ indicating that the correlations between items were sufficiently large for a PCA. Normality was checked by examining the values for the skewness and kurtosis measures. As previously stated, these values were within normal range according to some researchers (i.e., Kline, 2005).

For the current study, PCA was used as the factor extraction method as the goal was to reveal the underlying factors. Orthogonal varimax rotation was used as the rotation method. In order to determine the number of factors to be retained, Eigenvalues and a visual inspection of the scree plot were examined. Both of these methods resulted in retention of six factors. Table 5 presents the Eigenvalues and percentages of variance explained by each factor.

Table 5.

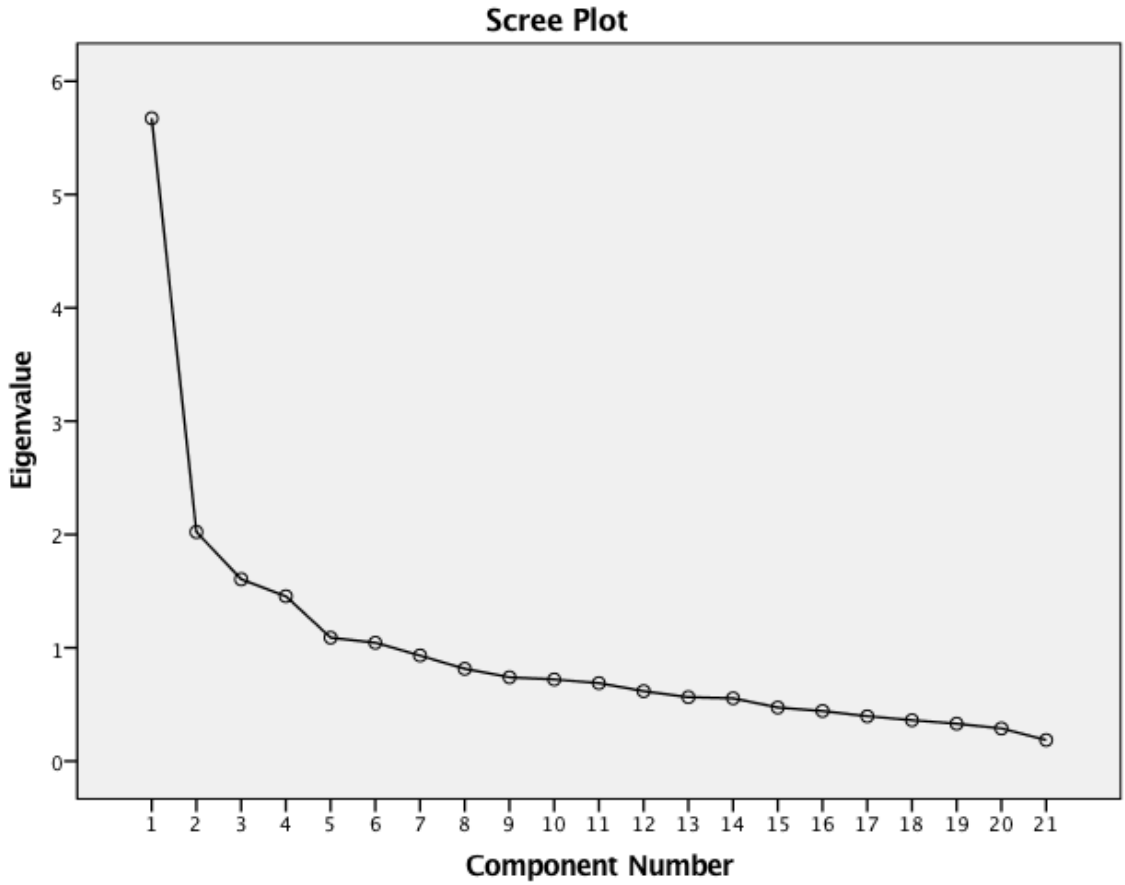
PCA Eigenvalues and Percentages of Variance Explained

Factor	**Initial Eigenvalues**			**Rotation Sums of Squares Loadings**		
	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %
1	5.67	27.01	27.01	3.02	14.39	14.39
2	2.02	9.63	36.64	2.88	13.71	28.11
3	1.61	7.64	44.28	2.58	12.27	40.38
4	1.46	6.93	51.21	1.82	8.64	49.02
5	1.09	5.19	56.40	1.52	7.24	56.26
6	1.05	4.98	61.38	1.08	5.12	61.38
7	.93	4.44	65.82			
8	.82	3.88	69.70			
9	.74	3.52	73.22			
10	.72	3.43	76.65			
11	.69	3.28	79.93			
12	.62	2.94	82.87			
13	.56	2.69	85.56			
14	.56	2.64	88.20			
15	.47	2.25	90.45			
16	.44	2.11	92.56			
17	.40	1.89	94.45			
18	.36	1.72	96.17			
19	.33	1.57	97.74			
20	.29	1.37	99.11			
21	.19	.89	100.00			

In total, six factors had Eigenvalues over 1 and those factors accounted for a cumulative variance of 61.38%. The first factor was comprised of five items and explained 14.39% of the variance. Factor 2 also contained five items and accounted for 13.71% of the variance. The third factor contained five factors and explained 12.27% of the variance. The last three factors contained two items each and accounted for 8.64%, 7.24%, and 5.12% of the variance respectively. In addition to the Eigenvalues, the scree plot was examined to decide how many factors to retain. Figure 1 contains the scree plot for the current study.

Figure 1.

Scree Plot for PCA for Current Study



Examination of the scree plot confirms that there are six factors that should be retained. Therefore, based on the Eigenvalues and the scree plot, it was decided that six factors would be retained for the current study. Following the decision on how many factors to retain and rotating the factors (i.e., using orthogonal varimax rotation), factor patterns were examined. Table 6 contains the factor loadings and the associated communalities.

Table 6.

Factor Loadings and Communalities for Rotated Matrix

	Factors						**Communalities**
Cause Item and Factor Name	**1**	**2**	**3**	**4**	**5**	**6**	
Factor 1: Personal Attributions							
1. General life stress	**.747**						.605
8. My own behavior or decisions	**.727**						.574
10. Mental attitude/negative views	**.699**						.521
11.Family worries about ASD	**.672**						.572
13. My own emotional state (e.g., depression, anxiety)	**.636**	.459					.389
Factor 2: Parental Risk Factors							
6. Poor medical care in the past		**.865**					.528
14. My or my partner's age		**.865**					.563
15. My own alcohol consumption		**.590**		.397			.569
16. My own tobacco consumption	.355	**.531**					.782
17. Accident or injury		**.416**			.332		.674
Factor 3: Environmental Risk Factors							
3. A germ or virus			**.757**				.675
4. Diet or eating habits			**.717**			.301	.706
7. Environmental pollution			**.697**				.653
19. Deterioration of my child's immunity			**.666**				.424
20. Toxins found in	.483		**.574**				.773

vaccines/immunizations			
Factor 4: Utero/Birth Stress			
9. In utero stress or accident	**.846**		.783
21. Stress at birth	**.740**		.594
Factor 5: Structural *Composition*			
2. Genetics		**.738**	.584
18. My child's brain structure		**.737**	.640
Factor 6: Metaphysical			
5. Chance or bad luck		**.757**	.652
12. Will of God		**-.384**	.627

All items exceeded the suggested minimum loading for an item (i.e., .32; Tabachnick & Fidell, 2001), with the lowest loading being -.384. All items loaded positively onto their respective factors, except for item 12 (factor six), which loaded negatively. This negative loading suggests that the two items for factor six have an inverse relationship; more specifically, the more someone believes their child's ASD is due to the "Will of God" (i.e., Item 12), the less someone believes their child's ASD is due to "Chance or bad luck" (i.e., Item 5) and visa-versa. The communalities, which represents the total amount of variance an original variable shares with all other variables, were all above .2, which means all values were in the moderate to high range (Yong & Pearce, 2013) and no one item had a high amount of unique variance. The current factor loadings presented in a complex structure with several cross-loading items. However, items were considered conceptually and the highest factor loadings were determined to represent the best fit for each item on their respective factors when considered with the other items on that factor.

Inter-item Spearman correlations between the individual items within a factor were also examined. Factors were found to have a range of correlations. Spearman correlations for factors with five items (i.e., Personal Attributions, Parental Risk Factors,

Environmental Risk Factors) ranged from .38 to .88. Mean inter-item Spearman correlations were .55 for factor one, .49 for factor two, and .40 for factor three. Inter-item correlations for factors with two items (i.e., In Utero/Birth Stress, Structural Composition, Metaphysical) were at .53, .30, and .11 respectively. Clark and Watson (1995) suggest that individual and mean inter-item correlations should fall between .15 and .50, indicating a moderate correlation. A majority of the correlations fell within this suggested range.

Naming the factors. The first factor contained the following five items: "General life stress", "My own behavior or decisions", "Mental attitude/negative views", "Family worries about ASD", and "My own emotional state (e.g., depression, anxiety)". This set of items appears to encompass stress, behaviors, and psychological states; thus, factor one was labeled Personal Attributions. The second factor contained the following five items: "Poor medical care in the past", "My or my partner's age", "My own alcohol consumption", "My own tobacco consumption", and "Accident or injury". These items seem to represent various parental risk factors that a person may attribute to the cause of their child's ASD. Therefore, factor two was labeled Parental Risk Factors. The third factor contained the following five items: "A germ or virus", "Diet or eating habits", "Environmental pollution", "Deterioration of my child's immunity", and "Toxins found in vaccines/immunizations". These items all attribute cause to environmental origins, which resulted in factor three being named Environmental Risk Factors. The fourth factor contained the items "In utero stress or accident" and "Stress at birth". These two items both deal with stress specific to the pregnancy and birthing process; thus, this factor was labeled In Utero/Birth Stress. Factor five was composed of the items "Genetics" and "My

child's brain structure", which both indicate problems with the structural composition of an individual. Factor five was labeled Structural Composition. Finally, factor six was comprised of the items "Chance or bad luck" and "Will of God". These items explain forces of nature beyond one's control. Thus, factor six was labeled Metaphysical.

Research Question Two: Consensus Coding Group, Categorization of Treatments, and Treatment Data

The goal of the second part of the current study was to categorize the 116 treatments into treatment categories for analysis. The categorization scheme was determined using a consensus coding focus group (i.e., NGT). Once the scheme was determined, the treatments were categorized using large-scale systematic reviews.

Consensus coding group outcome. A nominal group technique (NGT) was used in the current study to generate the categorization scheme for ASD treatments. The focus group was composed of six professionals from three different local agencies (i.e., Texas Children's Hospital/Baylor College of Medicine, Harris Center for Mental Health and IDD, University of Houston) and three graduate students from the University of Houston. All focus group participants completed a brief background survey (see Appendix G). Results of this survey may be seen in Table 7. For this table, categories with no endorsements were removed for readability purposes.

Table 7.

Brief Background Survey for Focus Group Participants (N = 9)

Question		%
Currently a graduate student?		33.3
Year in Graduate School		
	First Year	11.1
	Fourth Year	11.1

	Fifth Year or Beyond	11.1
	Not a Student	66.7
Current Employment Setting[a]		
	University – Graduate Student	33.3
	University – Faculty	33.3
	Community Mental Health	11.1
	Hospital	33.3
	Outpatient	11.1
Years in Current Setting		
	Less than One Year	33.3
	2-4 Years	22.2
	Over 5 Years	44.4
Past Employment Settings[a]		
	University Clinic	33.3
	Public School	44.4
	Hospital[b]	88.9
	Community Mental Health	33.3
	Research Lab	22.2
	Residential Treatment Center	22.2
Capacity of Work with Individuals with ASD[a]		
	Assessment	88.9
	Individual Therapy (including behavioral)	77.8
	Group Therapy	77.8
	Family Therapy	44.4
	Consultative Services	66.7
	Research	88.9
Years Working with Individuals with ASD		
	1-10 Years	44.4
	10-20 Years	44.4
	Over 20 Years	11.1

[a] Participants were asked to select all that apply
[b] This included in-patient hospital and academic medical center

Three focus group participants were graduate students in a range of years of study (i.e., first year through fifth years). A majority of participants were currently employed as graduate students, university faculty, and/or in a hospital setting with most participants in their respective settings for less than one year or over five years. A majority of focus

group participants endorsed working in a hospital setting in the past (88.9%). Focus group participants indicated having worked with individuals with ASD in various capacities (i.e., assessment, individual therapy, group therapy, family therapy, consultative services, research) across a range of years.

The procedures for the NGT technique are outlined in detail in the procedures section. Results from the NGT revealed that focus group participants generated twelve possible categorization schemes, which could be used to categorize ASD treatments. These categorization schemes were meant to provide various ways from which treatment categories could be generated and, ultimately, treatments could be categorized. The generated categorization schemes and labels for the schemes were: (1) outcome-focused (e.g., what symptom is the treatment trying to target?), (2) provider/setting/implementation (e.g., who provides the treatment?, What setting is the treatment meant for?), (3) demand/resources (e.g., incorporates resources needed and cost to implement treatment), (4) theoretical basis/discipline (e.g., behavioral treatment versus a biological treatment), (5) developmental (e.g., chronological age or developmental age the treatment is meant for), (6) delivery model (e.g., systems treatment versus individual treatment), (7) dangerousness (e.g., how risky is the treatment?), (8) evidence-base (e.g., what evidence does the treatment have to support effectiveness?), (9) treatment outcome (e.g., duration and effect of treatment, generalizability), (10) acceptability by provider (e.g., does the treatment provider support use of the treatment?), (11) longevity of treatment (e.g., how long does the treatment take to implement?), and (12) popularity (e.g., what is the prevalence of use of the treatment?).

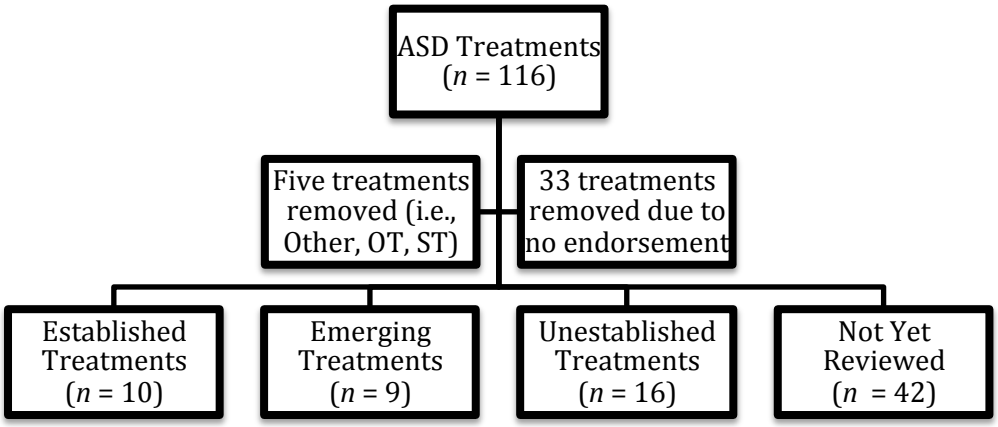
Following the generating of the twelve aforementioned categorization schemes, each focus group participant ranked the categories from 1 to 12 with 1 being the "most desirable categorization scheme" and 12 being the "least desirable categorization scheme". Results from the focus group participant ranking revealed that the evidence-based categorization scheme was the most desirable method by which to categorize treatments.

Categorization of treatments. Once the categorization scheme was identified via the focus group outcome (i.e., by evidence-base), the treatments were categorized. As previously mentioned, one way to report the most accurate representation of evidence-based data is to utilize large-scale systematic reviews (MAGI, 2012; Mulrow, 1994). Therefore, several large-scale reviews were identified and used to categorize the treatments (Huffman, et al., 2011; National Autism Center, 2009, 2015; Odom et al., 2010; Warren et al., 2011; Wong et al., 2014; Young et al., 2010), which are outlined in detail previously.

It should be recognized that there is no uniform definition of evidence-based practice and definition of this construct varies across systematic reviews. However, there are various core themes inherent in each review including: (a) the quality characteristics of research studies is assessed (e.g., research design, measurement of dependent and independent variables, intervention effects), (b) similar outcomes were found across more than one study conducted by independent research groups, and (c) evidence was considered by professionals with expertise (MAGI, 2012; National Autism Center, 2015; Wong et al., 2014). For the purposes of the current study, the categories of evidence-base treatments were: (a) Established, (b) Emerging, and (c) Not Established. This and similar

methods of categorizing treatments by evidence-base is supported within the literature (Chambless & Hollon, 1998; National Autism Center, 2014). Furthermore, an additional category was created for the current study (i.e., Not Yet Reviewed). The Not Yet Reviewed category contained those treatments that were not included in any of the systematic reviews aforementioned, suggesting they have not yet been studied. Therefore, four categories of treatments (i.e., by evidence-base) were created. For the current study, data were collected on 116 different treatments. Figure 2 provides a pictorial representation of how the treatments were categorized.

Figure 2. *Treatment Categorization Flow Chart*



Five treatments were removed from consideration from the current study. One of these treatments was labeled as "Other", which was removed from consideration, as it was unable to be categorized. Furthermore, four additional treatments (i.e., occupational therapy – school, occupational therapy – private, speech therapy – school, speech therapy – private) were removed from consideration because these are not considered unique ASD treatments, but rather broader treatment categories. Also, after examining the treatment data, it was discovered that 33 treatments were not endorsed as being currently used by *any* participant. These treatments were removed from consideration, resulting in

77 remaining treatments to categorize. The treatments that were removed due to lack of participant endorsement may be seen in Appendix I. Of the remaining 77 treatments, 10 treatments fell into the Established category, nine were categorized as Emerging, 16 were categorized as Not Established, and 42 were categorized in the Not Yet Reviewed category. A breakdown of how each treatment was categorized by each utilized systematic review may be seen in Appendix J; Appendix K provides a list of the various treatments within each category. Several treatments were reviewed by multiple systematic reviews. In these cases, if the level of evidence-base varied across review, the treatment was categorized by the highest level of evidence available (e.g., Established over Emerging).

Treatment data. A total of 77 treatments for ASD were examined for the current study. The current study examined whether or not parents were *currently* using any of these treatments. On average, participants endorsed using a mean of 2.85 (*SD* = 3.08) current treatments for their child with ASD. Table 8 provides a frequency and the corresponding percentages of the number of different treatments participant's reported currently using.

Table 8.

Frequencies and Percentages of Endorsement of Current Treatment Use

	PeP (n = 326)	
Number of Treatments	Frequency	%
0	79	24.2
1-3	144	44.1
4-6	69	21.3
7-9	23	7.1
10-13	6	1.8
14-17	5	1.5

Interestingly, 79 participants (24.2%) did not endorse current use of any of the 77 treatments. The largest number of participants (*n* = 144; 44.1% endorsed using one to three current treatments. Following that, 69 participants (21.3%) endorsed using four to six current treatment types. Only 34 participants (10.4%) endorsed currently using seven or more treatments, with the highest number of treatments currently used at 17.

As previously mentioned, the treatments were categorized into four categories by level of evidence, which was based on systematic reviews. Table 9 presents the frequencies and percentages of treatments used within each category.

Table 9.

Percentages of Endorsement of Current Treatment Use By Category

	Established	Emerging	Not Established	Not Yet Reviewed
Number of Treatments Endorsed	%	%	%	%
0	44.2	77.3	60.1	58.6
1	25.8	19.3	20.2	24.2
2	15.6	2.1	10.1	10.1
3	7.1	1.2	5.5	4.0
4	5.5	0.0	2.5	1.5
5	1.5	0.0	0.3	0.3
6	0.3	0.0	0.9	0.9
7	0.0	0.0	0.3	0.3

Across all categories, the largest majority of participants (*n* = 252; 77.3%) endorsed using no current treatments within the emerging category. When treatment use was endorsed, the largest frequency of use was one treatment across categories with 84 participants (25.8%) using a single treatment within the Established category, 63 participants (19.3%) using a single treatment within the Emerging category, 66

participants (20.2%) using a single treatment within the Not Established category, and 79 participants (24.2%) using one treatment within the Not Yet Reviewed category.

Research Question Three: Poisson Regressions

A series of Poisson regressions were performed to investigate the relationship between participant beliefs about the cause of their child's ASD and current treatment use. The moderators used in the current analyses were current child age and onset type (i.e., Early Onset, Plateau, Early Onset Plus Regression, Regression). As stated previously, for this analysis, the Likert scale data was modified from 0 ("Strongly Disagree") to 4 ("Strongly Agree") to allow for simpler interpretation of the intercept.

Descriptive statistics. The means, standard deviation, and skewness and kurtosis of each of the six Cause subscale factors may be seen in Table 10.

Table 10.

Means, Standard Deviations, and Skewness/Kurtosis of each Cause Factor

Factors	*M*	*SD*	Skewness	Kurtosis
1: Personal Attributions	.78	.71	.791	-.054
2: Parental Risk Factors	.61	.58	.942	.579
3: Environmental Risk Factors	1.47	.86	.201	-.456
4: In Utero/Birth Stress	1.50	1.10	.236	-.905
5: Structural	2.95	.78	-.870	.886
6: Metaphysical	1.31	.97	.273	-.570

A majority of the means (83%) are under 2, indicating that on average, participants had a tendency to "Strongly Disagree" or "Disagree" with many of the items within the Personal Attributions, Parental Risk Factors, Environmental Risk Factors, In Utero/Birth Stress, and Metaphysical factors. For the Structural Composition factor, the mean suggests that many participants tended to "Strongly Agree" or "Agree" with the

items representing that factor on average. All skewness/kurtosis statistics were within acceptable ranges (i.e., =/- 3 for skewness and =/- 8 for kurtosis; Parent, 2012). For the child age variable (continuous moderator), the mean was 13.56 (*SD* = 3.45). For the onset type variable (categorical moderator), 76.7% of children with ASD experienced an Early Onset, 9.2% experienced a Delay plus Regression, 8.3% experienced a Plateau, and 5.8% experienced a Regression. Additionally, visual inspections of the histograms for each cause factor were conducted (see Appendix L). Most of the factors (i.e., Factor 1/Personal Attributions, Factor 2/Parental Risk Factors, factor four/In Utero/Birth Stress, and factor six/Metaphysical) were generally skewed to the right indicating the average scores tended to lean towards 0 or 1 ("Strongly Disagree" or "Disagree"). Factor 5 (i.e., Structural Composition) was skewed left meaning many participants' average ratings leaned towards 3 or 4 ("Agree" or "Strongly Agree"). Factor 3 (i.e., Environmental Risk Factors) was bimodal with spikes at 0 ("Strongly Disagree") and 1 ("Disagree").

For the current study, two sets of moderated Poisson regressions were performed. The first investigated whether parental perceptions of cause of their child's ASD predicted *overall* frequency of current treatment use. The second examined whether parental perceptions of cause predicted frequency of current treatment use *within the four created evidence-based categories*.

Poisson regression: predicting overall frequency. Prior to conducting this analysis, the assumption that the means and variance of the model are similar was tested using the Pearson Chi-Square statistic, which should be close or equal to one. For this analysis, the Pearson Chi-Square was 2.639, which indicates that the data are overdispersed (i.e., variance is larger than the mean). A likely explanation for this

overdispersion is the excess number of participants who reported using no current treatments, leading to an excess number of zeros in the dataset. This is a common problem in psychological studies (Atkins, Baldwin, Zheng, Gallop, & Neighbors, 2013; Hua, Wan, Wenjuan, & Paul, 2014) and is discussed further in the Limitations section. Furthermore, to check for problems with multicollinearity (i.e., high correlations between predictor variables, which can affect calculations regarding individual predictors), intercorrelations between factor scores were checked and found to be in the low to moderate range with the highest correlation at .49. Therefore, issues related to multicollinearity were not deemed to be of major concern in the current study.

The first Poisson regression investigated: (1) whether the independent variables (i.e., six factors of the Cause subscale) predicted the overall frequency of current treatment use, and (2) whether the moderating variables (i.e., current child age and onset type) influenced these potential relationships. For the categorical moderator (i.e., onset type), the reference variable was the regression type meaning the other three categorical variables are interpreted in relation to this variable. The intercept was defined as the number of treatments when there is a 0 ("Strongly Disagree") on all factor scales, regression onset type equals 0 (i.e., not present), and the mean age equals 0 (centered to the mean age of 13.56 years). The significance cut-off level was set at .05. Tables 11 through 15 depict the regression coefficients (i.e., *B*), Wald statistics, odds ratios (i.e., *Exp(B)*), and 95% confidence intervals between each of the predictors, moderators, and interactions with frequency of overall current treatments. The results of this analysis are presented in Table 11.

Table 11.

Poisson Regression with Predictors, Moderators, and Interactions on Overall Frequency of Current Treatment Use

Predictor	*B*	S.E.	Wald	*p*	*Exp(B)*	95% CI for *Exp(B)* Lower	Upper
(Intercept)	.219	.399	.301	.583	1.245	.569	2.727
F1 (Personal Attributions)	-.334	.254	1.736	.188	.716	.436	1.177
F2 (Parental Risk Factors)	-.185	.350	.280	.597	.831	.419	1.649
F3 (Env Risk Factors)	***.301***	***.108***	***7.747***	***.005***	***1.351***	***1.093***	***1.670***
F4 (In Utero/Birth Stress)	.296	.165	3.240	.072	1.345	.974	1.858
F5 (Structural Comp)	.045	.126	.130	.719	1.046	.818	1.339
F6 (Metaphysical)	***.329***	***.108***	***9.187***	***.002***	***1.389***	***1.123***	***1.718***
Child Age	-.077	.053	2.154	.142	.926	.835	1.026
Onset Type (EO)[a]	.243	.442	.303	.582	1.275	.537	3.031
Onset Type (P)[a]	***-2.599***	***1.154***	***5.073***	***.024***	***.074***	***.008***	***.714***
Onset Type (EOR)[a]	-.908	.890	1.039	.308	.403	.070	2.310
F1xChild Age	-.027	.020	1.740	.187	.974	.936	1.013
F1xEO	.112	.261	.185	.667	1.119	.671	1.866
F1xP	***.894***	***.374***	***5.714***	***.017***	***2.444***	***1.175***	***5.087***
F1xEOR	***1.193***	***.354***	***11.383***	***.001***	***3.297***	***1.649***	***6.594***
F2xChild Age	.023	.025	.845	.358	1.023	.975	1.073
F2xEO	-.074	.361	.042	.839	.929	.458	1.885
F2xP	-.264	.574	.211	.646	.768	.249	2.366
F2xEOR	-.763	.486	2.468	.116	.466	.180	1.208
F3xChild Age	-.007	.014	.216	.642	.993	.966	1.022
F3xEO	.037	.121	.092	.761	1.037	.819	1.314
F3xP	.447	.250	3.197	.074	1.563	.958	2.550
F3xEOR	-.149	.244	.375	.541	.861	.534	1.390
F4xChild Age	***-.027***	***.012***	***4.803***	***.028***	***.974***	***.951***	***.997***
F4xEO	-.230	.170	1.828	.176	.795	.570	1.109
F4xP	-.224	.220	1.033	.309	.799	.519	1.231
F4xEOR	-.348	.199	3.044	.081	.706	.478	1.044
F5xChild Age	.022	.015	2.169	.141	1.022	.993	1.053
F5xEO	.017	.137	.015	.903	1.017	.778	1.329
F5xP	.682	.352	3.750	.053	1.978	.992	3.944
F5xEOR	***.553***	***.262***	***4.452***	***.035***	***1.739***	***1.040***	***2.906***
F6xChild Age	.017	.013	1.801	.180	1.017	.992	1.042
F6xEO	***-.348***	***.117***	***8.863***	***.003***	***.706***	***.561***	***.888***
F6xP	***-.766***	***.281***	***7.428***	***.006***	***.465***	***.268***	***.806***
F6xEOR	***-.412***	***.198***	***4.357***	***.037***	***.662***	***.450***	***.975***

[a] For the Onset Type variable, EO = Early Onset, P = Plateau, and EOR = Delay plus Regression; The fourth onset type (i.e., Regression) is the reference variable

For Tables 11 through 15, the Wald statistic is the test statistic for the individual regression coefficients. This statistic is used to determine whether a predictor in a model is making a statistically significant prediction of the frequency of current treatment use. The odds ratio (i.e., *Exp(B)*) is calculated by the exponent constant (approximately 2.72) raised to the power of *B*. An odds ratio of 1 would indicate that there is no relationship between the predictor and the dependent variable. If an odds ratio is greater than 1, this is suggesting that with every one unit increase for the predictor (e.g., moving up one point on the Likert scale or, for example, from "Strongly Disagree" to "Disagree"), the odds that there is a higher number of overall current treatments used *increases*. If an odds ratio is less than 1, for every unit increase in the predictor variable, the odds that there is a higher number of current treatments used *decreases*. The confidence intervals suggest that, with repeated trials, 95% of the confidence intervals would include the true Poisson regression coefficient. The interpretation of the categorical moderator (i.e., onset type) is similar. For this variable, the Regression onset type is the comparison or reference variable. For example, suppose there is a significant interaction between a predictor variable (i.e., one of the six factors) and a level of the categorical moderator (e.g., Early Onset type). In this example, an odds ratio over 1 would be interpreted to mean that for every unit increase in the predictor variable, the odds of an increase in frequency of treatments is that much higher *when* the onset type is Early Onset compared to the reference variable (i.e., Regression type). All interpretations of significant interactions with onset type is in comparison to this reference variable.

The results from Table 11 indicate the presence of several significant interactions and main effects with *overall* current frequency of treatments. Main effects for predictors

are presented first followed by the significant interactions. The intercept results indicate that, on average, for a participant who strongly disagrees with all causal factors (i.e., for all six factors there is a score of 0), who has a child with ASD who is 13.56 years old (mean age), and who does not have the Regression onset type (reference variable), the number of predicted current treatments is 1.245.

There were two significant main effects with Environmental Risk Factors [OR = 1.351, $p < .01$]) and the Metaphysical factor [OR = 1.389, $p < .01$]). This suggests that when the Environmental Risk Factor scale increases by one point on the Likert scale, the number of overall current treatments increases by 35%. Likewise, for every increase in the Metaphysical scale, the frequency of overall current treatments increases by 39%. It is important to remember that the two items from the Metaphysical factor (i.e., Chance or bad luck, Will of God) had an inverse relationship. Therefore, when considering the current findings, an increase or decrease in this scale is referring to an increase or decrease in either the Will of God item *or* the chance item.

For the Personal Attributions factor, there was a significant interaction with the Plateau (OR = 2.444, $p <.05$) and Delay plus Regression (OR = 3.297, $p < .01$) onset types. This means that for every categorical increase on the Personal Attributions factor scale (e.g., from "Strongly Disagree" to "Disagree") the number of current overall treatments increased by 144% when the onset type is Plateau; similarly, there was a 230% increase in the number of overall current treatments when the onset type is Delay plus Regression.

The In Utero/Birth Stress factor had a significant interaction with child age (OR = .974, $p < .05$), indicating that for every categorical increase on the In Utero/Birth Stress

scale, the frequency of overall current treatments decreased by 3% when taking into account current child age. The Structural Composition factor interacted significantly with Delay plus Regression type (OR = 1.739) suggesting that for every categorical increase on this factor scale, the number of overall treatments increased by 73% when the onset type is Delay plus Regression. For the Metaphysical factor, the interactions with all three onset types were significant (i.e., Early Onset [OR = .706, $p < .01$], Plateau [OR = .465, $p < .01$], and Delay plus Regression [OR = .662, $p < .05$]); these significant values suggest for every categorical increase in the Metaphysical scale, there was a 29%, 53%, and 34% decrease, respectively, in overall frequency of current treatments when considering the various onset types.

Poisson regression: predicting frequencies within each evidence-based category. In addition to the overall frequency of current treatments, four additional Poisson regressions were run to test the potential of the independent variables, moderators, and interactions to predict frequency of current treatment use *within each of the four developed evidence-based treatment categories* (i.e., Established, Emerging, Not Established, Not Yet Reviewed). The results of these analyses may be seen in Tables 12 through 15.

Table 12.

Poisson Regression with Predictors, Moderators, and Interactions on Frequency of Current Treatment Use within the Established Category

						95% CI for *Exp(B)*	
Predictor	*B*	S.E.	Wald	*p*	*Exp(B)*	Lower	Upper
(Intercept)	-.278	.638	.190	.663	.757	.217	2.644
F1 (Personal Attributions)	-.675	.405	2.777	.096	.509	.230	1.126
F2 (Parental Risk Factors)	-.216	.568	.145	.703	.805	.265	2.451
F3 (Env Risk Factors)	.278	.175	2.533	.112	1.321	.938	1.861

Predictor	*B*	S.E.	Wald	*p*	*Exp(B)*	95% CI for *Exp(B)* Lower	Upper
F4 (In Utero/Birth Stress)	.276	.276	1.001	.317	1.318	.768	2.262
F5 (Structural Comp)	.066	.197	.113	.737	1.068	.726	1.571
F6 (Metaphysical)	.215	.174	1.525	.217	1.240	.881	1.743
Child Age	.042	.080	.271	.603	1.042	.891	1.219
Onset Type (EO)[a]	.243	.700	.120	.729	1.275	.323	5.032
Onset Type (P)[a]	-1.650	1.730	.909	.340	.192	.006	5.704
Onset Type (EOR)[a]	1.396	1.661	.706	.401	4.037	.156	10.669
F1xChild Age	-.038	.033	1.369	.242	.962	.903	1.026
F1xEO	.418	.418	1.000	.317	1.519	.669	3.447
F1xP	***1.359***	***.682***	***3.970***	***.046***	***3.891***	***1.022***	***14.804***
F1xEOR	***1.708***	***.658***	***6.743***	***.009***	***5.520***	***1.520***	***20.041***
F2xChild Age	-.028	.041	.451	.502	.973	.897	1.055
F2xEO	.028	.585	.002	.962	1.028	.327	3.235
F2xP	.480	.957	.251	.616	1.615	.247	10.542
F2xEOR	-.002	.797	.000	.998	.998	.209	4.760
F3xChild Age	.013	.024	.315	.574	1.014	.967	1.062
F3xEO	-.067	.194	.121	.728	.935	.639	1.367
F3xP	-.313	.416	.566	.452	.731	.323	1.654
F3xEOR	***-1.142***	***.495***	***5.319***	***.021***	***.319***	***.121***	***.842***
F4xChild Age	-.025	.020	1.541	.214	.975	.937	1.015
F4xEO	-.260	.284	.841	.359	.771	.442	1.344
F4xP	.128	.397	.105	.746	1.137	.523	2.474
F4xEOR	-.031	.362	.007	.933	.970	.477	1.972
F5xChild Age	-.008	.023	.104	.747	.993	.948	1.039
F5xEO	-.027	.214	.016	.898	.973	.640	1.479
F5xP	.269	.515	.273	.601	1.308	.477	3.586
F5xEOR	-.363	.494	.542	.462	.695	.264	1.830
F6xChild Age	-.014	.020	.445	.505	.987	.948	1.027
F6xEO	-.295	.188	2.475	.116	.744	.515	1.075
F6xP	-1.052	.560	3.525	.060	.349	.117	1.047
F6xEOR	-.232	.348	.444	.505	.793	.401	1.569

Within the Established treatment category, there were no significant main effects. Results indicated there was a significant interaction between the Personal Attributions factor and Plateau onset type (OR = 3.891, $p < .05$), meaning that for every categorical increase in this factor scale, the frequency of a currently used treatments within the Established category increased 289% when the onset type was Plateau compared to the

reference variable (i.e., Regression onset type). Similarly, a significant interaction was found between the Personal Attributions Factors and Delay plus Regression (OR = 5.520, $p < .01$) suggesting that for every unit increase in the scale for this factor, there is a 452% increase in frequency of current treatment use in this category when the onset type was Delay plus Regression. An additional significant interaction was found with Environmental Risk Factors and Delay plus Regression (OR = .319, $p < .05$) which suggests that for every unit increase on this factor, there is a 68% decrease in the number of treatments used within this category when considering this onset type.

Table 13.

Poisson Regression with Predictors, Moderators, and Interactions on Frequency of Current Treatment Use within the Emerging Category

						95% CI for *Exp(B)*	
Predictor	*B*	S.E.	Wald	*P*	*Exp(B)*	Lower	Upper
(Intercept)	-1.743	1.032	2.856	.091	.175	.023	1.321
F1 (Personal Attributions)	-.359	.706	.258	.611	.698	.175	2.787
F2 (Parental Risk Factors)	.628	.939	.447	.504	1.874	.298	11.803
F3 (Env Risk Factors)	.107	.283	.142	.706	1.112	.639	1.936
F4 (In Utero/Birth Stress)	-.153	.476	.104	.748	.858	.337	2.181
F5 (Structural Comp)	.188	.331	.324	.569	1.207	.632	2.307
F6 (Metaphysical)	.328	.300	1.198	.274	1.388	.771	2.499
Child Age	-.061	.160	.143	.706	.941	.687	1.289
Onset Type (EO)[a]	.367	1.194	.095	.758	1.444	.139	15.002
Onset Type (P)[a]	-5.688	10.400	.299	.584	.003	.000	.576
Onset Type (EOR)[a]	-.921	2.496	.136	.712	.398	.003	53.052
F1xChild Age	-.061	.067	.816	.366	.941	.825	1.074
F1xEO	.383	.740	.268	.605	1.467	.344	6.258
F1xP	2.523	1.536	2.699	.100	12.461	.615	252.677
F1xEOR	.811	1.127	.518	.472	2.250	.247	20.468
F2xChild Age	.107	.0766	1.966	.161	1.113	.958	1.294
F2xEO	-1.216	.9919	1.503	.220	.296	.042	2.071
F2xP	-9.087	6.175	2.166	.141	.000	.000	20.391
F2xEOR	-1.900	1.458	1.699	.192	.150	.009	2.603
F3xChild Age	-.020	.047	.189	.664	.980	.894	1.074
F3xEO	.032	.340	.009	.925	1.032	.530	2.011

Predictor	*B*	S.E.	Wald	*P*	*Exp(B)*	95% CI for *Exp(B)* Lower	Upper
F3xP	4.326	2.843	2.315	.128	75.629	.287	19904.632
F3xEOR	-.024	.685	.001	.972	.976	.255	3.736
F4xChild Age	.015	.039	.144	.704	1.015	.940	1.096
F4xEO	.224	.499	.202	.653	1.251	.471	3.324
F4xP	.446	.888	.252	.615	1.562	.274	8.901
F4xEOR	-.234	.626	.140	.709	.791	.232	2.700
F5xChild Age	-.003	.047	.004	.953	.997	.909	1.093
F5xEO	-.286	.373	.589	.443	.751	.361	1.561
F5xP	-.080	2.864	.001	.978	.923	.003	252.903
F5xEOR	.492	.750	.429	.512	1.635	.376	7.113
F6xChild Age	.031	.041	.568	.451	1.031	.952	1.118
F6xEO	-.285	.336	.720	.396	.752	.389	1.453
F6xP	-1.728	1.046	2.733	.098	.178	.023	1.378
F6xEOR	.026	.545	.002	.963	1.026	.352	2.986

Within the Emerging evidence-base treatment category, there were no significant interactions or main effects found.

Table 14.

Poisson Regression with Predictors, Moderators, and Interactions on Frequency of Current Treatment Use within the Not Established Category

Predictor	*B*	S.E.	Wald	*P*	*Exp(B)*	95% CI for *Exp(B)* Lower	Upper
(Intercept)	-1.165	.799	2.128	.145	.312	.065	1.492
F1 (Personal Attributions)	-.372	.508	.537	.464	.689	.255	1.866
F2 (Parental Risk Factors)	-.358	.742	.232	.630	.699	.164	2.991
F3 (Env Risk Factors)	.367	.225	2.669	.102	1.443	.929	2.241
F4 (In Utero/Birth Stress)	***.637***	***.315***	***4.095***	***.043***	***1.891***	***1.020***	***3.504***
F5 (Structural Comp)	-.292	.266	1.211	.271	.746	.443	1.257
F6 (Metaphysical)	***.545***	***.222***	***6.011***	***.014***	***1.725***	***1.116***	***2.666***
Child Age	-.187	.111	2.836	.092	.829	.667	1.031
Onset Type (EO)[a]	-.191	.885	.047	.829	.826	.146	4.675
Onset Type (P)[a]	-5.414	2.900	3.485	.062	.004	.000	1.310
Onset Type (EOR)[a]	-1.543	1.708	.816	.366	.214	.008	6.079
F1xChild Age	-.033	.043	.573	.449	.968	.889	1.053
F1xEO	-.012	.522	.001	.981	.988	.355	2.747
F1xP	.534	.699	.585	.445	1.706	.434	6.714
F1xEOR	1.189	.680	3.053	.081	3.282	.865	12.451

Predictor	*B*	S.E.	Wald	*P*	*Exp(B)*	95% CI for *Exp(B)* Lower	Upper
F2xChild Age	.020	.050	.164	.685	1.020	.926	1.124
F2xEO	-.064	.766	.007	.933	.938	.209	4.204
F2xP	.532	.988	.290	.590	1.703	.246	11.798
F2xEOR	-.062	.977	.004	.949	.940	.138	6.382
F3xChild Age	.016	.028	.308	.579	1.016	.961	1.073
F3xEO	.196	.246	.631	.427	1.216	.750	1.971
F3xP	.649	.515	1.590	.207	1.914	.698	5.248
F3xEOR	.353	.490	.517	.472	1.423	.544	3.719
F4xChild Age	-.033	.025	1.717	.190	.968	.921	1.016
F4xEO	-.587	.326	3.254	.071	.556	.294	1.052
F4xP	***-.881***	***.403***	***4.775***	***.029***	***.415***	***.188***	***.913***
F4xEOR	-.608	.384	2.504	.114	.544	.256	1.156
F5xChild Age	.025	.032	.612	.434	1.025	.963	1.091
F5xEO	.430	.286	2.266	.132	1.538	.878	2.692
F5xP	***2.027***	***.833***	***5.917***	***.015***	***7.592***	***1.483***	***38.878***
F5xEOR	.631	.539	1.370	.242	1.879	.653	5.403
F6xChild Age	***.056***	***.025***	***5.048***	***.025***	***1.058***	***1.007***	***1.111***
F6xEO	***-.518***	***.238***	***4.750***	***.029***	***.596***	***.374***	***.949***
F6xP	-.851	.463	3.377	.066	.427	.172	1.058
F6xEOR	***-.936***	***.435***	***4.634***	***.031***	***.392***	***.167***	***.920***

Within the Not Established category of treatments, there were two significant main effects and five significant interactions. The main effect for the In Utero/Birth Stress factor was significant (OR = 1.891, $p < .05$) indicating that for every categorical increase, there was an 89% increase in the number of treatments used within this category. There was also a significant main effect for the Metaphysical factor (OR = 1.725, $p < .05$) suggesting that for each categorical increase on this scale, there was a 73% increase in the number of treatments used within this category.

For the In Utero/Birth Stress factor, there was a significant interaction with Plateau onset type (OR = .415, $p < .05$) indicating a 58% decrease in frequency of treatments used within this category with every categorical increase in the factor scale when the onset type is Plateau compared to the reference variable. The interaction

between the Structural Composition factor and Plateau onset type was also significant (OR = 7.592, $p < .05$) suggesting that for every categorical increase on this factor scale, there is a 659% increase in the number of treatments used within this category when the onset type is Plateau. For the Metaphysical Factor, there were three significant interactions with child age (OR = 1.058, $p <. 05$), Early Onset type (OR = .956, $p < .05$), and Delay plus Regression type (OR = .392, $p < .05$). The interaction with child age suggests that for incremental increases in this factor scale, frequency of current treatments within this category increases 6% when considering child age. Also, for every increase in this factor score, treatment use decreases 4% or 60% in respect to onset type.

Table 15.

Poisson Regression with Predictors, Moderators, and Interactions on Frequency of Current Treatment Use within the Not Reviewed Category

						95% CI for *Exp(B)*	
Predictor	*B*	S.E.	Wald	*p*	*Exp(B)*	Lower	Upper
(Intercept)	-2.502	.988	6.415	.011	.082	.012	.568
F1 (Personal Attributions)	.082	.571	.021	.886	1.086	.354	3.326
F2 (Parental Risk Factors)	-.633	.762	.688	.407	.531	.119	2.367
F3 (Env Risk Factors)	***.530***	***.250***	***4.508***	***.034***	***1.699***	***1.042***	***2.772***
F4 (In Utero/Birth Stress)	.326	.368	.784	.376	1.385	.673	2.851
F5 (Structural Comp)	.231	.291	.631	.427	1.260	.712	2.231
F6 (Metaphysical)	.368	.246	2.245	.134	1.445	.893	2.339
Child Age	-.156	.109	2.041	.153	.856	.691	1.060
Onset Type (EO)[a]	1.061	1.063	.996	.318	2.888	.360	23.197
Onset Type (P)[a]	-.271	2.360	.013	.909	.763	.007	77.780
Onset Type (EOR)[a]	-2.013	1.786	1.270	.260	.134	.004	4.430
F1xChild Age	-.001	.037	.001	.972	.999	.928	1.074
F1xEO	-.201	.583	.119	.730	.818	.261	2.563
F1xP	.989	.960	1.060	.303	2.687	.409	17.645
F1xEOR	1.089	.734	2.204	.138	2.971	.705	12.514
F2xChild Age	.050	.047	1.152	.283	1.051	.960	1.152
F2xEO	.488	.780	.391	.532	1.629	.353	7.511
F2xP	-2.480	2.399	1.069	.301	.084	.001	9.219
F2xEOR	-1.338	1.052	1.617	.204	.262	.033	2.063

Predictor	*B*	S.E.	Wald	*p*	*Exp(B)*	95% CI for *Exp(B)* Lower	Upper
F3xChild Age	-.052	.029	3.158	.076	.950	.897	1.005
F3xEO	-.139	.273	.259	.610	.870	.510	1.486
F3xP	1.182	.789	2.244	.134	3.260	.695	15.303
F3xEOR	.002	.475	.000	.997	1.002	.395	2.543
F4xChild Age	-.035	.024	2.271	.132	.965	.922	1.011
F4xEO	-.163	.378	.186	.667	.850	.405	1.782
F4xP	-.102	.508	.040	.842	.903	.334	2.446
F4xEOR	-.635	.418	2.305	.129	.530	.234	1.203
F5xChild Age	***.074***	***.030***	***6.341***	***.012***	***1.077***	***1.017***	***1.141***
F5xEO	-.154	.311	.247	.620	.857	.466	1.575
F5xP	-.303	.836	.132	.717	.738	.143	3.803
F5xEOR	***1.376***	***.505***	***7.437***	***.006***	***3.960***	***1.473***	***10.649***
F6xChild Age	.010	.026	.144	.704	1.010	.961	1.061
F6xEO	-.349	.261	1.798	.180	.705	.423	1.175
F6xP	-.612	.587	1.088	.297	.542	.171	1.713
F6xEOR	-.594	.410	2.100	.147	.552	.247	1.233

Within the Not Reviewed category of treatments, there was one significant main effect and two significant interactions. Specifically, there was a significant main effect for Environmental Risk Factors (OR = 1.699, $p < .05$), suggesting that for every categorical increase in this scale, there is a 70% increase in the number of treatments used in this category.

For the Structural Composition factor, there was a significant interaction with child age (OR = 1.077, $p < .05$) and the Delay plus Regression onset type (OR = 3.960, $p < .01$). The significant child age interaction suggests that for every categorical increase in the Structural Composition factor scale, there was an 8% increase in number of current treatments used in this category when child age is considered. The interaction with onset type indicates there is a 296% increase in the number of treatments used within this category when the onset type is Delay plus Regression compared to the reference variable (i.e., Regression onset type).

Chapter V

Discussion

The current study contributed to the literature in several ways. First, the current study conducted a principal components analysis (PCA) on the Cause subscale of the IPQ-RA. This study improved on what was previously known about the factor structure of the Cause subscale of this measure (which was conducted by Al Anbar et al., 2010) by capitalizing upon a larger sample size, additional subscale items, and using a geographically representative (i.e., from 12 sites across the U.S.) sample of parents whose children had rigorously confirmed ASD diagnoses (i.e., were participants in the Simons Simplex Collection). Secondly, the current study employed a focus group technique (i.e., NGT) to generate a categorization scheme of ASD treatments from professionals with expertise in ASD, which provides a procedural method to categorize treatments compared to what is found in the literature. Furthermore, the current study provides information about how parental perceptions of cause of their child's ASD contributes to current parental treatment decisions, which were analyzed in several ways that are unique to the current study. Finally, two important moderating factors (e.g., current child age, onset type) were examined as potentially influencing the relationship between parental perceptions of cause and frequency of current treatment use. Taken together, these study aims and their outcomes are unique to the current study and signify new contributions to the literature in the area of parent treatment selection for children with ASD.

Demographics of the Sample

The current study collected data from participants across the United States. Overall, the sample was a majority Caucasian (85.3%), 1.5% African American, 2.5% Asian American, 6.7% more than one race, and 3.7% other. According to the U.S. Census Bureau (2010), the current sample represents an overrepresentation of Caucasian participants (2010 census listed at 72.4%) and those who selected more than one race (2.9%), and an underrepresentation of all other racial categories (i.e., 2010 Census indicates races represented in the U.S. include African American = 12.6%, Asian = 4.8%, Other = 6.2%). The largest discrepancy between the current study demographics and the U.S. Census Bureau (2010) statistics was the underrepresentation of African American participants. The disproportionally lower representation of African American participants, especially in genetic research studies pertaining to ASD, has been widely recognized as an issue in the literature (Hilton et al., 2010). Several posited reasons for this disparity include that African American children are less likely than Caucasian children to receive an ASD diagnosis (Mandell et al., 2009) and there may be a potentially higher number of existing barriers (i.e., societal and research procedure limitations) that prevent African American participation (Hilton et al., 2010). Regardless, differences in actual incidence or clinical presentation of ASD has not been found by race or ethnicity (Chaidez, Hansen, & Hertz-Picciotto, 2012; Dyches, Wilder, Sudweeks, Obiakor, & Algozzine, 2004) and more efforts need to be made to produce research that is reflective of the general population demographics (Nowell, Brewton, Allain, & Mire, 2015).

Furthermore, 72.6% of participants in the current study were from households earning an annual income of $66,000 thousand or more; the most current statistics

indicate the median household income is $56,516 (U.S. Census Bureau, 2015). In fact, 42.9% of the current participants in the current study reported household incomes of over $100,000, representing a very high socioeconomic status (SES). Additionally, 39% of current participants indicated they had earned a Bachelor's Degree and 34.7% of responders reported having earned an advanced degree (e.g., Master's, PhD, MD, DDS, OD, etc.). Current statistics of adults in the U.S. who are 25 years or older indicate that 32.5% of the population has earned a Bachelor's Degree with 12% of the population having earned an advanced degree (U.S. Census Bureau, 2015). Therefore, current participants are from a higher SES and have a higher education level than the overall US population. For these reasons, study findings may not be generalizable to all parents of children with ASD in the U.S. *It should be noted that for most the Discussion section, the term "participants" was changed to "parents" to promote readability and understanding*.

Research Question One: What is the factor structure of the Cause subscale of the IPQ-RA?

The current study utilized a PCA (i.e., type of EFA) to investigate the factor structure of the 21-items of the IPQ-RA. Before conducting the PCA for the current study, descriptive statistics examined parental perceptions of cause of their child's ASD. For a majority of the causal items, parents tended to "Disagree" or "Strongly Disagree" with a particular cause contributing to their child's ASD, which aligns with previous research and may indicate that parents are more certain about what does not cause ASD (Goin-Kochel et al., 2014). However, for the "Genetics" item, 86.2% of parents indicated "Agree" or "Strongly Agree" with genetic factors being a causal factor to their child's

ASD. This is not surprising given that the original study (i.e., SSC) was primarily a genetics research study; thus, parents who believed genetics played a factor in the development of their child's ASD may have been more drawn to participate. Additionally, 48.2% of parents and 69% of parents, respectively, reported "Agree" or "Strongly Agree" with the items "Environmental Pollution" and "My Child's Brain Structure" contributing to the development of their child's ASD. Several studies in the literature have documented potential causal links between development of ASD and genetic factors (Muhle et al., 2004; Ozonoff et al., 2011; Sanders et al., 2011), environmental pollution (Windham et al., 2006), and brain structure (Aylward et al., 2002; Courchesne et al., 2001; Nickl-Jockschat et al., 2012). Indeed, genetic factors and variations in brain structure are more researched and solidified potential causes of ASD, while causal contributions of environmental pollution (e.g., hazardous air pollutants) to the development of ASD is more controversial with several studies reporting contradictory findings (Abdullah et al., 2012; Kalkbrenner et al., 2010).

Findings of the current study are largely consistent with previous research on parental perceptions of cause of ASD. Specifically, many parents reported believing their child's ASD may be linked to genetic factors, environmental pollution (Dardennes et al., 2011; Goin-Kochel et al., 2014; Mercer et al., 2006), and brain structure/abnormalities (Dardennes et al., 2011; Goin-Kochel et al., 2014). Interestingly, the current study did not find that a high number of parents strongly agreed with toxins found in vaccinations, with only 22.7% of parents endorsing this as a potential cause; this finding is contradictory to findings in previous research, which found 40% (Mercer et al., 2006) and 41.8% (Goin-Kochel et al., 2014) of parents attributed toxins in vaccinations as a cause. Notably, these

studies occurred several years ago, and research regarding the vaccine-autism link has accumulated in the last several years. Current findings suggesting less frequent parental beliefs that vaccinations may be a causal factor is hopeful, given that this causal connection has been widely discredited in numerous studies (Heron & Golding, 2004; Stehr-Green et al., 2003; Madsen et al., 2003; Taylor et al., 2014). It should also be noted that it is very likely that parents in the current study attribute the cause of their child's ASD to numerous causal factors, which would be consistent with previous literature (Mercer et al., 2006); however, it was beyond the scope of the current study to investigate this notion.

Results from the PCA investigating the factor structure of the IPQ-RA Cause subscale produced six factors which were named: Personal Attributions (Factor 1), Parental Risk Factors (Factor 2), Environmental Risk Factors (Factor 3), Utero/Birth Stress (Factor 4), Structural Composition (Factor 5), and Metaphysical (Factor 6). The first three factors were comprised of five items each, while the last three factors contained two items each.

As described earlier in this paper, Al-Anbar et al. (2010) modified the original IPQ-R (Moss-Morris et al., 2002) to be applicable to parents of children with ASD (i.e., IPQ-RA). The Cause subscale of the IPQ-RA used in the Al Anbar et al. (2010) study contained 18 items and these authors ran a PCA of the cause items. Based on their results, the 18 Cause subscale items resulted in three factors (as opposed to six factors found in the current study), and these three factors accounted for 61% of the total variance. Although the authors did not provide *precisely* what items loaded onto their three factors, the study indicated their factors were labeled Personal Attributions (e.g., item examples

included my own behavior, alcohol intake, smoking behavior, parental age, and accident or injury), Environmental Causes (e.g., pollution, diet, germ or virus, deterioration of the immune system, past poor medical care), and Heredity Attribution (e.g., heredity, chance or bad luck) (Al Anbar et al., 2010). The current study included the 18 items from the Al Anbar et al. (2010) study, with some additional items included by Mire et al. (2015) to reflect other potential parental attributions of cause. Given that the IPQ-RA cause subscale used in the current study varies in several ways from the cause subscale used in the Al Anbar et al. (2010) study, results are not directly comparable. However, the PCA's from the current study and that of Al Anbar et al. (2010) did produce a similar total variance (i.e., both approximately 61%). Furthermore, the current study produced some factors that were conceptually similar to those found in the Al Anbar et al. (2010) study (e.g., Environmental Risk Factors), although the addition of the new items likely altered the number and composition of factors overall.

Research Question Two: Consensus Coding Group, Categorization of Treatments, and Treatment Data

Consensus coding group. The current study utilized a specific type of focus group called a nominal group technique (NGT) to develop the categorization scheme by which to categorize the 116 treatments in the current study. Current literature has used a variety of ways to categorize ASD treatments such as by hypothesized mechanism (e.g., immune modulation, behavioral; Levy et al., 2003), by treatment feature (e.g., interpersonal relationships, skill-based, cognitive; Hess, Morrier, Heflin, & Ivey, 2008), and by evidence-base (e.g., established, emerging, not established; National Autism Center, 2009; Odom et al., 2010; Young et al., 2010). The methods of categorizing ASD

treatments vary across studies and often studies do not provide the procedures by which treatment categories are developed. The current study sought to utilize a methodical approach to determine how treatments would be categorized through use of a NGT.

Results from the NGT focus group used in the current study produced twelve different categorization schemes for ASD treatments (e.g., by outcome-focus, by treatment effects, by evidence-base), which are listed previously. These numerous generated categorization schemes are reflective of the current literature, which utilizes a variety of means to categorize ASD treatments; also, current results highlighted the difficult nature and variability of the process of categorizing ASD treatments. Ultimately, the focus group employed in the current study decided that categorizing treatments by *evidence-base* was the most preferable method. This result was not surprising given that this categorization method is a commonly used framework in empirical literature, and the focus group participants consisted of professionals and graduate students involved with families with ASD in a clinical and/or research capacity. While classifying and scrutinizing treatments based on their level of scientific evidence has been a widespread approach for professionals over the past several decades, it should be noted that this is only one component of determining treatment effectiveness. Furthermore, it is also important to consider the differences between what a professional considers important regarding treatments versus what a parent might consider important, which is discussed further in the Limitations section. However, for the current study, treatments were categorized by level of evidence-base, which is described further in the following section.

Categorization of treatments and treatment data. As previously outlined, treatments were categorized by evidence-base using available large-scale systematic

reviews. The current study collected data on 116 different treatments, which was modeled after several previous studies (Green, 2007; Mire, 2012). However, just as prevalence rates for ASD increase, so do the number of treatments (Bowker, D'Angelo, Hicks, & Wells, 2011); thus, the treatments used in the current study should not be considered an exhaustive list of all available ASD treatments. Common categories that delineate varying levels of evidence were used in the current study and were labeled Established, Emerging, and Not Established. Furthermore, for the purposes of the current study, an additional category labeled Not Yet Reviewed was created to capture those treatments that were included in the current study, but were not mentioned in any of the systematic reviews suggesting they have not yet been studied.

Interestingly, the current study found that 33 of the treatments were not reported as currently used by *any* of the parents. It may be that parents of children with ASD within the current study did not find these treatments useful or perhaps were not exposed to these treatments by provider resources. These treatments were removed from consideration in the current study and included largely treatments that would have belonged to the Not Established or Not Yet Reviewed categories (e.g., Dolphin Therapy, Holding Therapy, Watsu; See Appendix I). Furthermore, 42 of the treatments which were reported as currently used by some parents fell into the Not Yet Reviewed category. These finding highlight the complicated task parents are faced with when choosing treatments for their children with ASD. Not only are there a plethora of ASD treatment options available, but parents have varying resources (e.g., time, money) and knowledge about treatments (Stephenson, Carter, & Kemp, 2012). Also, parents get their information about different treatments from numerous sources (e.g., books, websites, autism

newsletters, testimonials, word of mouth; Mackintosh et al., 2005; Matson, Adams, Williams, & Rieske, 2013; Miller et al., 2012), many of which do not involve consultation with a trained professional who may (or may not) promote use of evidence-based treatments. Furthermore, some sources of information that provide treatment information to parents (e.g., autism websites) may provide inaccurate information about the evidence-base of ASD treatments (Stephenson et al., 2012). More concerning is the finding that several advocacy websites have been found to provide intentionally misleading information utilizing references to "experts", non peer-reviewed citations, and data generated by the organization (Di Pietro, Whiteley, Mizgalewicz, & Illes, 2013). All of these factors complicate the already time consuming and overwhelming task of parents selecting treatment(s) for their children with ASD, resulting in frequent parental selection of scientifically unsubstantiated treatments.

At the time of data collection, parents endorsed using an average of approximately three current treatments; however, 79 parents (24.2%) endorsed no current treatment use. This finding of no current treatment use is consistent with a study by Bowker et al., (2011), which found that 23% of their sample (n = 970 parents of children with ASD) reported *never* using a treatment. Although this study is only reporting *current* treatment use, the finding is worth considering. In the current study, frequency of current treatment use ranged from zero to 17 different treatments, with a majority of parents (72.5%) reporting using between one and nine current treatments. It has been found that parents of children with ASD use multiple treatments simultaneously, with one study reporting use of four to six current treatments (Goin-Kochel et al., 2007) and another

study reporting between approximately two and seven current treatments (Regehr & Feldman, 2009).

When looking at frequency of current treatment use within the evidence-based categories, findings varied. For instance, 25.8% of parents reported using one treatment within the Established treatment category with approximately the same amount of parents (i.e., 24.2%) reporting using at least one treatment from the Not Yet Reviewed category. It is likely that parents were currently using several treatments across numerous evidence-based categories, and the number of treatments within each category likely impacted these frequencies (e.g., Established category has 10 treatments while Not Yet Reviewed category has 42 treatments). However, the aims of the current study were to examine prediction of overall frequency of current treatment use and treatment use by category.

Research Question Three: Does parental perceptions of cause of ASD predict frequency of treatment use overall and by evidence-base category?

The third aim of the current study was to investigate whether parental perceptions of the cause of their child's ASD predicted frequency of current treatment use *overall* and *within the four created evidence-base categories*. While some previous research has examined the link between parental perceptions of cause and parent selected treatments for ASD (Al Anbar et al., 2010; Dardennes et al., 2011; both of these studies used the same sample), more research is needed as results have implications for treatment and public-health related decisions (e.g., whether or not to vaccinate a child; Yudelll et al., 2013). Also, treatments that have strong evidence of improving outcomes for individuals with ASD (e.g., Applied Behavior Analysis [ABA]), receive more state and federal program support and are more likely to be reimbursed by insurance companies

(Dillenburger, McKerr, & Jordan, 2014), thus lowering the already increased cost of caring for an individual on the spectrum. The subsequent paragraphs provide a detailed discussion of the findings for the third research question. Findings are discussed in the following order: (1) discussion of parental perceptions of cause predicting *overall* number of treatments, (2) discussion of parental perceptions of cause predicting frequency of current treatment use *within the four evidence-base categories*, (3) discussion of the moderators' (i.e., onset type and child age) influence on parental perceptions of cause and *overall* frequency of current treatment use, and (4) discussion of moderators' influence on parental perceptions of cause and frequency of current treatment use *within the four evidence-base categories*. The creation of evidence-based categories as dependent variables and the inclusion of the current moderators to examine their influence on the aforementioned relationships are unique to the current study. Due to the large number of examined relationships and the exploratory nature of this research question, only significant findings are discussed.

Does parental perceptions of cause predict *overall* frequency of treatments?

When examining *overall* frequency of treatment use, results indicated that the more parents believed that environmental risk factors and/or factors beyond their control (i.e., chance/bad luck or will of God) contributed to their child's ASD, the *more* overall treatments they currently endorsed using (i.e., by 35% or 39%, respectively). These perceptions represent parental perceptions of cause that are largely external in nature (i.e., not intrinsic to the child). The environmental risk factors represent several parental perceptions about cause of ASD including germ or virus, diet or eating habits, pollution, deterioration of child's immunity, and toxins found in vaccines. The factors beyond

parental control contain two external components (i.e., chance/bad luck, will of God). Past research has found that some parents of children with ASD may attribute environmental factors (e.g., vaccines, pollution; Goin-Kochel, et al., 2014; Mercer et al., 2006; Selkirk et al., 2009) and factors beyond control (Goin-Kochel et al., 2014) to the cause of their child's ASD.

However, with regard to how these perceptions impact frequency of treatment choices, the research is sparse. It is plausible that parents who believe that external elements contributed to their child's ASD may seek a variety of treatment options in an effort to improve their child's functioning. Relatedly, it is also possible that parents who believe their child's ASD is caused by an external force, may believe that they are able to counteract (or improve) their child's condition by manipulating the environment or introducing external elements (e.g., medication, vitamins, behavioral interventions) that impacts the child. Notably, it is important to point out that there may be numerous treatments that parents have used over their child's *lifetime* that is not captured in the current study, which focused on *current* treatments.

Does parental perceptions of cause predict frequency of treatments use *within evidence-based categories*? Within the Not Established category, results suggested that when parents agree more with causes related to in utero/birth stress and/or factors beyond their control (i.e., chance/bad luck or will of God), the frequency of currently using treatments within the Not Established category *increased* by 89% and 73%, respectively. It is possible that parents with these causal perceptions were willing to try a larger variety of different treatments and were not focused on which treatments were evidence-based when making their selection; specifically, treatments within the Not

Established category are mainly vitamins (e.g., Vitamin B6), medications that are not FDA approved (e.g., Haldol), and some other treatments that, while not evidence-based, are considered common homeopathic treatments (e.g., acupuncture, casein/gluten-free diets). However, it should also be noted that some of the treatments in this category have been deemed not helpful for individuals for ASD and may also be harmful (e.g., facilitated communication, chelation; APA, 1994; Brent, 2013).

Another result suggested that parents are more likely to use more treatments in the Not Yet Reviewed category (i.e., by 70%) when they agree more that environmental risk factors contributed to the development of their child's ASD. This result suggests that parents who perceive environmental influences to be an etiological factor may be likely to use treatments that are scientifically unsubstantiated. Al Anbar et al. (2010) found that parents endorsing external causes (such as environmental factors and factors beyond control) increased use of metabolic treatments (e.g., special diets) and vitamin supplements. This finding is aligned with the findings of the current study, as the Not Established and Not Yet Reviewed categories contain several of these treatments (e.g., casein free/gluten free diets, Vitamin A, Vitamin C). Furthermore, research reports a high frequency of use of these and other CAM or nonconventional treatments (e.g., chelation therapy) with parents of children with ASD, which have little to no empirical support (Green et al., 2006; Levy et al., 2003, Wong & Smith, 2006). More specifically, studies have indicated that 21% to 84% of parents have tried various CAM treatments (Senel, 2010), and a different study indicated 30% of their sample was receiving *only* non-empirically supported treatments (Regehr & Feldman, 2009). Considering this information, to the author's knowledge, the current study's investigation of parental

perceptions of cause predicting frequency of current treatment by evidence-base is a unique contribution to the literature.

Does child age and symptom onset play a role in parental perceptions of cause predicting *overall* frequency of treatments? The current study also examined onset type and current child age as potentially influencing the relationship between parents' perceptions of ASD cause and frequency of *overall* current treatment use. To the author's knowledge, this investigation constitutes an initial exploration into these relationships.

As previously mentioned, the ASD symptom onset patterns used in the current study were first proposed by Shumway et al., (2011) and provide a way to distinguish patterns for ASD symptom emergence. These patterns are important as they may help researchers understand how parents explain their child's ASD, which influences treatment choices they make (Goin-Kochel et al., 2014). The four onset types are Early Onset (i.e., symptoms of ASD existent in the first year with no later loss of skills), Delay plus Regression (i.e., early symptoms existed as well as a loss of skills), Plateau (i.e., no existing symptoms in the first year and no loss of skills), and Regression (i.e., no early symptoms of ASD, but a later loss in skills) (Shumway et al., 2011).

When looking at *overall* frequency of current treatment use, patterns of symptom onset influenced parental perceptions of cause and treatment choices in several instances. Specifically, when parents endorsed a greater belief that their child's ASD was due to something specific to the parent (i.e., life stress, parental behaviors/decisions, mental attitude/negative views, family worries about ASD, parental emotional state), there was a 144% *increase* in overall treatments when the parent did not see symptoms within the

first year and the child experienced no loss of skills (i.e., Plateau onset); furthermore, there was a 230% *increase* in overall treatment use when the parent observed a regression in skills (i.e., Delay plus Regression onset). Similarly, when parents agreed more with causal factors related to genetics and child brain structure, there was a 73% *increase* in current overall treatment use when the parent observed a regression (i.e., Delay plus Regression onset). It is possible that parents who observe a regression in their child's skills are more likely to pursue a greater number of treatments, especially if parents' attribute the cause of their child's ASD to be external to the child and attributable to themselves.

Interestingly, the more parents perceived their child's ASD to be due to factors beyond their control (i.e., chance/bad luck or will of God), there was a *decrease* in frequency of overall current treatments when: (a) ASD symptoms were observed in the first year (i.e., Early Onset; 29% decrease), (b) no symptoms observed in the first year and no loss of skills (i.e., Plateau onset; 53% decrease), and (c) when a loss of skills was observed (i.e., Delay plus Regression onset; 34% decrease). Overall, these results indicated that the number of treatments currently used was *lower* for parents who agreed with causal factors that were beyond their control (i.e., chance/bad luck or will of God) across all symptom pattern types. In other words, it seems that the more parents agreed that the cause of their child's ASD is beyond their control, the fewer treatments they pursued at a single point, when onset type is taken into account. One researcher has suggested that parents who perceive their child's ASD to be fate or God's will were less stressed overall (Mickelson, Wroble, & Helgeson, 1999). Perhaps belief in this cause provides a buffer to parental stress that results in less pursuit of treatments; however,

exploring parental stress' influence on this relationship was beyond the scope of the current study.

Previous research involving onset type is sparse and has focused on the potential connection between onset type and parental beliefs about cause of their child's ASD (Goin-Kochel et al., 2014; Goin-Kochel & Myers, 2005). Researchers indicated that parents who observed their child experiencing a congenital onset (i.e., no regression, similar to Early Onset type) were more likely to believe in a prebirth cause of their child's ASD (e.g., genetics, brain abnormality, in utero stress); inversely, those parents who reported a developmental regression were more likely to believe in an external or environmental etiology (Goin-Kochel et al., 2014; Goin-Kochel & Myers, 2005). In other words, parents who have children who experience developmental regressions, which represent an observed loss in already developed skills, may attribute the cause of that child's ASD to an environmental or external cause. However, parents who have children who have demonstrated symptoms from early in life (i.e., did not show developmental regression) may be more likely to attribute those symptoms to a prebirth cause (as symptoms have always been observed or observed from a young age) (Goin-Kochel et al., 2014; Goin-Kochel & Myers, 2005). Results from the current study were mixed. For example, parents who agreed more with both external (i.e., causes specific to the parent) and internal (genetics, brain structure) causes demonstrated an increase in current frequency of overall treatments when a regression or loss of skills was observed (i.e., Delay plus Regression onset). However, it is plausible that when parents observe a regression in their child's skills, they have a tendency to pursue more treatments regardless of whether the perception of cause is internal or external.

Also, child age was found to influence the relationship between parents who attributed the cause of their child's ASD to utero/birth stress and current overall treatment use. Specifically, the more parents attributed the cause of their child's ASD to in utero/birth stress, the *fewer* treatments they reported currently using (by 3%) when considering child age. Although statistically significant, this was a small finding that may have little practical significance. Research suggests that younger children may receive more treatments than older children (Green, 2007) and the types of treatments younger children receive may differ characteristically from older children (Kochel et al., 2007; Mire et al., 2015). The current finding suggested that differences in child age may result in a slight *decrease* in current overall frequency of treatment use the more parent's attribute in utero/birth stress as being the causal factor of their child's ASD.

Does child age and symptom onset play a role in parental perceptions of cause predicting frequency of treatments *within evidence-based categories?* Within the Established category of treatments, symptom onset type was found to influence the relationship between parental beliefs about cause and selection of evidence-based treatments. Specifically, when parents agreed more that their child's ASD was due to something specific to the parent, parents used *more* treatments when they observed no symptoms within first year and no loss of skills (i.e., Plataea onset; 289% increase) and when they observed a loss in skills (i.e., Delay plus Regression onset; 452% increase). Furthermore, increased belief in environmental risk factors was related to a *decrease* (by 68%) in the frequency of using Established treatments when a regression was observed (i.e., Delay plus Regression onset). These are interesting findings that suggest differences in frequency use of evidence-based treatments (increase versus decrease) when parental

perceptions of cause are different but both external to the child with ASD (i.e., environmental and due to the parent) and when the parent observes a loss in skills (i.e., Delay plus Regression onset). This suggests that the more parents perceive the cause of their child's ASD to be due to something specific to the parent and they have observed a loss in skills, they are more likely to *increase* current number of evidence-based treatments. However, the more parents perceive that the cause of their child's ASD is due to environmental factors combined with an observed loss in skills, parents actually use *fewer* evidence-based treatments. It is plausible that these latter parents are pursuing treatments within other categories (e.g., CAM treatments), as previously discussed.

Within the Not Established category, symptom onset type was also found to influence the relationship between parental beliefs about cause and frequency of treatment use. For parents who agreed more with causes related to in utero/birth stress and observed no symptoms during first year and no loss in skills (i.e., Plateau onset type), the number of treatments used within this category *decreased* by 58%. Furthermore, when parents believe casual factors are attributed to genetics and brain abnormalities and observe no symptoms during first year and no loss in skills (i.e., Plateau onset type), parents' current use of Not Established treatment types *increased* 659%. Previous research has demonstrated that parental beliefs in a genetic cause led to an increase in use of metabolic treatments (e.g., vitamin supplements; Al Anbar et al., 2010); however, the current study did not delineate between specific treatment types, as previously mentioned. Furthermore, the onset type (i.e., Plateau) seems to make a unique contribution to this relationship, but potential reasons for this requires further study. Finally, when parents agreed more with causes that were beyond their control (i.e.,

chance/bad luck or will of God), current treatment use within this category *decreased* 4% when symptoms of ASD existent in the first year with no later loss of skills (i.e., Early Onset type) and 60% when a regression was observed (i.e., Delay plus Regression onset type). These results are similar to previous results found in the current study and suggest that parents who hold these beliefs may be less likely to seek treatment.

When considering treatments in the Not Yet Reviewed category, as parents agreed more with the causes of genetics and brain structure, the *more* likely they were to use treatments within this category, when considering child age (8% *increase* in treatments). As with the previous finding that included child age, the influence is small and may not constitute a practically significant finding.

The current study made several unique contributions to the literature including identifying six factors of parents' causal attributions to ASD development; exploring a treatment categorization scheme created by professionals; and investigating how parental perceptions of the cause of their child's ASD predict the number of treatments parents pursue and whether or not these selected treatments have scientific support. Despite these strengths, the current study is not without several limitations, which are discussed in detail in the following paragraphs.

Limitations

Demographics, study design, and variables. As previously discussed, parents for the current study are a majority Caucasian, high SES, and have a higher level of education than would be found in the general population (U.S. Census Bureau, 2010; 2015). Therefore, results may not be generalizable to families of more diverse backgrounds. Furthermore, a majority of the participants in the current study were

mothers (88%). Although research suggests that the mother in a family system is typically more involved with the care and choosing treatments for their children with ASD (Benson et al., 2008; Dardas & Ahmad, 2014), the results from the current study may not be as generalizable to families where fathers are the primary providers.

One study design limitation is that treatment data were collected using a detailed web-based survey. Although the data were checked for missing entries before analysis, parents may have quickly answered the questions and not carefully chosen answers. Furthermore, the current study was not able to compare treatment information with other sources (e.g., school records, medical records), which limits the studies ability to confirm treatments children with ASD were receiving. Additionally, the number of treatments presented to parents in the survey (i.e., 116 different treatments) may have been overwhelming leading to the possibility that parents may have skipped over some treatments and/or only provided information about treatments they could recall while taking the survey.

Another potential study design limitation is that the Likert scale data for the Cause subscale were treated as continuous. The method of treating Likert scale data as continuous, as opposed to ordinal, is a controversial and ongoing issue in the literature. Researchers who believe Likert scale data should be treated as ordinal persist that the psychometric distance between Likert scale categories is not equal (Cummins & Gullone, 2000) and treating these variables as continuous may create non-normal distributions that, in turn, distort interpretation of results (Leung, 2011). However, Likert scale data are often treated as continuous, especially in psychological research studies, and several researchers purport that this method is acceptable, especially when there are five or more

points on a scale and the Likert items are used to create scales (Brown, 2011; Maurer & Pierce, 1998).

Concerning the predictor variables in the current study, there were limitations with regard to the distributions of the cause items and the Cause subscale factors. Specifically, the Cause subscale items were generally skewed via histogram visual inspections. This is not necessarily surprising, as it demonstrates that parents had certain perceptions about the causal attributions of their child's ASD. For instance, item 2 ("Genetics") was highly skewed left, meaning that a majority of parents selected "Strongly Agree" or "Agree" to this being a potential cause; as discussed previously, the original research study (i.e., SSC) focused on genetics, so it is likely parents would agree with that potential cause. Furthermore, the Cause subscale factors were generally non-normally distributed (according to a visual inspection of the histograms), which may result in distortion of the results. However, the skewness and kurtosis statistics for the individual cause items and the factors were within acceptable ranges. Regardless, future research using these data might use different estimation techniques (e.g., Maximum Likelihood Parameter [MLR]), in which standard errors are more robust to non-normality.

Principal components analysis. Regarding the principle components analysis (PCA) used in the current study, three of the six factors only had two items. In an article discussing best practices for factor analysis, Costello and Osborne (2005) report that factors with less than three items may be unstable and weak. However, for the three factors with two items each, several indices were robust (e.g., communalities above .4;

high factor loadings) and the decision was made to include these factors as predictors in the current study.

Another potential limitation was that the current PCA solution produced several cross-loading items, which may indicate that these items were associated with more than one factor. Ideally, researchers aim for a simple solution in which each item only loads onto one factor. Different rotation methods were attempted, but the current solution (using orthogonal rotation) produced the cleanest model with the least amount of cross-loadings. Some researchers have indicated that whether or not to retain cross-loading items (e.g., Matsunga, 2015) is largely a judgment call and items reinforced by the literature may support the decision to leave items in the model. For the current study, all items had support as potential causes of ASD; therefore, it was decided that all items would remain in the model. Future research using this data might explore whether eliminating items produces a cleaner factor structure. Lastly, for the Metaphysical factor, the inter-item correlation was low (i.e., .11), which suggests these items are not strongly related to one another and this factor may not be viable. However, this factor was considered relevant to understanding the relationships explored in the current study and was retained.

Consensus coding focus group. One potential limitation relevant to the focus group used in the current study was that the focus group participants represented a convenience sample that all work within the same geographical area. More specifically, only professionals and students who were known to work with individuals and families with ASD were invited to participate in the focus group and all of the focus group participants work within the same area. However, while this may be a problematic

sampling technique in some situations, this is a common technique with focus groups (Nagle & Williams, 2013).

What may be considered a more problematic limitation regarding the focus group relates to the validity of the results. That is, the focus group resulted in determination of how *professionals* may categorize ASD treatments, which may be characteristically different from how *parents* may categorize treatments. In other words, this study's results ultimately depict how *parent* perceptions of the cause of their child's ASD may predict frequency of treatment use within categories conceptualized by *professionals*. For the current study, the focus group outcome resulted in treatments being categorized according to their evidence-base. While this is a common method of distinguishing treatments among professionals, this may not be how parents perceive or think about available treatments. As discussed previously, parents typically select a variety of treatments for their children, which often contain a combination of validated and non-validated treatments (Green et al., 2006). It is also reported that a variety of factors affect treatment selection including accessibility, cost (Goin-Kochel et al., 2009), professional referrals (Green, 2007), time and effort to promote success of treatment (Mackintosh et al., 2012), parenting style, media influences (Levy & Hyman, 2005), child age (Goin-Kochel et al., 2007), and parental perceptions (Goin-Kochel et al., 2009; Mandell & Novak, 2005). It is likley that some parents of children with ASD are not aware of the strength of evidence of a current treatment and, thus, may not consider it when selecting treatments. In fact, a current study by Deyro, Simon, and Guay (2016) investigated parents' perceptions of the evidence-base of ASD treatments. Results indicated that parents have a variable understanding of whether or not a treatment is evidence-based;

furthermore, whether or not a treatment was rated as having scientific support did not determine for parents whether treatments were efficacious (Deyro et al., 2016).

Treatment data. There were also several limitations to consider regarding the treatment data. One limitation was that the current study used the Green (2007) study to develop a list of individual treatments on which data were collected. Although this study presented a comprehensive list of 116 different types of ASD treatments, it does not include *all* of the ASD treatments available. Therefore, there are likely several treatments being used by parents, which were not captured in the current study. Also, parents may be utilizing a variety of different "treatments" with their children that they may not consider "treatments" (e.g., vitamins, regular massages); thus, some parents may not have reported everything their child is receiving.

Furthermore, treatments were categorized into evidence-based categories using systematic reviews. As mentioned previously, there is no uniform definition of evidence-base treatment and definition of this construct varied slightly among utilized reviews. Additionally, 42 of the treatments used in the current study had not been reviewed by any of the systematic studies. This may have resulted from these 42 treatments never being studied in the literature and/or the fact that the most recent review was dated from 2015; therefore, more recent studies may have provided empirical evidence of any of these 42 treatments, which were not represented in the current study. Lastly, data on treatment use was based solely on retrospective participant report. However, the current study only focused on treatments children with ASD were *currently* using making it more likely that parent report for these treatments was accurate.

Poisson regressions. One limitation for the regression analyses was that the data was overdispersed, which indicates that the variance was found to be larger than the mean. One potential reason for this is the excessive number of parents who reported using *no* current treatments, resulting in an excessive number of zeroes in the data. While this is a common phenomena in behavioral studies (Hua et al., 2014), it may inhibit the model's ability to make accurate predictions. Use of alternative methods (e.g., zero-inflated models) have been suggested in the literature, although these methods are new and do not have as much research support. Suggestions for exploration of the current data using alternative methods is discussed in the following section.

Future Directions

In the previous section, limitations to the current study were reviewed and possible approaches to addressing these limitations were discussed. There are also several future directions for this research area, which are proposed in the following paragraphs.

Further exploration of the treatment and cause data. The current study focused on treatments that were *currently* used by parents in order to gain insight into how *current* parent perceptions of cause of their child's ASD may predict *current* treatment choices. However, the PeP study also collected data on whether a parent had *ever* used a treatment across the child's lifespan. Thus, future research could explore the use of *ever* having used a treatment. Also, as discussed previously, literature has made several connections with child age and types of treatments that parents select (Green et al., 2006; Goin-Kochel et al., 2007; Mire et al., 2015). While the current study investigated current child age as a potential moderating variable between the relationship

of perceptions of cause and treatment selection, additional research could investigate child age (or grouped age categories) as predictor variables.

Furthermore, additional exploration of the descriptive treatment data may provide further insight into the factors that may influence parent treatment decisions. For instance, future research studies could investigate potential reasons *why* 79 parents in the current study did not endorse *any* current treatments; also, what were the characteristics and potential reasons why some parents only used established treatments and others only used treatments from the Not Yet Reviewed category?

Furthermore, future research could also incorporate the open text portion of the Cause subscale of the IPQ-RA. Specifically, parents were able to enter (in an open text field) what causes they perceived to be most relevant (in rank order). Parents were instructed to enter causes that were included in the 21-item scale and/or causes that were not queried. Exploration of this data could provide further information about what causes parents considered *most* important and could help researchers understand additional causes that were not listed as future items to add to the scale.

Expand the potential of focus groups. For the current study, a Nominal Group Technique (NGT) was used to conduct a focus group of how professionals and students involved in ASD research and clinical work would categorize treatments. As discussed previously, categorizing ASD treatments is complicated due to the sheer number of treatments and the various ways in which treatments can be grouped together. These complications are reflected in the literature as various studies and reviews have categorized treatments differently, resulting in lack of consensus in the literature that may impact generalizability and replication efforts. Future research could utilize the NGT

model, perhaps across various regional areas of the United States, to further explore how professionals categorize ASD treatments. Not only would these efforts work towards a more unified understanding of categorizing treatments, but differences in categorization across various regions/cultural areas may yield interesting findings. Furthermore, the current focus group produced twelve different ways that ASD treatments could be categorized. Future research in this area could explore whether these other categorization schemes (e.g., categorizing by theoretical basis, demand/resources) are viable ways to group treatments. Also, the current study only utilized professionals in the field of psychology as participants in the focus group. However, utilizing other professionals (e.g., medical, occupational therapists) could elucidate how professionals across disciplines understand treatments and would choose to group them. Lastly, focus groups including parents of children with ASD would be even more inclusive and help professionals to understand directly from parents what factors are important in regards to ASD treatments.

Utilize additional analytic methods. The current study utilized Poisson regressions as a means of conducting analyses using "count data" (i.e., frequency counts of treatments). As previously mentioned, the current study data were overdispersed, which may be due to the excessive number of zeroes in the data (i.e., parents who reported not using *any* current treatments). Although not as well studied as Poisson regressions, new analytic methods, such as the zero-inflated Poisson regression (Atkins et al., 2013; Hua et al., 2014) may be an alternative to the methods used in the current study. Furthermore, the current study utilized a PCA to examine the underlying factor structure of the Cause subscale of the IPQ-RA. These factors were used as predictor variables to

examine the relationship of parent use of current treatments overall and within developed evidence-based categories. Future research might focus on conducting a factor analysis of the parent reported treatment data. If found to produce viable factors, additional analyses could investigate whether parent perceptions of cause predicts frequency of treatment use within, essentially, parent created categories, which could improve the validity of the results.

Additionally, the current study examined prediction of frequency of treatment use overall and conducted separate analyses for each evidence-based treatment category. However, this does not elucidate possible *interactions* between the categories of treatment use. Previous research has supported that parents of children with ASD use various treatments simultaneously (Goin-Kochel et al., 2007; Regehr & Feldman, 2009), a notion that was consistent with current study results. It is likely that parents were using treatments across multiple evidence-based categories, which would also be consistent with the literature that parents use both empirically and non-empirically supported treatments (Green et al., 2006; Levy et al., 2003; Regehr & Feldman, 2009; Senel, 2010). Future research could develop an outcome variable that would demonstrate these interactions.

Call to Professionals: Importance of Collaboration

Parents are largely responsible for undertaking the extremely stressful task of navigating through the sea of treatment options available for their children with ASD (Green et al., 2006). This stressor is compounded by several other stressors placed on parents of children with autism, such as *lifetime* cost of care (Baio, 2012; Buescher, Cidav, Knapp, & Mandell, 2014; Cidav, Marcus, & Mandell, 2012; Gurney et al., 2006)

and coping with co-occurring child physical and mental health problems (Krakowiak et al., 2008; Lecavalier et al., 2006; Leyfer et al., 2006; Molloy & Manning-Courtney, 2003). One qualitative study investigating the impact of having a child with ASD indicated several parents reported exhaustion/sleep problems, struggling with schools to retain services, marital strain, impact on career trajectory, and a negative impact on social life (e.g., loss of friends, restrictions on going out) (Myers, Mackintosh, & Goin-Kochel, 2009).

Overall, several studies have demonstrated that these cumulative stressors weigh on parents with children with ASD resulting in poorer health (i.e., mental and physical; Allik, Larsson, & Smedje, 2006; Bromley, Hare, Davison, & Emerson, 2004) and generally a lower quality of life in comparison to parents of typically developing children (Mugno, Ruta, D'Arrigno & Mazzone, 2007). Thus, these stressors are severely impactful and constitute a heavy burden on parents who care for children with ASD. What can we as professionals and researchers do to lighten the load? How can we have a positive impact on the lives of parents and their children with ASD?

One way may be to help guide parents towards more evidence-based treatment options that are demonstrated to have a positive impact on their child's functioning. That starts with gaining a deeper understanding of *why* parents choose treatments. While there are several reasons why a parent may choose a particular treatment (or set of treatments), understanding parental perceptions (e.g., such as cause) or cognitions that drive treatment selection behaviors may be one avenue to understanding this complex and interactive process. Furthermore, it may be that clinicians should provide adjunctive treatment that focuses on treatment for the parent. This might include providing parents with

information about support networks (i.e., parent support groups) and resources about what it means to parent a kid with ASD.

Many types of professionals work with parents and children with ASD including psychologists, medical professionals, therapists in various domains (e.g., occupational, speech, behavioral), school personnel, and social workers. While research has shown that parents receive information about treatments from multiple sources (e.g., internet, media influences, advocacy groups; Di Pietro et al., 2013; Levy & Hyman, 2005; Wong & Smith, 2006), parents often consult with various professionals to gather information about treatment options. In fact, a very recent study found that 48.5% of parents in their sample were influenced most by professional referrals in deciding treatment options for their child with ASD in comparison to other sources (e.g., general media, other parents, autism organizations) (Deyro et al., 2016). This is a hopeful finding, but also demonstrates how important it is for various professionals who work with these families to promote similar information when it comes to evidence-based treatments. Professionals need to work together to promote effective and empirically supported treatments, but also professionals need to work with parents to discover what is driving their decisions and *why*. As previously discussed, the current study is working towards answering *why* parents are selecting certain treatments.

Knowing this information could help various professionals approach treatment planning with parents in a more meaningful way; more specifically, professionals may be able to engage in more targeted psychoeducation with parents and may be able to influence parental perceptions about cause to align with more evidence-based treatment

options. It is imperative that we work together to have a meaningful and positive impact on the lives of these families and their children.

Chapter VI

Conclusion

The current study utilized a large sample of participants who have children with ASD to address several research questions. The overall main goal of the study was to investigate participant perceptions of cause of their child's ASD and how these perceptions predict current treatment choices. In order to achieve this goal, several steps were necessary, each making a unique contribution to the literature.

First, the current study conducted a factor analysis on the Cause subscale of the IPQ-RA. While a factor analysis of this subscale had previously been conducted by Al Anbar et al. (2010), the current study conducted a second factor analysis using a larger sample size, confirmed ASD diagnoses, and with additional questions added to the scale (i.e., 21 items from 18 items). Findings produced a six-factor solution, which constituted 61.38% of the overall variance. The percentage of explained variance was consistent with previous research.

Second, the current study aimed to address the complexity of separating ASD treatments into categories by conducting a specific type of focus group called a Nominal Group Technique (NGT). Focus group members consisted of nine professionals and students working with children and families with ASD in a research and/or clinical capacity. Results from the focus group produced 12 different ways to group ASD treatments together. Focus group members voted on each of the 12 methods and the final ranking revealed that grouping treatments by evidence-base was the most desirable method. Once this was established, ASD treatments used in the current study were categorized by their level of evidence using large-scale systematic reviews. Interestingly,

33 of the treatments used in the current study were not being currently used by *any* of the participants, which highlights the sheer number of ASD treatments available and suggests that participants may not find some treatments useful. Furthermore, 79 participants in the current study reported not using *any* current treatments, which is consistent with previous research. Three categories depicting varying levels of evidence-base were established (i.e., Established, Emerging, Not Established) and an additional category of Not Yet Reviewed was created to represent the treatments being used by participants that have not been studied in the literature. These categories were then used as outcome variables in the following research questions.

Lastly, five Poisson regressions were run to investigate whether participant perceptions of cause (represented by the six cause subscale factors) predicted overall frequency of treatment use and frequency of treatment use within the four aforementioned evidence-base categories. Current child age and regression onset type (i.e., Early Onset, Plateau, Delay plus Regression, and Regression) were investigated as potential moderators. When looking at overall frequency, belief in external factors of cause (i.e., environmental risk factors and metaphysical factors) *increased* number of current treatments. Furthermore, participant beliefs in utero/birth stress and metaphysical causal factors *increased* frequency of treatment use within the Not Established category, while belief in environmental risk factors *increased* use of treatments within the Not Yet Reviewed category. Onset type seemed to moderate the relationship between participant beliefs of cause and frequency of treatment use overall and within the evidence-based categories in several instances, suggesting this may be an important factor in determining frequency of treatment use. While current child age yielded a few significant findings, the

impact was low and may have little practical significance. To the author's knowledge this is the first study to investigate these specific relationships and represents an exploration into how these variables may influence the relationship between participant beliefs in cause of ASD and treatment choices.

Limitations were related mainly to use of a homogenous population and non-normal distributions of the Cause subscale items and factors. Other limitations regarding the factor analysis (e.g., cross-loading items), the focus group (e.g., convenience sample, validity issues), and the Poisson regression analyses (e.g., overdispersion) were also discussed. Several potential future directions for this research area include continuing to explore the rich treatment data that was gathered in the current study, conducting additional focus groups with a larger variety of stakeholders (e.g., medical professionals, parents), and use of additional analytic techniques. For instance, further exploration of the descriptive treatment data could investigate potential reasons *why* a large number of participants in the current study did not endorse *any* current treatments. It would also be worthwhile to delineate specific participant characteristics and their choice of treatments within the various evidence-based categories.

Choosing from the plethora of available treatment options for children with ASD is an arduous task for many parents. Oftentimes, parents are barraged with information about treatments from a variety of sources and end up selecting numerous treatments for their children with a varying level of empirical evidence. Professionals who work with children with ASD and their families should collaborate and strive to understand the factors that drive parental treatment selection. Through understanding, professionals can approach the task of treatment planning with parents in a more informed manner and

promote treatments that will have a positive and meaningful impact in the functioning of children with ASD.

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Appendix A

Illness Perception Questionnaire – Revised for Autism Cause Subscale

Causes of Your Child's Autism Spectrum Disorder (ASD)

We are interested in what you consider as likely contributing factors to your child's autism spectrum disorder (ASD). There is no correct answer to this question. What interests us most is your own perspective on the factors that may have caused your child's ASD rather than what others, including a physician or other professional may have suggested. Below is a list of some parents' opinions. Please indicate to what extent you agree or disagree with these causes by checking the appropriate box.

	Possible Causes	Strongly disagree	Disagree	Neither disagree nor agree	Agree	Strongly Agree
1	General life stress	☐	☐	☐	☐	☐
2	Genetics	☐	☐	☐	☐	☐
3	A germ or virus	☐	☐	☐	☐	☐
4	Diet or eating habits	☐	☐	☐	☐	☐
5	Chance or bad luck	☐	☐	☐	☐	☐
6	Poor medical care in the past	☐	☐	☐	☐	☐
7	Environmental pollution	☐	☐	☐	☐	☐
8	My own behavior or decisions	☐	☐	☐	☐	☐
9	In utero stress or accident	☐	☐	☐	☐	☐
10	Mental attitude/negative views	☐	☐	☐	☐	☐
11	Family worries about ASD	☐	☐	☐	☐	☐
12	Will of God	☐	☐	☐	☐	☐
13	My own emotional state (e.g., depression, anxiety)	☐	☐	☐	☐	☐
14	My or my partner's age	☐	☐	☐	☐	☐
15	My own alcohol consumption	☐	☐	☐	☐	☐
16	My own tobacco consumption	☐	☐	☐	☐	☐
17	Accident or injury	☐	☐	☐	☐	☐
18	My child's brain structure	☐	☐	☐	☐	☐
19	Deterioration of my child's immunity	☐	☐	☐	☐	☐
20	Toxins found in vaccines/immunizations	☐	☐	☐	☐	☐
21	Stress at birth	☐	☐	☐	☐	☐

On the lines below, please rank order the three most important causal factors you believe underlie your child's ASD. You may use reasons from the table above or add any other opinions.

For me, the most likely contributing factors are:

1. _____
2. _____
3. _____

Appendix B

Parent Reported Treatment Use Across Age

Treatment Use

We are interested in understanding more about what types of treatments are utilized by parents of children with autism spectrum disorder (ASD). Below, you will find a list of treatments that some parents have used or tried for their children. Please indicate which, if any, of the following you have ever tried by checking the box next to the treatment.

If you select a treatment, another box will appear asking you to indicate how old your child was when you tried or used the treatment. For example, if your child started taking Abilify when s/he was 8 1/2 and stopped when s/he was 9, you would choose: 8 years old and 9 years old. Please choose the AGES, even if it was only for a short time, like days or weeks.

Abilify/aripiprazole	Cylert/pemoline	Inderal/propranolol
Acupuncture	Dance therapy	Infant massage
Adderall	Depakote/valproic acid/divalproex sodium	Institute for human potential (doman-delacto patterning)
Antihistamine (sleep aid)	Dexedrine/dextroamphetamine	Integrated movement therapy
Applied behavior analysis (Private)	Diflucan/fluconazole	Interactive metronome
Applied behavior analysis (School)	Dilantin/phenytoin	Intravenous immunoglobulin
Aromatherapy	Discrete trial training (Lovaas)	Irlen lenses
Atavin/lorazepam	DMG (dimethylglycine)	Joint action routines
Auditory integration training	Dolphin therapy	LEAP
Augmentive and Alternative Communication	Eden program	L-Glutamine
Azrin 24-h toilet training	Electro-Aversive Therapy	Lindamood bell
Baudhuin preschool	Extended breast-feeding	Lithium
Bethanechol Medication	Facilitated communication	Magnesium
Bolles Sensory Learning	Fast forward	Mega-vitamin therapy
Buspar/buspirone	Feingold diet	Melatonin
Casein-free diet	Floor time	Multisensory environments (Snoezelen)
Catapres/clonidine	Folic acid/folate	Music therapy
Chelation	Gentle teaching	Naltrexone
Clathration	Giant steps	Neural therapy
Clonopin/ clonazepam	Gluten-free diet	Neurofeedback (biofeedback)
Clozaril/clozapine	Hagashi school	Nystatin
Cognitive/behavioral	Haldol/haloperidol	Occupational Therapy

therapy		(Private)
Conductive education	Holding therapy	Occupational Therapy (School)
Craniosacral manipulations	Homeopathy	Omega-3/Fatty acids
Options	Secretin Medication	Valium/diazepam
Osteopathy	Self-injurious behavior inhibiting system (SIBIS)	Van Dijk approach
Paxil/paroxetine	Sensory integration	Vancomycin
Pentoxifylline	Social stories	Visual integration training
Pepcid	Social skills training (SST)	Visual schedules
Picture exchange communication systems (PECS)	Speech therapy (Private)	Vitamin A
Probiotics	Speech therapy (School)	Vitamin B6
Prozac/fluoxetine	Sporanox/ itraconazole	Vitamin C
Pyridoxine	TEACCH	Watsu
Rapid prompting	Tegretal/carbamazepine	Weighted vest/blanket
Reduced L-glutathione	Tenex/Intuniv/guanfacine	Xanax/alprazolam
Risperdal/risperidone	Thorazine/chlorpromazine	Yeast-free diet
Ritalin/methylphenidate	Tofranil/imipramine	Zoloft/sertraline
Rolfing	Transfer factor	Other (Open Response)
Rhythmic entertainment interventions	Vagal nerve stimulation	

Appendix C

Recruitment Email sent to Parents

Dear [Parent First Name],

You are being contacted because you have expressed interest in studies applicable to the Simons Simplex Collection (SSC) families. Here at the University of Houston, we are currently studying how family factors influence or are related to the types of treatments parents try for their children with autism spectrum disorder (ASD). This is very important research that may help us better understand the complex processes and factors that are related to navigating treatment-seeking. Ultimately, we hope that such findings may help professionals to ***better serve and support families*** of children with ASD.

If you choose to participate, you will be asked to complete an online survey that asks about your perspectives and experiences as a parent of a child with ASD. The survey platform is secure, designed specifically for this kind of research. It will take about 1 hour, 20 minutes to complete; you may finish the survey in one sitting or return to it later. All parents who complete the survey will have the option to be entered in a random drawing for one of four iPad minis that will be given away when the study period is over. More specific details about this study are provided below.

As a participant, you will use a unique eight-digit ID number (i.e., XX.XXXX.XX) to access the survey. This number is very important and allows us to protect you by keeping your survey responses separate from all identifying information.

Your unique eight-digit ID number is:

XX.XXXX.XX

Please keep this number for the duration of your participation. Do not copy and paste the ID, but hand enter it into the survey.

To participate, click on the study link below:

[Survey Link]

Or copy and paste the URL below into your internet browser:

[URL]

We are grateful for your consideration of participating in this study!

Sincerely,

Dr. Sarah Mire
ssmire@central.uh.edu
713-743-6448

Appendix D

Consent Form Included within Recruitment Email

Please read the additional information below about this study. You may wish to print this information for your records.

PROJECT TITLE: Parental Perceptions and Family Stress: Implications for Treatment-Seeking for Children with Autism Spectrum Disorder

You are being invited to take part in a research project conducted by Dr. Sarah Mire from the Department of Educational Psychology at the University of Houston.

NON-PARTICIPATION STATEMENT

Taking part in the research project is voluntary and you may refuse to take part or withdraw at any time without penalty or loss of benefits to which you are otherwise entitled. You may also refuse to answer any research-related questions that make you uncomfortable. Choosing not to participate will have no effect on your standing within the Simons Simplex Collection (SSC), the Interactive Autism Network (IAN) Research Project, or the SSC@IAN.

PURPOSE OF THE STUDY

The goal of this study is to learn more about how family factors influence or are related to the types of treatments parents try or pursue for their children with autism spectrum disorder (ASD).Based on other research, we believe that there are many complex factors that can impact how parents choose treatments, including what families know and think about the ASD diagnosis itself and how the ASD diagnosis impacts whole families, as well as the specific needs of individual children.

You are eligible for participation in this study because you are an SSC family. We have chosen to recruit from SSC families because we already know that participants have a confirmed ASD diagnosis, and because the variability among the children with ASD from the SSC is very high. That is, SSC children have a range of skills, abilities, and limitations, and we are very interested in capturing the views of parents who have children with a range of abilities.

PROCEDURES

You will be one of approximately 2,760 subjects invited to take part in this project. To participate in this study, you will be asked to complete a total of five (5) questionnaires online. No questionnaire is expected to take more than about 20 minutes, with all questionnaires expected to take about 1 hour, 20 minutes to complete. You can take them online, and you'll be able to save your progress and come back to the questionnaires later if you need to. If there are any specific questions you don't want to answer, you can skip the question. Also, you are free to stop participation at any time, including before you start answering, while you're answering, or even after you've completed the questionnaires.
The titles and topics of the five questionnaires are as follows:

1. Revised Illness Perception Questionnaire- Revised- Autism Spectrum Disorder (IPQ- RASD) parents' thoughts and beliefs about ASD and having a child with ASD
2. Parenting Stress Index-Fourth Edition-Short Form (PSI-4-SF) - for parents of children who are currently age 11 or younger

   OR

   Stress Index for Parents of Adolescents (SIPA) - for parents of children currently age 12 and older, parents' reports about stress in the parent-child relationship and general life stress
3. Family Adjustment Measure (FAM) - parents' reports about family impact associated with raising a child with special needs
4. Treatment history questionnaire - checklist of what treatments parents have ever chosen for their child, and at what age(s)
5. Demographic questionnaire - your child's age, family factors (i.e., living arrangements, family resources), ASD severity

Our study team will give away four (4) iPad Mini tablets once all the data collection is finished, and any family who has completed all the questionnaires will have a chance to win one of these in a random drawing. You will be able to choose whether you wish to enter the random drawing once you have completed the surveys. After completing all the surveys, you will have the option of clicking on a link which will allow you to enter your contact information (name, email, phone number, home address) if you would like to be eligible for the random drawing to be held once all data collection is complete. The purpose of the link being separate from the survey is to further protect your information from being identifiably linked with you or your family. Also, your contact information collected if you opt to participate in the random drawing will be stored in an electronic, password-protected document that is separate from any data identification numbers.

CONFIDENTIALITY

Every effort will be made to maintain the confidentiality of your participation in this project. Each participant will be assigned an eight-digit ID number by SSC@IAN, and this is the number that will be used to make sure all questionnaires are properly associated with the right family. The ID number will appear on all written/electronic materials and will be available only to the principal investigator. The list pairing your family's name to the SSC ID numbers will be kept separate from all research materials and will be available only to the principal investigator. We will only use your name for contacting you and will retain only your study number (not your name) once data collection is complete. We will destroy your contact information once data collection is complete. We are asking for your consent for the Simons Foundation and the SSC@IAN to share with the University of Houston the phenotype data collected during your participation in the SSC. This information will be shared using your linked research ID number and using a secure file transfer system. We are also asking for your consent to share the data we collect during this study here at the University of Houston with the Simons Foundation and the SSC@IAN in order to add the information that was collected during your participation in the SSC. Confidentiality will be maintained within legal

limits.

RISKS/DISCOMFORTS

It is expected that only minimal risk is involved in participating in this study. Some participants may experience stress when completing questionnaires about their child's symptoms, their family's functioning, or their own perceptions about ASD and care giving for a child with ASD.

Some participants may also find completion of all questionnaires to be boring or tedious at times. However, the research team hopes that completion of the questionnaires online will increase convenience for participating parents.

BENEFITS

While you will not directly benefit from participation, your participation may help investigators better understand the complex processes and factors that are related to navigating treatment seeking for children with ASD. Ultimately, we hope that such findings may help professionals to better serve and support families of children with ASD.

ALTERNATIVES

Participation in this project is voluntary and the only alternative to this project is nonparticipation.

INCENTIVES/REMUNERATION

Each family who completes all the questionnaires for this study will have the opportunity to be included in a random drawing for one of four (4) iPad Mini tablets that will be given away at the conclusion of data collection.

PUBLICATION STATEMENT

The results of this study may be published in scientific journals, professional publications, or educational presentations; however, no individual subject will be identified.

SUBJECT RIGHTS

1. I understand that informed consent is required of all persons participating in this project.
2. I have been told that I may refuse to participate or to stop my participation in this project at any time before or during the project. I may also refuse to answer any question.
3. Any risks and/or discomforts have been explained to me, as have any potential

benefits.

4. I understand the protections in place to safeguard any personally identifiable information related to my participation.
5. I understand that, if I have any questions, I may contact Dr. Sarah Mire at University of Houston at 713-743-6448
6.

Any questions regarding my rights as a research subject may be addressed to the University of Houston Committee for the Protection of Human Subjects (713-743-9204). All research projects that are carried out by Investigators at the University of Houston are governed be requirements of the University and the federal government.

Appendix E

Recruitment Email to Potential Focus Group Participants

Hello XXXX,

I am contacting you because you are involved in improving the lives of children and families affected by Autism Spectrum Disorder (ASD). My name is Christie Brewton, and I am currently a fifth year doctoral candidate in the School Psychology PhD program at the University of Houston. My dissertation study is focused on ***understanding how the perceptions of parents of children with ASD may impact their treatment choices***. Dr. Sarah Mire with the University of Houston is the faculty sponsor of this dissertation study.

As you know, there are numerous treatments for ASD (i.e., over 100) and categorizing these treatments into meaningful categories is a challenge. As part of my dissertation study, I am conducting a focus group consisting of 5-10 professionals and graduate students who are involved in ASD research, educational, and/or clinical work. The goal of the focus group is to develop a strategy for categorizing various ASD treatments into categories, which will be used in analyses for my dissertation study.

Participation in this focus group will involve a time commitment of approximately two hours on a single day, which will be spent in a single meeting at the University of Houston campus during the month of April or May 2016. Light snacks and drinks will be served during the meeting. During the focus group meeting, you will be asked to complete a brief demographic questionnaire, hear a 10-15 minute presentation and introduction to the topic (i.e., regarding treatments for ASD), and be involved in the process of developing a strategy for the categorization of treatments. I would also like to include your name and institution as an honorable mention in my final dissertation document.

If you are willing to contribute to my dissertation study in this way, please respond by email to cmbrewto@gmail.com. Also, please feel free to email me with any additional questions you may have.

Thank you for your considering this invitation, and for your commitment to improving the lives of children and families with ASD!

Christie Brewton, B.S.

Fifth Year Doctoral Candidate

University of Houston

Appendix F

Consent Form for Focus Group Participants

UNIVERSITY OF HOUSTON
CONSENT TO PARTICIPATE IN RESEARCH FOCUS GROUP

PROJECT TITLE:

You are being invited to take part as a focus group member in a research project conducted as part of a dissertation project by Christie Brewton under the supervision of Dr. Sarah Mire (dissertation chair) from the Department of Education at the University of Houston.

NON-PARTICIPATION STATEMENT

Taking part in the research project is voluntary and you may refuse to take part or withdraw at any time without penalty or loss of benefits to which you are otherwise entitled. You may also refuse to answer any research-related questions that make you uncomfortable.

PURPOSE OF THE STUDY

As part of this study, you are being asked to participate in a focus group. The purpose of the focus group will be to develop a strategy for categorizing publicly known treatments for Autism Spectrum Disorder (ASD). Focus group members will be asked to attend one group meeting lasting approximately two hours. Beyond the two-hour meeting, focus group members will not have any further time commitments.

PROCEDURES

- You will be one of approximately 8-15 subjects invited to take part in this project.
- Focus group members will be asked to attend a single two-hour meeting. No further contact will be necessary.
- Focus group members will be asked to complete a basic demographic questionnaire during the two-hour meeting (e.g., "What setting do you currently work in?"). Information from the brief demographic questionnaire will not be use in the current study's analysis, only to gather some very basic information about the members of the focus group.
- The remainder of the two-hour meeting will consist of the following: (1) listening to a brief presentation on the purpose of the meeting (i.e., to separate ASD treatments into categories), (2) independently generating ideas about how to separate ASD treatments into categories, and (4) participating in a group discussion about generated ideas.

CONFIDENTIALITY

Every effort will be made to maintain the confidentiality of your participation in this project as a focus group member. Signed consent documents will be enclosed in a locked compartment with the principle investigator and maintained for three years. After three years, this consent document will be destroyed.

RISKS/DISCOMFORTS

There are no foreseeable risks for participation in this project as a member of the focus group.

BENEFITS

There will be no immediate benefits to individual participants. However, the current study has a potential benefit to the investigators and society as it may help us understand the association between parental perceptions of cause of their child with ASD and treatment selection. Members of the focus group may learn additional information about publicly known treatments for ASD.

ALTERNATIVES

Participation in this project as a focus group member is voluntary and the only alternative to this project is non-participation.

COSTS

There are no anticipated costs for participating in this project as a member of the focus group.

INCENTIVES/REMUNERATION

Members of the focus group who will need to travel from UH affiliated institutions (e.g., Texas Children's Hospital) will have their parking on the UH campus paid for. Also, light snacks and drinks will be provided during the meeting. Finally, focus group members will be presented with the option of including their name as an honorable mention in the final dissertation document.

PUBLICATION STATEMENT

The results of this study may be published in scientific journals, professional publications, or educational presentations; however, no individual subject will be identified.

SUBJECT RIGHTS

1. I understand that informed consent is required of all persons participating in this project.

2. I have been told that I may refuse to participate or to stop my participation in this project at any time before or during the project. I may also refuse to answer any question.

3. Any risks and/or discomforts have been explained to me, as have any potential benefits.

4. I understand the protections in place to safeguard any personally identifiable information related to my participation. I understand that, if I have any questions, I may contact Christie Brewton at 281-6650-7368 or by email at cmbrewto@gmail.com. I may also contact Dr. Sarah Mire, faculty sponsor, at 713-743-6448 or by email at ssmire@uh.edu.

5. **Any questions regarding my rights as a research subject may be addressed to the University of Houston Committee for the Protection of Human Subjects (713-743-9204). All research projects that are carried out by Investigators at the University of Houston are governed be requirements of the University and the federal government**.

SIGNATURES

I have read (or have had read to me) the contents of this consent form and have been encouraged to ask questions. I have received answers to my questions to my satisfaction. I give my consent to participate in this study, and have been provided with a copy of this form for my records and in case I have questions as the research progresses.

Study Subject (print name): _____

Signature of Study Subject: _____

Date: _____

I have read this form to the subject and/or the subject has read this form. An explanation of the research was provided and questions from the subject were solicited and answered to the subject's satisfaction. In my judgment, the subject has demonstrated comprehension of the information.

Principal Investigator (print name and title): _____

Signature of Principal Investigator: _____

Date: _____

Appendix G

Brief Background Survey for Focus Group Participants

BRIEF BACKGROUND SURVEY FOR FOCUS GROUP PARTICIPANTS

Thank you for participating in this focus group! The following questions are meant to gather some brief background information on focus group participants. Please select an answer to each of the following questions.

1. Are you currently a graduate student at the University of Houston (UH)?

    - Yes
    - No

2. If you are currently a graduate student at UH, what year are you in?

    - First year
    - Second year
    - Third year
    - Fourth year
    - Fifth year or beyond
    - Not a student

3. Please indicate which setting best characterizes your current place of employment. If you currently work within more than one setting, please choose all that apply.

    - University – Graduate Student
    - University – Faculty
    - Community Mental Health
    - Hospital
    - Inpatient
    - Outpatient
    - School
    - Private Practice
    - Other _____

4. How long have you been at your current place of employment?

    - Less than one year
    - 1-2 years
    - 2-3 years
    - 3-4 years
    - Over 5 years

5. Please briefly list the previous settings for which you have worked. Include setting type and number of years in each setting (ex: Inpatient hospital – 3 years, Public school – 2 years)

_____
_____
_____
_____

6. In what capacity have you worked with children, adults, and/or families with ASD? Please select all that apply.

    - Assessment
    - Individual Therapy (including behavioral therapy)
    - Group Therapy
    - Family Therapy
    - Consultative Services
    - Research
    - Other_____
      _____

7. How many years have you been working with children, adults, and/or families with ASD?

    - Less than 1 year
    - 1-5 years
    - 5-10 years
    - 10-15 years
    - 15-20 years
    - Over 20 years

Appendix H

Meeting Agenda for Focus Group Participants

Focus Group Meeting Agenda

When: Thursday May 19th from 3:00 to 5:00

Where: University of Houston, Farish Hall Building, Room 217 (Map and Parking Directions below on next page)

3:00 – 3:15: Brief introductions, consent forms, completion of brief demographic survey

3:15 – 3:30: Brief presentation and introduction to the topic question (i.e., What is the best categorization scheme for ASD treatments?)

3:30 – 3:45: Silent generation of ideas related to topic question

3:45 – 4:00: Round table sharing of ideas related to topic question

4:00 – 4:30: Clarification and discussion of ideas related to topic question; ideas are ranked and categorization scheme is chosen.

4:30 – 5:00: ASD treatment sorting activity

5:00: End of meeting

Appendix I

ASD Treatments Removed due to Lack of Participant Endorsement

Azrin 24-h toilet training
Baudhuin preschool
Bolles Sensory Learning
Clathration
Clozaril/clozapine
Conductive education
Cylert/pemoline
Dilantin/phenytoin
Dolphin therapy
Electro-Aversive Therapy
Extended breast-feeding
Fast forward
Feingold diet
Gentle teaching
Hagashi school
Holding therapy
Infant massage
Institute for human potential (doman-delacto patterning)
Integrated movement therapy
Irlen lenses
Joint action routines
LEAP
Natural Therapy
Pentoxifylline
Rapid prompting
Reduced L-glutathione
Rythymic Entertainment Interventions
Sporanox/ itraconazole
Tofranil/imipramine
Transfer factor
Valium/diazepam
Vancomycin
Watsu

Appendix J

Treatment Categorizations by Systematic Reviews

	CMS	NSP (2009)	AHRQ	StART	CTM	NSP (2015)	NPDC
ABILIFY/ARIPIPRAZOLE			1				
ACUPUNCTURE			3				
ADDERALL							
ANTIHISTAMINE				2			
ABA_PRIV and SCHOOL	1	1				1	
AROMATHERAPY							
ATIVAN/LORAZEPAM							
AUDITORY INTEGRATION TRAINING	3	3	3			3	2
AUGMENTIVE AND ALT COMM	2	2				2	
BETHANECHOL MEDICATION							
BUSPAR/BUSPIRONE							
CASEIN-FREE DIET		3		3		3	
CATAPRES/CLONIDINE							
CHELATION				3			
CLONOPIN/CLONAZEPAM							
COGNITIVE BEHAVIORAL THERAPY	1	2	3			1	1
CRANIOSACRAL MANIPULATIONS							
DANCE THERAPY							
DEPAKOTE/VALPROIC ACID							
DEXEDRINE/DEXTROAMPHETAMINE							
DIFLUCAN/PHENYTOIN							
DICRETE TRIAL TRAINING (LOVAAS)	1	1	1		1	1	1
DMG (DIMETHYLGLYCINE)			3				
EDEN PROGRAM					3		
FACILITATED COMMUNICATION		3					
FLOOR TIME		2			3	3	
FOLIC ACID/FOLATE							
GIANT STEPS							
GLUTEN-FREE DIET		3		3		3	
HALDOL/HALOPERIDOL			3				
HOMEOPATHY							
INDERAL/PROPRANOLOL							
INTERACTIVE METRONOME							
IV_IMMUNOGLOBULIN							
L-GLUTAMINE							
LINDAMOOD BELL							

LITHIUM				3			
MAGNESIUM			3	3			
MEGA-VITAMIN THERAPY							
MELATONIN			3	3			
MULTI-SENSORY ENVIRONMENT							
MUSIC THERAPY	2	2	3			2	2
NALTROXONE				2			
NEUROFEEDBACK			3	3			
NYSTATIN							
OMEGA-3/FATTY ACIDS			3	3			
OPTIONS							
OSTEOPATHY							
PAXIL/PAROXETINE							
PEPCID							
PECS	1	2	3			2	1
PROBIOTICS							
PROZAC/FLUOXETINE			3				
PYRIDOXINE							
RISPERDAL/RISPERIDONE			1	1			
RITALIN/METHYLPHENIDATE			3	2			
ROLFING							
SECRETIN MEDICATION				2			
SELF-INJURIOUS BEHAVIOR INH SYSTEM							
SENSORY INTEGRATION		3	3			3	2
SOCIAL STORIES	1	1				1	1
SOCIAL SKILLS TRAINING	1	2	3			1	1
TEACCH			3		1		
TEGRETOL/CARBAMAZEPINE							
TENEX/INTUNIV/GUANFACINE			3				
THORAZINE/CHLORPROMAZINE							
VAGAL NERVE STIMULATION							
VANCOMYCIN							
VISUAL INTEGRATION TRAINING							
VISUAL SCHEDULES	1	1				1	1
VITAMIN A							
VITAMIN B6			3	3			
VITAMIN C							
WEIGHTED VEST/BLANKET							
XANAX/ALPRAZOLAM							
YEAST-FREE DIET							

ZOLOFT/SETRALINE							

Categories of ASD Treatments
1 = Established (*n* = 10)
2 = Emerging (*n* = 9)
3 = Not Established (*n* = 16)
4 = Not Yet Reviewed (*n* = 42); represented by an entire blank row

Appendix K

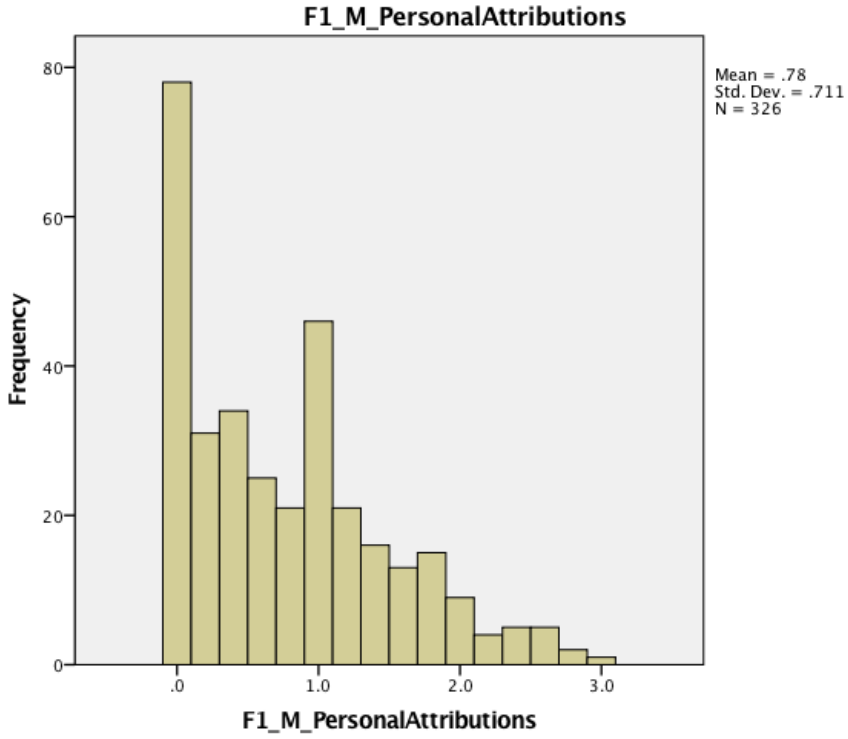
Treatments within each Evidence-Based Category

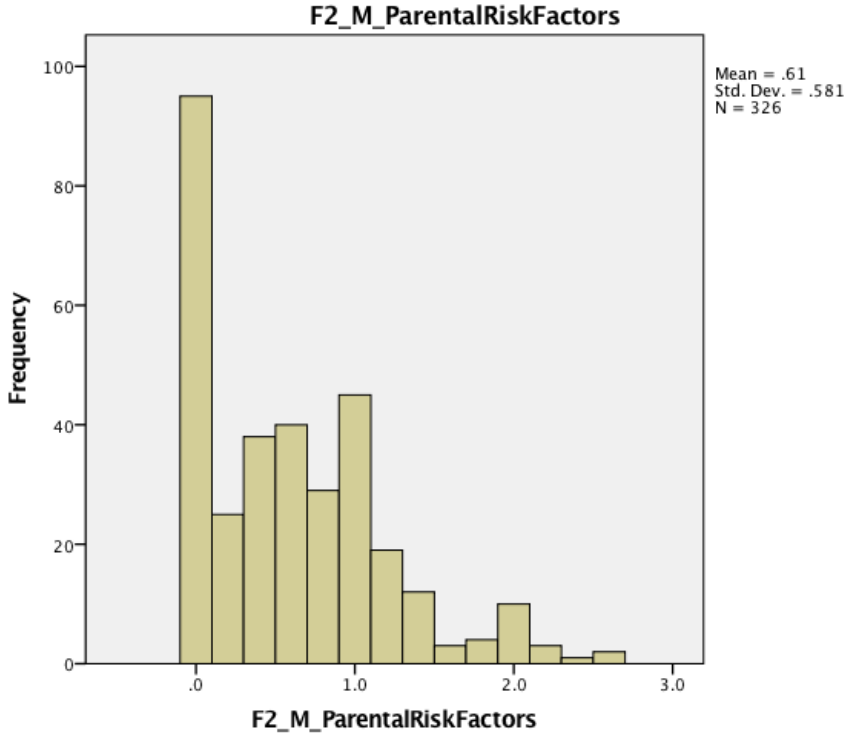
Established (n = 10)	Emerging (n = 9)	Not Established (n = 16)	Not Yet Reviewed (n = 42)	
Abilify/ Aripiprazole	Antihistamine	Acupuncture	Adderall	Multi-Sensory Environment
Applied Behavior Analysis	Auditory Integration Training	Casein-Free Diet	Aromatherapy	Nystatin
Cognitive Behavioral Therapy	Augmentive And Alt Comm	Chelation	Ativan/ Lorazepam	Options
Dicrete Trial Training (Lovaas)	Floor Time	Dmg (Dimethylglycine)	Bethanechol Medication	Osteopathy
Pecs	Music Therapy	Eden Program	Buspar/ Buspirone	Paxil/Paroxetine
Risperdal/ Risperidone	Naltroxone	Facilitated Communication	Catapres/ Clonidine	Pepcid
Social Stories	Ritalin/Methylphenidate	Gluten-Free Diet	Clonopin/ Clonazepam	Probiotics
Social Skills Training	Secretin Medication	Haldol/ Haloperidol	Craniosacral Manipulations	Pyridoxine
Teacch	Sensory Integration	Lithium	Dance Therapy	Rolfing
Visual Schedules		Magnesium	Depakote/Valproic Acid	Self-Injurious Behavior Inhibition System
		Melatonin	Dexedrine/ Dextroamphetamine	Tegretol/ Carbamazepine
		Neurofeedback	Diflucan/ Phenytoin	Thorazine/ Chlorpromazine
		Omega-3/Fatty Acids	Folic Acid/Folate	Vagal Nerve Stimulation
		Prozac/Fluoxetine	Giant Steps	Vancomycin
		Tenex/Intuniv/Guanfacine	Homeopathy	Visual Integration Training
		Vitamin B6	Inderal/ Propranolol	Vitamin A
			Interactive Metronome	Vitamin C
			Iv_Immunoglobulin	Weighted Vest/Blanket

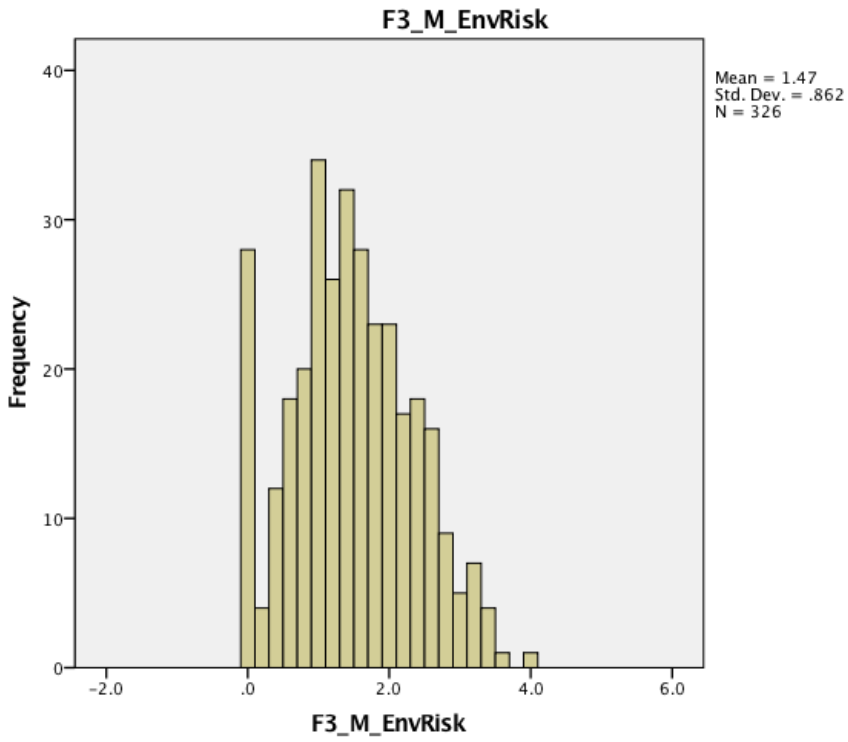
			L-Glutamine	Xanax/ Alprazolam
			Lindamood Bell	Yeast-Free Diet
			Mega-Vitamin Therapy	Zoloft/Setraline

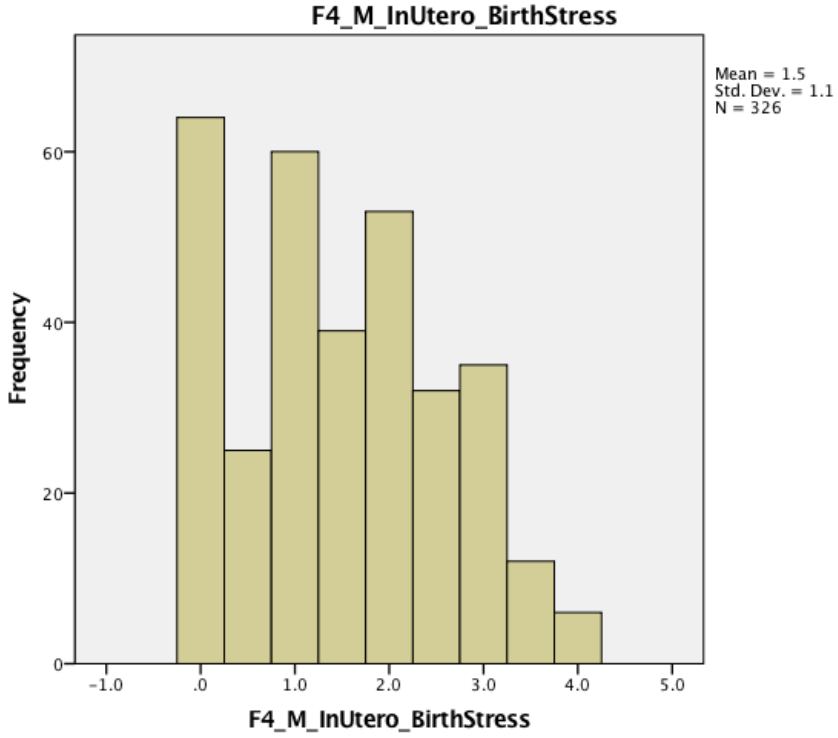
Appendix L

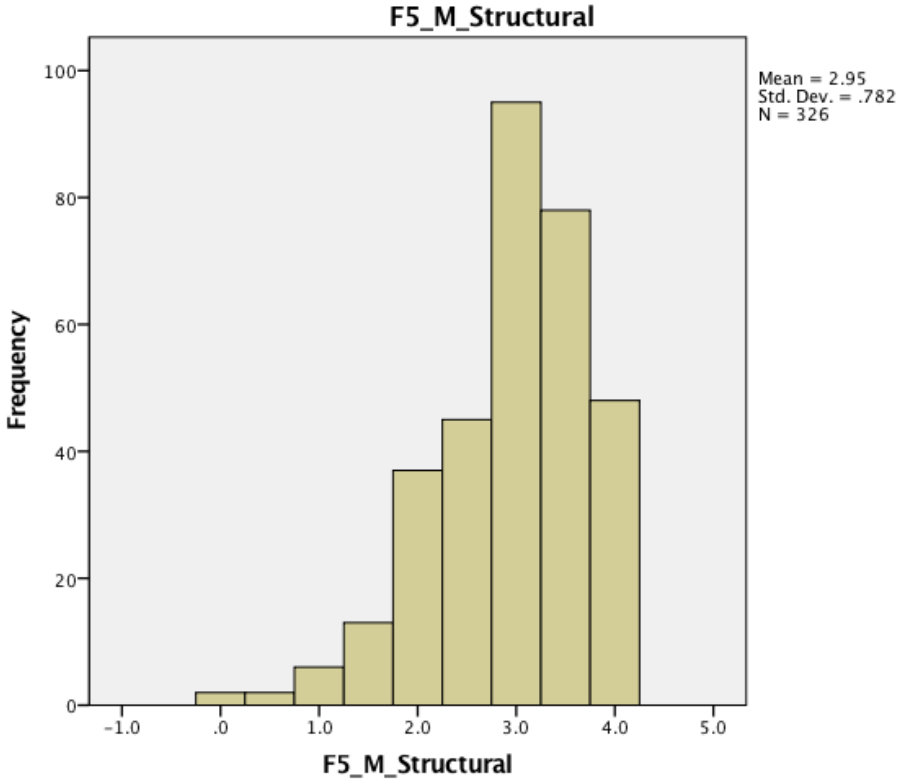
Histograms of the Six Cause Factors (Predictors)

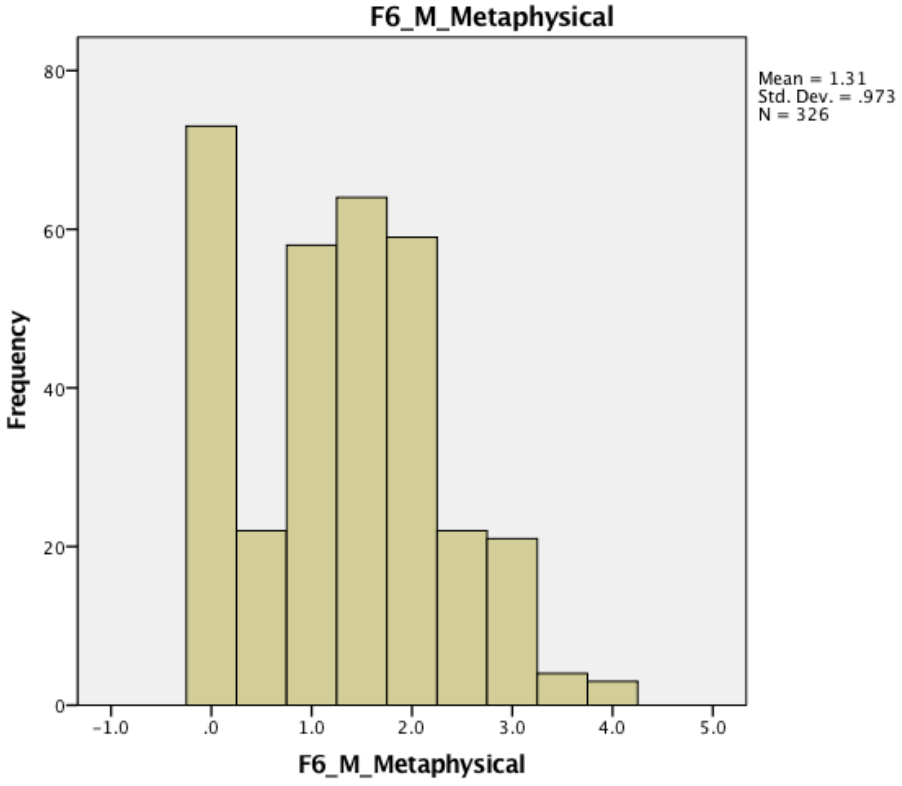

F1_M_PersonalAttributions
Mean = .78
Std. Dev. = .711
N = 326
Frequency
80
60
40
20
0
.0
1.0
2.0
3.0
F1_M_PersonalAttributions


F2_M_ParentalRiskFactors
Mean = .61
Std. Dev. = .581
N = 326
Frequency
100
80
60
40
20
0
.0
1.0
2.0
3.0
F2_M_ParentalRiskFactors


F3_M_EnvRisk
Mean = 1.47
Std. Dev. = .862
N = 326
Frequency
40
30
20
10
0
-2.0
.0
2.0
4.0
6.0
F3_M_EnvRisk


F4_M_InUtero_BirthStress
Mean = 1.5
Std. Dev. = 1.1
N = 326
Frequency
60
40
20
0
-1.0
.0
1.0
2.0
3.0
4.0
5.0
F4_M_InUtero_BirthStress


F5_M_Structural
Mean = 2.95
Std. Dev. = .782
N = 326
Frequency
100
80
60
40
20
0
-1.0
.0
1.0
2.0
3.0
4.0
5.0
F5_M_Structural


F6_M_Metaphysical
Mean = 1.31
Std. Dev. = .973
N = 326
Frequency
80
60
40
20
0
-1.0
.0
1.0
2.0
3.0
4.0
5.0
F6_M_Metaphysical