The biopsychosocial impact of Autism on families and the contribution of solar irradiance to its aetiology

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Thesis submitted in total fulfilment of the requirements for the degree of

Doctor of Philosophy

School of Health and Life Sciences

Federation University Australia

February, 2020

Acknowledgements

Let me acknowledge my principal supervisor Associate Professor Kathleen A. Moore for the continuous support and guidance. Her patience, motivation, immense knowledge, endless energy and guidance throughout the research helped me achieve the milestone of completing this research work. She was an inspiration for me throughout the research and made it possible for successful completion of this research.

I am also thankful to my co-supervisor, Dr. Evita March for always being supportive.

Finally, I am grateful to all of my colleagues at the Federation University Australia. Only reason to not mention any is because there is not enough space to name all who have welcomed me as a part of this academic community.

Statement of Authorship and Originality

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Dated: 26/02/2020

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List of Abbreviations

ABA	Applied Behaviour Analysis
ADHD	Attention-Deficit/Hyperactivity Disorder
APA	American Psychiatric Association
ASD	Autism Spectrum Disorder
ASSQ	Autism Spectrum Screening Questionnaire
BSA	Body Surface Area
CAST	Childhood Autism Spectrum Test
CBT	Cognitive Behavioural Therapy
CD	Conduct Disorder
CDC	Center for Disease Control and Prevention
DSM	Diagnostic and Statistical Manual of Mental Disorders
EPAP	European Protocol for Autism Prevalence
ESDM	Early Start Denver Model
ESI	Early Social Interaction
FAAR	Family Adjustment and Adaptation Response
F-COPES	Family Crisis Oriented Personal Evaluations Scales
FES	Family Environment Scale
FSS	Family Support Scale

HIV	Human Immunodeficiency Viruses
ICD	International Classification of Diseases
MSPSS	Multidimensional Scale of Perceived Social Support
OT	Occupational Therapy
OXTR	Oxytocin Receptor
PCA	Principal Components Analyses
PECS	Picture Exchange Communication System
PLS	Plain Language Statement
PRT	Pivotal Response Treatment
SEM	Structural Equational Modelling
SOC	Sense of Coherence
SPSS	Statistical package for the social sciences
USII	Unsupportive Social Interactions Inventory

Abstract

Autism Spectrum Disorder (ASD) is a lifelong disorder of unknown aetiology. A recent hypothesis is that a lack of Vitamin D is implicated in either the aetiology or maintenance of ASD. The human body synthesises Vitamin D from Ultraviolet-B (UVB) radiation found in sunlight. It follows that greater exposure to sunlight hours may be related to decreased rates of ASD. There are no interventions that target the causes of ASD rather therapies address either its symptoms or its comorbidities. ASD not only affects individuals, it also has an impact on their families. Family members have experienced social, occupational and personal costs associated with their child's ASD which can result in parental separation or divorce. While researchers have established some of the factors which contribute to the impact on families, this research has not addressed families living in regional areas nor have empirical studies used domain-specific scales. The aims in this thesis were: Study 1) to determine whether the prevalence rates of ASD vary as a function of exposure to sunlight by reviewing reported prevalence rates by latitude where, the greater the distance from the equator, the higher the expected prevalence rates; Study 2) to conduct interviews with parents and caregivers of children with ASD who live in a regional area to determine the factors which affect them and those which might protect them; and Study 3) use the interview data to develop domain specific measures and test a model of living with a child with ASD. The results of Study 1 revealed that there is an increase in the prevalence of ASD as distance from the equator increases lending some support to the hypothesis that Vitamin D is implicated in ASD. The 16 interviews conducted in Study 2 revealed seven themes: impact on finances; family life; child's health and behaviour, and schooling; child's future; limited support, and regional living. In Study 3, with 178 participants, domain-specific scales were developed to test a model of the impact of living with a child with ASD. Resilience manifested by social support and coping strategies, explained 54% of the variance in impact of living with a child with ASD which, was operationalised by financial and relationship costs, social impact and feelings. Family life as assessed in this thesis, is significantly impacted by living with

a child with ASD. The implications of these findings are discussed, especially around the need for greater exposure to outdoor activities and hence sunlight for children with ASD, more regional facilities and assistance for families, the importance of educational interventions for the public as well as enhancing levels of family resilience, as operationalised by support and coping strategies. Limitations of the studies and future research are discussed.

Chapter 1. Introduction

The Global Burden of Diseases, Injuries, and Risk Factors Study framework of the World Bank reports on epidemiological trends of diseases worldwide. In 2010, childhood-onset mental disorders including Attention-Deficit/Hyperactivity Disorder (ADHD), Conduct Disorder (CD) and Autism Spectrum Disorder (ASD) were included for the first time (Murray, Ezzati, Flaxman, Lim, Lozano, Michaud et al., 2012). Research into ASD has since burgeoned revealing high prevalence rates (e.g., 1.10% among 4 year-olds in Australia (May & Williams, 2018)) with males at 3 to 5 times greater risk (Burstyn, Sithole, & Zwaigenbaum, 2010; Loomes, Hull, & Mandy 2017). Despite the significance for those affected, and the family living with a child with ASD, its' aetiology is unknown. The high prevalence of ASD puts pressure on scientists to find a cure, while health providers have the difficult task of providing adequate healthcare to children with ASD and their families.

Investigations into the aetiology of ASD have suggested various causes which can be broadly classified as genetic (Heil & Schaaf, 2013; Sandin, Lichtenstein, Kuja-Halkola, Larsson, Hultman, & Reichenberg, 2014), gut related (Buie, 2015; Mayer, Padua, & Tillisch, 2014; Xu, M., Xu, X., Li, J., & Li, F., 2019), sunlight/Vitamin D (Grant & Cannell, 2013; Grant & Soles, 2009), and others (Guinchat, Thorsen, Laurent, Cans, Bodeau, & Cohen, 2012; Kern, King, Sykes & Geier, 2012). There is no conclusive evidence for any of these approaches.

That said, Cannell's (2008; 2017) proposal that vitamin D deficiency could be a risk factor for ASD has lead several researchers to offer various forms of support for his hypothesis. For instance, low levels of vitamin D have been found in individuals with ASD (Wang et al., 2016), their siblings (Fernell et al., 2015) and also there are reports of maternal deficiencies in Vitamin D (Magnusson et al., 2016). More recently, Grant and Cannell (2013) found an association between low levels of solar UVB radiation, which is present naturally in sunlight and which the body converts to Vitamin D, and high rates of ASD. If this is so, then the prevalence of ASD would be expected to be lower closer to

the equator where the hours and intensity of sunlight are greatest. This proposition is worth investigation as, if supported, greater time spent outdoors in sunlight may help ameliorate the symptoms of ASD among sufferers. If this is the case, it would lead to a reduction in the impact on families living with a child with ASD.

Aside from research into the aetiology of ASD, considerable effort has been expended to understand the impact on families living with a child with ASD in order to provide the most appropriate healthcare to the children with ASD and their families (Frantz, Hansen, & Machalicek, 2018; Hastings, Daley, Burns, & Beck, 2006; Singer, Ethridge, & Aldana, 2007). Psychosocial outcomes for the family of a child with ASD include poorer mental health (e.g., depression, anxiety) (Zimmerman, Ownsworth, O'Donovan, Roberts, & Gullo, 2016), poorer social and adaptive functioning (e.g., levels of independence, vocational, academic, and interpersonal functioning) (Zimmerman et al., 2016), a monetary burden due to greater investment in health care and less time for mothers in particular to engage in paid work (Dyke, Mulroy, & Leonard, 2009; Karst & van Hecke, 2012), high levels of fatigue (Giallo, Wood, Jellett, & Porter, 2013) and the need for constant vigilant parenting (Karst & van Hecke, 2012). Research has also identified economic (Saunders, Tilford, Fussell, Schulz, Casey, & Kuo, 2015) and psychosocial (Hayes, & Watson, 2013) difficulties associated with raising a child with ASD. Little is known about any compounding difficulties faced by families living in rural/regional Australia where facilities are often limited, and time and travel costs associated with accessing services may be prohibitive. Identifying and understanding any additional stressors associated with living in these areas will contribute to tailoring healthcare delivery to these families. It is also important to understand what factors might ameliorate the impact of living with a child with ASD. While past researchers (e.g., Bristol, 1987; McStay, Trembath, & Dissanayake, 2014; Paynter, Riley, Beamish, Davies, & Milford, 2013) have investigated various models to predict the burden on or adaptation of families living with a child with ASD none have done so with domain-specific measures relevant to these families.

The aims of the current series of studies are to: 1) explore the prevalence of ASD across the globe to ascertain if the prevalence rates of ASD increase with distance from the equator, that is, as the hours and intensity of sunlight containing UVB radiation which the body converts to Vitamin D, decrease the rate of ASD increases; 2) provide insight into the psychosocial impact of living with a child with ASD among parents and caregivers in a regional area, the Latrobe Valley in Victoria; 3) to develop domain-specific scales based upon the interview data from parents and caregivers that reflect, not just the factors which have an impact on their lives, but also those factors that may ameliorate this impact; and 4) as an extension of the scale development, to test a model of the impact on families of living with a child with ASD.

The results of these studies may have important implications for health care providers and inform educational interventions for both therapists and the public on the nature of ASD and its' management. The findings will especially target the parents and caregivers of children with ASD by contributing to our understanding of parental strategies to enhance their sense of resilience and promote a sense of positivity rather than burden.

Chapter 2. Autism Spectrum Disorder

Autism spectrum disorder (ASD) is an umbrella term for multiple neurodevelopmental conditions (Frye & Rossignol, 2011; Lai, Lombardo, & Baron-Cohen, 2014). It is a lifelong debilitating disease, which has an impact on both the individual and the family (Kamio & Inada, 2014; Lavelle et al., 2014). ASD is also considered costly to society in terms of healthcare support for, and loss of productivity by, the person effected (Lavelle et al., 2014).

People with ASD have difficulty expressing and understanding emotions; difficulty in understanding others' moods; impairment in expressive language; they avoid eye contact; have a preference for minimal changes to routine, and have restricted ways of using toys with a preference for isolated play (APA, 2013). They can demonstrate harmful behaviours and aggression (Duerden et al., 2012; Soke et al., 2016; Wolff et al., 2013), temper tantrums (Matson et al., 1996; Minshawi, Hurwitz, Morriss, & McDougle, 2015) and experience comorbidities with other conditions (Frye & Rossignol, 2016) such as anxiety disorder (Green & Ben-Sasson, 2010) and sleep disorders (Frye & Rossignol, 2016). Individuals with ASD have difficulty in establishing relationships with others, acting appropriately socially, and with independent living (Gray et al., 2014; Smith, Maenner, & Seltzer, 2012).

Diagnostically "Autism spectrum disorder is characterised by persistent deficits in social communication and social interactions across multiple contexts, including deficits in social reciprocity, nonverbal communicative behaviours used for social interaction, and skills in developing, maintaining, and understanding relationships" (DSM-5, American Psychiatric Association [APA], 2013, *p*.31). A diagnosis of ASD also "requires the presence of restricted, repetitive patterns of behaviour, interests, or activities" (APA, 2013, *p*.31). As the symptoms of ASD can change with the child's development, or symptoms may be masked by compensatory mechanisms, a diagnosis can be made based on historical information, provided that the current presentation causes significant impairment (APA, 2013).

2.1 Diagnostic Criteria

In Australia, a diagnosis of ASD is made against the diagnostic criteria in the Diagnostic and Statistical Manual of Mental Disorders (5th ed) (APA, 2013). A diagnosis of ASD is based on the level of impairments in social communication, restrictiveness and repetitiveness of behavioural patterns, and the presence of the above mentioned symptoms. Based on these criteria, the individual's ASD symptomology is described as meeting one of three severity specifiers (APA, 2013): levels one, two or three.

Those children diagnosed with Level 1 severity require support notably for their restricted/repetitive behaviours and impaired communications. Without supports in place, deficits in social communication can cause noticeable impairments such as in making or retaining friendships. Restricted/repetitive behaviours can cause significant interference with functioning in one or more contexts, such as difficulty switching between activities as well as problems of organisation and planning.

A diagnosis of Level 2 severity requires that the individual be provided with substantial support for marked deficits in their verbal and nonverbal social communication skills. These individuals also demonstrate inflexibility of behaviour, difficulty coping with change, and/or multiple restricted/repetitive behaviours which occur frequently enough to be obvious to the casual observer. Each of these behaviours interfere with the child's functioning in a variety of contexts and when encouraged to change or stop any of these behaviours, the child will typically experience distress and/or difficulty changing their focus or actions.

A diagnosis at Level 3 is the most severe. Those individuals diagnosed as level three require very substantial support for severe deficits in verbal and nonverbal social communication skills. They demonstrate restricted/repetitive behaviours including inflexibility of behaviour, extreme difficulty coping with change, or other restricted/repetitive behaviours, which markedly interfere with functioning in all spheres. They experience great distress/difficulty changing their focus or actions

notably those related to restricted/repetitive behaviours. Overall, the level of severity, and hence impairment, increases across the Levels from 1 to 3 but where, even at Level 1, symptoms can hamper the child's ability to achieve independence.

2.2 Prevalence of ASD

Epidemiological research into ASD can be traced back to 1966 when the entire 8–10 year old population of the County of Middlesex UK was screened to identify children with ASD (Lotter, 1966). A prevalence rate of 0.045% was found in that population of children. Since then epidemiological research has become an important part of the research efforts into ASD and this research has shown a continuous increase in the prevalence of ASD. The epidemic rate at which ASD has increased, or at least reports of it, may be judged by comparing a 2009 study's finding of 0.94% ASD prevalence rate in Cambridgeshire, UK (Baron-Cohen et al., 2009) to the earlier figure from Lotter (1966) of 0.045%. The 2009 data represent a 20-fold increase in the prevalence of ASD over nearly a half century. It can be asked if this increase is in fact accurate or an artefact of reporting or sampling. It does seem that better diagnosis and changes in diagnostic criteria over the period are thought to be among the reasons for this reported increase although there is no evidence to support this contention.

On the other side of the Atlantic in the USA, the prevalence rate of ASD for children aged under eight years was one in 500 in 1995, increasing to one in 250 in 2001 (Weintraub, 2011) and reaching one in 68 in 2010 (Wingate et al., 2014). Furthermore, the USA Center for Disease Control and Prevention (CDC) reported a significant increase in ASD in the years from 2002 to 2008 despite the use of consistent case definitions and surveillance methods (Baio, 2012). In the southern hemisphere in Australia, the most recent figures report the incidence of ASD in 4-year-olds as one in 90 (May & Williams, 2018). Baio's (2012) finding with respect to U.S data, which was collected using standardized criteria, can be taken to suggest that there is in fact an increase in the prevalence of ASD beyond reporting or sampling artefacts. These figures highlight the importance of exploring

every avenue to find the cause of ASD and management strategies for the families who live with children with ASD. A summary of the current theories on the aetiology of ASD is presented next.

2.3 Aetiology of ASD

Scientists have investigated a myriad of environmental and genetic factors in attempts to ascertain the aetiology of ASD but its' aetiology remains uncertain. Past studies have considered air pollution (Becerra, Wilhelm, Olsen, Cockburn, & Ritz, 2013; Kalkbrenner, Daniels, Chen, Poole, Emch, & Morrissey, 2010; Volk, Hertz-Picciotto, Delwiche, Lurmann, & McConnell, 2011; Volk, Lurmann, Penfold, Hertz-Picciotto, & McConnell, 2013; Windham, Zhang, Gunier, Croen, & Grether, 2006); environmental toxins such as mercury, nickel, selenium, lead, cadmium, aluminium, vinyl chloride, and trichloroethylene (Al-Farsi et al., 2013; Blaurock-Busch, Amin, Dessoki, & Rabah, 2012; Geier, Kern et al., 2012; Kinney, Barch, Chayka, Napoleon, & Munir, 2010); genetic heritability (Frazier et al., 2014; Heil & Schaaf, 2013; Sandin, Lichtenstein, Kuja-Halkola, Larsson, Hultman, & Reichenberg, 2014); hormonal imbalances, such as low levels of oxytocin (Anagnostou et al., 2012; Welch & Ruggiero, 2005); excess vasopressin (Carter, 2007), and more recently, deficiencies in Vitamin D (Cannell, 2008; 2017). Overall, these factors can be categorised into: (1) Genetic factors; (2) Gut related factors; (3) Sunlight/Vitamin D related factors; (4); Other and of course, those yet unknown (Figure 2.1). These possible aetiological factors are discussed next.

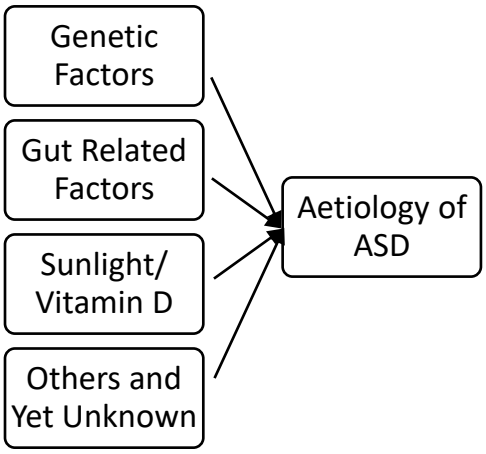

Figure 2.1: Proposed aetiologies of ASD

2.3.1 Genetic Factors

The genetic component of ASD is thought to involve either a transmitted genetic factor or a genetic variation. It is accepted that both of these may play a role towards an individual's susceptibility to ASD.

2.3.1.1 Transmitted Genetic Factor

Genetic transmission is the transfer of genetic information from parental genes to the next generation. Support for transmitted genetic influence in the aetiology of ASD is strengthened by reports from studies of twins and high-risk infant siblings, high-risk families and high-risk populations (Garon et al., 2009; Tick, Bolton, Happé, Rutter, & Rijsdijk, 2016). The concordance rate for ASD is reported as 60-70% in monozygotic twins but a lesser rate of 5-30% in siblings (Bailey et al., 1995; Ozonoff et al., 2011). Results from the largest twin study of those with ASD which was conducted in California by Hallmayer, Cleveland, Torres, Phillips, Cohen, Torigoe et al. (2011) with 54 monozygotic twins and 138 dizygotic pairs, found similar heritability levels to Bailey et al. for ASD. Hallmayer et al. reported a 70% chance of a monozygotic twin developing ASD when the other twin was diagnosed with ASD while the rate was 35% for dizygotic twins. Overall, these results from twin and sibling studies are consistent but fail to offer any reasons for the stronger relationship between monozygotic twins and other siblings whether dizygotic twins or not.

2.3.1.2 Genetic Variations

Genetic variation refers to the genetic differences between individuals and populations and for reference they are divided into common variants and rare variants. The genetic architecture of ASD involves the interplay of both common and rare genetic variations and their subsequent impact on hundreds of genes (De Rubeis et al., 2014).

Common variation refers to a genetic variation which is present in more than 1% of the population (Yoo, 2015). Consistently reported among the common gene variants related to ASD are gamma-aminobutyric acid A receptor, gamma-aminobutyric acid receptor subunit beta-3, oxytocin

receptor (Yamasue, 2016; Rijlaarsdam et al., 2017); reelin (Skaar et al., 2005); serotonin transporter (Sutcliffe et al., 2005; Ma et al., 2010); N-methyl-D-aspartate receptor (Lee, Choi, & Kim, 2015; Yoo, Cho, Park, Yang, & Kim, 2012); arginine vasopressin receptor 1A (Yang et al., 2010); engrailed homeobox 2 (Wang et al., 2008); hepatocyte growth factor receptor (Rudie et al., 2012), and contactin-associated protein-like 2 genes (Penagarikano & Geschwind, 2013).

Rare variation is a genetic variation which is present in 1% or less of the population (Yoo, 2015). The well-known rare variations related to ASD are single-gene disorders; mutation in the FMR1 gene; mutations in tuberous sclerosis complex 1; mutations in tuberous sclerosis complex 2; Dup15q syndrome; deletions in the 16p11.2 region; Rett syndrome (mutation in MeCP2), and neurofibromatosis (mutations in NF1) (Yoo, 2015).

In ASD patients, both common and rare variants can be inherited from a paternal and/or maternal source (Alonso-Gonzalez, Rodriguez-Fontenla, & Carracedo, 2018) or they may appear as a de novo ("new") gene mutation in the affected person (De Rubeis et al., 2014). Based on the type of genetic variation it is classified as copy number variation (CNV) (Pinto et al., 2010), single nucleotide variation (SNV) (Gadow, Roohi, DeVincent, Kirsch & Hatchwell, 2010; Kim et al., 2002; Sanders et al., 2015; Wu et al., 2005), or as small insertions and deletions (indels) of genes (Iossifov et al., 2012).

Indel is a genetics term to describe the insertion or deletion of genes in the genome of an organism such as the deletions of genes within the region 15q11–13 of chromosome 15 in ASD patients (Veltman, Craig, & Bolton, 2005). The term CNV refers to the phenomenon in which sections of a genome are repeated and this repetition number in the genome varies between individuals, such as microdeletion syndrome in chromosome 2 in the region of 2p15-2p16.1 in patients with ASD (Liu et al., 2011). This syndrome is due to a small deletion in the chromosome. SNV refers to the phenomenon in which the substitution of a single nucleotide occurs at a specific position in the genome such as a mutation in the FMR1 gene in ASD patients.

Clearly a wide range of genetic variations are proposed to be involved in ASD and these consist of interplays between gene-gene and gene-environment interactions (Yoo, 2015). This complexity contributes to the difficulty in investigating the genetic architecture of ASD and to date no conclusive evidence is present to categorically assign causality to any single genetic variation. Ongoing research is required to increase our knowledge of the genetic factors related to ASD.

2.3.2 Gastrointestinal Tract Related Factors

The microbiota in the Gastrointestinal Tract (GIT) has been shown to be involved in the modulation of brain development (Heijtz et al., 2011), brain functions (Bienenstock & Collins, 2010; Cryan & Dinan, 2012) and behaviours (Bienenstock & Collins, 2010; Cryan & Dinan, 2012; Heijtz et al., 2011). There is recent and growing global interest in the gut-brain axis and its involvement in both typical development as well as in neurodevelopmental disorders (Doenyas, 2018). The role of the gut-brain axis has also been implicated in mood-swings (Mangiola et al., 2016), behavioural deficits (Critchfield, Van Hemert, Ash, Mulder, & Ashwood, 2011), anxiety (Mazurek et al., 2013), and sensory over-responsivity (Mazurek et al., 2013), and is also under investigation in the aetiology and symptomatology of ASD (Louis, 2012; Mayer et al., 2014; Vuong & Hsiao, 2017).

The gut microbiota, over the millennia of human evolution, have become integrated with the immune system, metabolism and nervous system (Sampson & Mazmanian, 2015; Sommer & Bäckhed, 2013) and these gut-adapted bacteria and their metabolites might, according to some researchers, have a critical role in the aetiology of ASD (Buie, 2015; Xu et al., 2019). In fact, research has shown a link between the GIT and ASD which operates via the gut-brain axis (Mayer, Padua, & Tillisch, 2014). This association was further highlighted in a recent meta-analysis of nine studies by Xu et al. (2019).

The nine studies considered by Xu et al. included 254 patients with ASD and each study had investigated the association between gut microbiota and ASD. Xu et al. found that children with ASD had lower percentages of Akkermansia, Bacteroides, Bifidobacterium, and Parabacteroides and a

higher percentage of Faecalibacterium in the total detected microflora in their gut compared to control participants of similar ages. They also found less abundance of Enterococcus, Escherichia coli, Bacteroides, and Bifidobacterium and higher abundance of Lactobacillus in the gut microbiota in the children with ASD. Although these data suggest an association between ASD and alterations in the microbiota composition in the gut (Buie, 2015; Xu et al., 2019) additional research is necessary to evaluate these associations and provide evidence for their respective roles in the aetiology, or even the maintenance, of ASD and its symptoms (Xu et al., 2019).

We know that gastro-intestinal symptoms such as abdominal pain, gaseousness, diarrhoea and constipation are strongly correlated with the severity of ASD (Adams, Johansen, Powell, Quig, & Rubin, 2011; Chaidez, Hansen, & Hertz-Picciotto, 2014). This knowledge, combined with a growing body of reports from animal models (e.g., rodent models) (Argyropoulos, Gilby, & Hill-Yardin, 2013; Hsiao et al., 2013) and human epidemiological studies (Adams et al., 2011; Rosenfeld, 2015) have linked disruptive alterations in the gut microbiota or dysbiosis to ASD symptomology. Despite these findings, there is no conclusive evidence pointing to gut related disorders as being the cause of ASD, rather these symptoms might stem from related behaviours such as poor diet and lack of exercise. Perhaps a more novel yet interesting hypothesis regarding the aetiology or maintenance of ASD and its' symptoms is related to the levels of Vitamin D in the body or, more specifically, exposure to sunshine. This latter is because when human skin is exposed to solar Ultraviolet-B (UVB) radiation in sunlight, Vitamin D is produced in the body.

2.3.3 Sunlight Related Factors

It has been known since the 1970s that light penetrates the human skull and the brain, and that it can travel a 13.3 cm path from one side of the human head to the other (Jobsis, 1977). Numerous studies have documented the direct impact of different light frequencies on different brain parts (Alkozei et al., 2017; Loving et al., 2005; Rybak et al., 2006; Vandewalle et al., 2007) and

the indirect impact via the eyes (Berson, Dunn, & Takao, 2002; Dijk, & Lockley, 2002; Dijk & Archer, 2009) and the skin (Campbell & Murphy, 1998; Denda, 2015).

Among the effects of light are a positive impact on alertness (Lockley, Evans, Scheer, Brainard, Czeisler, & Aeschbach, 2006), on cognitions (Vandewalle et al., 2006), on brain function regulation (Vandewalle et al., 2007), and memory (Alkozei, Smith, Dailey, Bajaj, & Killgore, 2017) as well as a reductions in the severity of attention deficit disorder (Rybak, McNeely, Mackenzie, Jain, & Levitan, 2006), all of which are related to some extent to the symptoms present in ASD.

Despite growing interest in sunlight's impact on human physiology very little research is available on the impact of solar exposure and outdoor activity on the children with ASD. Yet many of the comorbidities associated with ASD are already known to have a relationship with lack of physical activity and exposure to sunlight. To this end, some research suggests that exposure/physical activity in natural environments improves symptom expression in attention-deficit/hyperactivity disorder (ADHD) (Kuo & Taylor, 2004; Taylor & Kuo, 2011; Taylor, Kuo, & Sullivan, 2001) as well as behavioural outcomes in people with ASD (Couper et al., 2013; Yuill et al., 2007). However, no study has examined the impact of long-term sunlight exposure on children with ASD.

The small number of studies that have explored outdoor activities and their effects on the health and behavioural outcomes of children with ASD have all focused on children's outdoor activities in the playground during school recess periods (Couper et al., 2013; Kretzmann, Shih, & Kasari, 2015; Lang et al., 2011; Locke, Shih, Kretzmann, & Kasari, 2016; Menear et al., 2006; Yuill et al., 2007). Clearly these times are of brief duration and further limited by school terms. A recent study conducted by Li, Larsen, Yang, Wang, Zhai and Sullivan (2019) in China, documented the results of exposure to nature for children with ASD. They found that children with ASD who were exposed to nature, that is, who spent more time in green spaces and children's parks in the city during the day time, experienced motor-sensory, emotional and social benefits. As sunlight leads to the production of Vitamin D in the body (Holick, 2004), which others have considered in terms of the

aetiology of ASD, it might be that increased levels of Vitamin D produced by greater outdoor exposure to sunlight is implicated in the improvements noted in these children.

2.3.3.1 Ultraviolet-B and Vitamin D

Cannell (2008) proposed that a deficiency in Vitamin D could contribute to the aetiology of ASD as Vitamin D regulates dozens of proteins involved in brain development. Several researchers since his initial proposal have reported low levels of Vitamin D in individuals with ASD (Kočovská et al., 2014; Wang et al., 2016), their siblings (Fernell et al., 2015) and also maternal deficiencies in Vitamin D (Chen, Xin, Wei, Zhang, & Xiao, 2016; Dealberto, 2011; Magnusson et al., 2016). Overall, researchers have suggested that these results can be interpreted to indicate an association between Vitamin D deficiency and autism-related traits (Cannell, 2017, Vinkhuyzen et al., 2018). Support for Cannell's hypothesis also comes from reports that some level of improvement in autistic symptoms has been achieved following the administration of Vitamin D supplements (Jia, Wang, Shan, Xu, Staal, & Du, 2015; Stubbs, Henley, & Green, 2016) as well as that inferred by exposure to sunlight in Li et al.'s (2019) study described above. Despite tentative support for an association between Vitamin D and ASD, no conclusive evidence has yet emerged, although related work tends to offer some support.

This related research has reported an association between low solar UVB radiation and high prevalence rates of ASD (Grant & Cannell, 2013; Grant & Soles, 2009; Mazahery, Camargo, Conlon, Beck, Kruger, & von Hurst, 2016). When human skin is exposed to solar UVB radiation in sunlight, 7-dehydrocholesterol, which is present in the skin, converts to pre-Vitamin D3 and provides the main source of natural Vitamin D to the body (Grant & Cannell, 2013; Holick, 2004). The kidneys then convert pre-Vitamin D into Vitamin D (Holick, 2004). When levels of UVB are low in areas of reduced sunlight hours or people's limited exposure to sunlight, this precludes, to some extent, the natural production of Vitamin D in the body (Ghayan, 2017). This work has led to the argument that lack of

exposure to sunlight may have a role in the aetiology of ASD (Ghayan, 2017; Ghayan, A. & Ghayan, S., 2017; Grant & Cannell, 2013 Mazahery et al., 2016).

Support for this hypothesis is strengthened by the higher rates of ASD among non-Caucasian races (Kawa et al., 2016) as they require higher levels or longer periods of solar exposure to absorb the necessary level of sunlight by virtue of their darker skin. Interestingly, in terms of these differential exposure rates to absorb sunlight, Bolton, McDonald, Curtis, Kelly, and Gallagher (2014), Dealberto (2011), and Lehti, Hinkka-Yli-Salomäki, Cheslack-Postava, Gissler, Brown, and Sourander (2013) have all associated black ethnicity with higher rates of ASD compared to Caucasians within the same geographical areas. A recent review of 17 published studies concerning immigrants and ethnic minorities in Europe by Kawa et al. (2016) found that in 15 of those studies, a higher prevalence rate of ASD was indicated among children of immigrants in comparison to native children. The majority of these immigrant children were from outside Europe, notably from Africa and South America where skin pigmentation is typically darker and, it can be argued, the levels or hours of sunlight needed to generate pre-Vitamin D3 are greater.

In summary, race or more specifically skin pigment based differences in the prevalence of ASD and the growing evidence for a link between ASD and Vitamin D strengthen the arguments for an association between exposure to sunlight and the subsequent production of Vitamin D in the body. This hypothesis brings into focus the entire spectrum of sunlight. It may be that this line of argument can be strengthened further if there is a link between the prevalence rates of ASD and sunlight (converted to Vitamin D) as the hours and intensity of sunlight vary by latitude. Broadly speaking, if the prevalence rates of ASD vary across latitudes, that is, North and South of the equator, where at the equator the hours of sunlight per day and per year are greatest, this may contribute to the evidence that sunlight (Vitamin D) is implicated in the aetiology of ASD. Such a proposition warrants initial investigation and will be a focus of a literature review to be provided later in this thesis.

2.3.4 Others and Yet Unknown

A large number of other factors have been investigated for their role in the aetiology of ASD, such as the child's sex, maternal age, and perinatal and neonatal factors. The most significant risk factor towards ASD is being born male (Guinchat et al., 2012; Hallmayer et al., 2011; Williams, MacDermott, Ridley, Glasson, & Wray, 2008). One study has indicated that boys have up to a 5-fold higher prevalence rate for ASD than girls (Burstyn et al., 2010) although a recent meta-analysis of 54 studies by Loomes et al. (2017) identified the male-to-female ratio as closer to 3:1. In Australia, Williams et al. (2008) identified 81% to 86% of children with ASD were male. It is not clear why this gender imbalance exists and how it relates to the fact of being born male.

Maternal age has been investigated as a familial risk factor linked to ASD (Guinchat et al., 2012). Maternal age over 35 years is significantly associated with an increased risk of ASD in the child (Burstyn et al., 2010; Durkin et al., 2008; Williams, Helmer, Duncan, Peat, & Mellis, 2008; Zhang et al., 2010). Offspring of fathers aged over 40 years have also been identified as being at a higher risk of ASD (Durkin et al., 2008) but again the reasons behind these increased odds are currently unknown.

Research on the relationship between perinatal factors and ASD has focussed on premature birth, breech birth (Burstyn et al., 2010), prolonged labour (Brimacombe, Ming, & Lamendola, 2007) and low birthweight children (Burstyn et al., 2010; Ikejiri, Hosozawa, Mitomo, Tanaka, & Shimizu, 2016). Studies have reported individuals born prematurely are at an increased risk of ASD (Brimacombe et al., 2007; Buchmayer, Johansson, Johansson, Hultman, Sparén, & Cnattingius, 2009; Schendel, & Bhasin, 2008; Zhang et al., 2010; Leavey, Zwaigenbaum, Heavner, & Burstyn, 2013). Recently, Agrawal, Rao, Bulsara, and Patole (2018) reviewed 18 published studies on preterm infants and any subsequent association with ASD. They concluded that the prevalence of ASD was significantly higher in the preterm sample of infants compared with babies born at term, with the

overall prevalence rate for ASD in these preterm infants at 7% (Agarwal et al., 2018). The reasons for this higher rate of ASD in preterm infants are unclear.

Neonatal factors such as Appearance, Pulse, Grimace, Activity and Respiration which are assessed by the APGAR Test have also been investigated for a possible role in the aetiology of ASD. The APGAR Test is performed on newborns at 1 and 5 minutes after birth. The 1-minute test is used as an indication of how well the baby has tolerated the birthing process while the 5-minute test indicates how well the baby is coping in the world at that time. A low 1-minute APGAR test score has been linked with increased risk of ASD (Burstyn et al., 2010; Polo-Kantola, Lampi, Hinkka-Yli-Salomäki, Gissler, Brown, & Sourander, 2014); a low 5-minute APGAR test score has also been associated with higher risk for ASD (Wang, Geng, Liu, & Zhang, 2017). A low score APGAR Test itself is not an aetiological factor but a measure that identifies a susceptibility to ASD. It does not seem that any preventative measures can be implemented at this time.

Postnatal factors such as abnormal zinc deficiency in the child (Yasuda, Yoshida, Yasuda, & Tsutsui, 2011) may also contribute to the pathogenesis of ASD. As discussed earlier a large body of research is available on the environmental and birth related factors associated with ASD. While not considered to be directly causal of ASD, they are seen as possible susceptibility factors.

2.4 Comorbidities

ASD is associated with several comorbidities (Frye & Rossignol, 2016; Mannion & Leader, 2013) the most common of which is an anxiety disorder (Green & Ben-Sasson, 2010; Gordon-Lipkin, Marvin, Law, & Lipkin, 2018; Mazurek et al., 2013; Van Steensel, Bögels, & Perrin, 2011; Vasa et al., 2013; White, Oswald, Ollendick, & Scahill, 2009; Williams, Leader, Mannion, & Chen, 2015). The results of a meta-analysis of 31 studies by Van Steensel et al. (2011) found that upwards of 40% of children with ASD had at least one comorbid anxiety disorder. Symptoms of ADHD also frequently co-occur in people with ASD (Salazar et al., 2015; Van der Plas, Dupuis, Arnold, Crosbie, & Schachar, 2016). Obsessive compulsive disorder (Leyfer et al., 2006; Van der Plas et al., 2016), sensory over-

responsivity (Green & Ben-Sasson, 2010; Green, Ben-Sasson, Soto, & Carter, 2012; Green et al., 2013; Marco, Hinkley, Hill, & Nagarajan, 2011; Mazurek et al., 2013; Schauder & Bennetto, 2016), gastrointestinal problems (Klukowski, Wasilewska, & Lebensztejn, 2015; Mayer et al., 2014; Mazurek et al., 2013) and sleep disorders (Klukowski et al., 2015; Rossignol & Frye, 2011) are also present in children with ASD. In fact, paediatric sleep disorder is reported by the parents or caregivers of children with ASD in up to 50% of cases (Miano, Giannotti, & Cortesi, 2016). Children with ASD also have an increased risk of a mood disorder (Gordon-Lipkin et al., 2018).

Other comorbidities include seizures, hearing impairments, headaches, bacterial and viral illnesses, and ear, nose and throat infections (Bauman, 2010). A high incidence of gross and fine motor skill dysfunctions such as motor apraxia and toe-walking have also been identified (Matson, Matson, & Beighley, 2011). Children with ASD have increased rates of dental caries, periodontal disease, bruxism and pica (Stein, Polido, Mailloux, Coleman, & Cermak, 2011) typically the result of poor oral self-care. Poor diet and both underweight (McCoy, Jakicic, & Gibbs, 2016; Mouridsen, Rich, & Isager, 2008) and obesity rates are also higher in children with ASD and are discussed in a later section. Children with ASD face many functional challenges in engaging in appropriate behaviours, and these include their atypical eating and often lack of physical activity, which are discussed next.

2.5 Functional Challenges of ASD

2.5.1 Behaviours

The stereotypical behaviours and interests of children with ASD include echolalia, motor stereotypies (e.g., hand flapping, body rocking), compulsions (such as ordering, hoarding, and touching) (Bodfish, Symons, Parker, & Lewis, 2000; Lewis, & Bodfish, 1998), and unhealthy obsessions and repetitive behaviours (such as repetitive speech and routines) (Calderoni, Bellani, Hardan, Muratori, & Brambilla, 2014; Kim, Lim, & Kaang, 2016; Leekam, Prior, & Uljarevic, 2011; Wolff, Hazlett, Lightbody, Reiss, & Piven, 2013). Additionally, harmful behaviours and aggression are often present and can include tantrums and self-harm (Duerden et al., 2012; Minshawi, Hurwitz,

Morriss, & McDougle, 2015; Soke et al., 2016; Wolff et al., 2013), and destructive/aggressive behaviours notably towards others (Mazurek, Kanne, & Wodka, 2013; Hill et al., 2014; Kaat, & Lecavalier, 2013) which can often reach extreme levels (Bauminger et al., 2010; Carroll et al., 2014; Carter Leno et al., 2018; Ghayan, A. & Ghayan, S., 2014; Yang et al., 2017). Children with ASD are attentive to routines and often have difficulty adjusting to unfamiliar surroundings or changes in their routines (APA, 2013) all of which are often difficult for parents and others to understand and manage.

2.5.2 Atypical Eating

Children with ASD have been found to consume significantly more daily servings of sweetened beverages and energy-dense foods than typically developing children (Evans et al., 2012). This difference can be attributed to problem eating behaviours such as food selectivity (Martins, Young, & Robson, 2008; Sharp et al., 2013) that additionally coincide with typical preferences for low-nutrition, energy-dense foods and the rejection of fruits, vegetables, and whole grains (Ahearn, Castine, Nault, & Green, 2001; Evans et al., 2012; Schreck, Williams, & Smith, 2004). It may be that parents, at times, provide these preferred beverages and foods in attempts to pacify their child. Thus, to some extent, unwittingly perpetuating the behaviours.

Feeding problems in children with ASD have been associated with abnormal growth and developmental and psychiatric conditions such as Anorexia Nervosa (Keen, 2008), and malnutrition (Tang, Piazza, Dolezal, & Stein, 2011). Children with ASD have an higher prevalence rate of underweight than in the general population (Mouridsen et al., 2008) conversely, higher rates of overweight and obesity are also described in individuals with ASD (Criado et al., 2017; Egan, Dreyer, Odar, Beckwith, & Garrison, 2013; Hill, Zuckerman, & Fombonne, 2015; Sharp et al., 2013; Whiteley, Dodou, Todd, & Shattock, 2004; Xiong et al., 2011).

The prevalence of obesity in children with ASD is 30.4% compared to 23.6% in children without ASD (Curtin, Anderson, Must, & Bandini, 2010). A recent study reported an odds ratio of

overweight/obesity 1.57 times higher in children with ASD than in the general population of children of similar ages while those with severe ASD symptoms were 1.7 times more likely to be classified as overweight/obese compared with children with mild ASD symptoms (Levy et al., 2019). Similarly, children with ASD are 48% more likely to be underweight than typically developing children (McCoy et al., 2016). Overall, atypical eating behaviours are significantly more common in children with ASD (70.4%) than in children with other disorders (13.1%) or in typically developing children (4.8%) (Mayes & Zickgraf, 2019).

The higher rates of overweight, obesity, and inactivity in children with ASD can be attributed to a number of factors ranging from sensory stimulation (both under- and over-stimulation) and social anxiety to physical barriers and disabilities (Obrusnikova & Cavalier, 2011). Obesity is a highly prevalent comorbidity in children with ASD and can lead to significant health consequences (Curtin, Anderson, Must, & Bandini, 2010; Tyler, Schramm, Karafa, Tang, & Jain, 2011). As discussed, the prevalence of obesity in children with ASD is 30.4% compared with 23.6% in age-matched children without ASD (Curtin et al., 2010). Among children with chronic disabilities, the prevalence of obesity is greater in children with ASD than in children with other developmental disabilities, including ADHD and learning disability (Chen, Kim, Houtrow, & Newacheck, 2010). It is clear that the difficulties associated with the child's problem eating behaviours can place a significant strain on the family and its' routines. The higher than normal rates of underweight in children with ASD has been attributed to eating disorders and a restricted dietary range (Sedgewick, Leppanen, & Tchanturia, 2019).

2.5.3 Physical Activity

Engaging in physical activity has positive effects on people's physical health (Barber, Eccles, & Stone, 2001; Bhat, Landa, & Galloway, 2011; Law, 2002), their psychosocial well-being (Barber et al., 2001; Feldman, & Matjasko, 2005; Law, 2002; LeBlanc et al., 2012; Timmons et al., 2012), socio-emotional wellbeing, and for children in particular, their developmental functioning (Bhat et al., 2011). Physical activity is associated with a sense of competence, improved social, emotional and

psychological skills and enhanced life satisfaction (Eccles, Barber, Stone & Hunt, 2003; Law, 2002; Mahoney, Cairns, & Farmer, 2003). Physical activity has been linked to enhanced cognitive performance as well. Neuroimaging (fMRI) studies have demonstrated that higher fitness levels in children are associated with increased hippocampal and dorsal striatum volume, areas of the brain associated with attention span, focus, and problems involving response and motor coordination (Chaddock et al., 2010a; Chaddock et al., 2010b).

An analysis of 59 articles published between 1947 and 2009 showed significant improvements in achievement and cognitive scores for children who engaged in higher levels of physical activity (Fedewa & Ahn, 2011). Encouraging participation in the recommended levels of physical activity combined with reduced levels of sedentary behaviour may support the psychosocial wellbeing of young children (LeBlanc et al., 2012; Timmons et al., 2012) including those with ASD. Despite the acknowledged benefits of exercise, children with ASD spend more time in sedentary activities than children who do not have ASD (Jones et al., 2017; Mazurek, & Engelhardt, 2013; Must et al., 2014) and, in combination with poor diet, this makes them more susceptible to overweight or obesity.

Health issues in children with ASD are complicated further by the unhealthy lifestyle of many children with ASD including the diet related issues mentioned above and a lack of physical activities (Jones et al., 2017; Mazurek, & Engelhardt, 2013; Must et al., 2014). This latter might also have an impact on their siblings and parents by also reducing their participation in physical and social activities.

Play is the primary occupation of childhood. Children have the right to rest and leisure, to engage in play and recreational activities appropriate to their age, and to participate freely in the cultural life and the arts (Office of the United Nations High Commissioner for Human Rights, 1990). Children with ASD do not always engage in these activities. In addition to developmental opportunities inherent in outdoor play, active outdoor play can significantly contribute to health-related development (Wyver, Tranter, Naughton, Little, Sandseter, & Bundy, 2010) including

exposure to solar UVB leading to Vitamin D production in the body as discussed earlier. Outdoor play refers to play that takes place outside and is typically an active, freely chosen process rather than outcome oriented, and it is intrinsically motivating (Bundy, 1993). Yet, for children with ASD, participation in outdoor activities including play is often restricted or disrupted (Blagrave & Colombo-Dougovito, 2019; Stanish, Curtin, Must, Phillips, Maslin, & Bandini, 2017). Parents often limit the outdoor activities of these children due to: 1) concerns for the safety of their child, 2) the probability that the child's behaviour will affect the activity especially if others are involved, 3) lack of their child's acceptance in the community if group activities are involved, and 4) limited opportunity and time for outdoor activities (Blagrave & Colombo-Dougovito, 2019).

Individuals with ASD spend more time engaged with electronic screen media than any other leisure activity, with television and movie viewing being more popular than computer usage (Shane & Albert, 2008). An inverse relationship between increased TV viewing and indicators of psychosocial health has been reported in children from the general population (LeBlanc et al., 2012) and this effect is also likely in children with ASD and may, in fact, be stronger. This engagement with screen media limits both exposure to outdoor exposure to sunlight and possible engagement in physical activities and exercise. It may also limit attempts to have the child socialise, one of the limiting factors in ASD. There is no known cure for ASD but pharmacological treatments and non-pharmacological therapies have been used as interventions in children with ASD to reduce the impact of symptoms and these are discussed next.

2.6 Chapter Summary

ASD is a set of heterogeneous neurodevelopmental conditions. Reports on the prevalence of ASD have shown steady rates of increase over the past 45 years with the latest estimates in Australia indicating that one in 90 4-year-olds have ASD, with males more susceptible to ASD than females. Various possible aetiological factors have been investigated including genetics, GIT, environmental factors and, more recently, deficiencies in Vitamin D, all without any conclusive evidence. ASD is

associated with several comorbidities the most common of which is an anxiety disorder (up to 40%). Obsessive-compulsive disorder, sensory over-responsivity, gastrointestinal problems and sleep disorders are also present in children with ASD. The stereotypical behaviours and interests of the children with ASD pose numerous challenges for the families and these may, at times, involve aggression and self-harm. The unhealthy lifestyles of many children with ASD, such as lack of physical activity, more screen time, and atypical eating pose further functional challenges for parents. There are also higher rates than normal of both overweight and underweight present in children with ASD and the lack of physical activity may be a significant contributor.

Chapter 3. Treatments for ASD

There is no known cure for ASD but early interventions are crucial to help reduce the impact of symptoms and provide individuals with skills to improve their overall lifestyle and wellbeing. These interventions can be either pharmacological or non-pharmacological or a combination of both. Pharmacological treatments for children with ASD include psychostimulants, antipsychotic drugs and antidepressants which provide symptomatic relief in some children. Non-pharmacological therapies such as early intensive behavioural interventions are designed to promote adaptive behaviours, and help to improve expressive and receptive language in children with ASD (Peters-Scheffer, Didden, Korzilius, & Sturmey, 2011). There are also biomedical or holistic practitioners who promote alternative treatments that have no evidence base and no biological plausibility in ASD (Levy & Hyman, 2008). Singer and Ravi (2015) cautioned about the possible harm associated with certain non-evidence-based treatments, such as the misuse of chelating agents to reduce toxic levels of mercury and lead and the misuse or overuse of hyperbaric oxygen chamber for oxidative stress and inflammation reduction. A review of the pharmacological and non-pharmacological treatments currently used with children with ASD follows.

3.1 Pharmacological Treatments

Currently evidence-based pharmacological treatments for ASD are limited to the treatment of comorbidities, not the symptoms of ASD itself (Lord, Elsabbagh, Baird, & Veenstra-Vanderweele, 2018). Pharmacological treatments cover different classes of drugs such as antipsychotics, antidepressants, broad-spectrum penicillins, penicillins/cephalosporins, central nervous system stimulants, bronchodilators/spasm relaxants, asthma preventives, antimigraine drugs and hormone treatments. No biomedical agent has been shown to reliably improve social communication in children with ASD however, antipsychotic drugs have been shown to effectively reduce challenging and repetitive behaviours in these children (Lai et al., 2014) but the adverse effects of these medicines (such as haloperidol), are grounds for concern (McPheeters et al., 2011). This next section

focuses on the main drug classes and the most prescribed medications used in the treatment of related disorders in children with ASD.

3.1.1 Antipsychotic Drugs

Antipsychotics are the most frequently prescribed drugs for children who demonstrate aggressive and disruptive behaviours associated with ADHD, oppositional defiant and conduct disorder (CD), and also for adolescents with mood and anxiety disorders (Patten, Waheed, & Bresee, 2012). They are also widely used in the treatment of schizophrenia and numerous other psychotic disorders (Baldessarini & Tarazi, 2005). These drugs work by targeting neurotransmitter receptors such as dopamine and serotonin. The reason they are prescribed to children with ASD is because they help improve the symptoms of irritability and agitation demonstrated by many of these children, and they help with associated comorbidities such as ADHD and anxiety. Commonly prescribed antipsychotic drugs for children with ASD are risperidone, aripiprazole, quetiapine, ziprasidone, and to a lesser extent, olanzapine (Posey, Stigler, Erickson, & McDougle, 2008). Typical antipsychotics have been used as well.

The term *Typical* antipsychotic refers to the class of antipsychotic drugs first developed in the 1950s which are used to treat psychosis and schizophrenia. Typical antipsychotics like haloperidol have been used successfully to improve social withdrawal and stereotypy symptoms in children with ASD, however, they are regarded as less safe due to the side effects of drug-induced movement disorders such as tardive dyskinesia which can occur with higher dose frequency (Anderson, Campbell, Grega, Perry, Small, & Green, 1984; Campbell et al., 1978; Campbell, Anderson, Small, Perry, Green, & Caplan, 1982; Campbell, Armenteros, Malone, Adams, Eisenberg, & Overall, 1997).

Atypical antipsychotics were introduced after the 1970s. Randomised controlled trials of atypical antipsychotics like risperidone (Kent et al., 2013) and aripiprazole (Owen et al., 2009) have demonstrated improvement in the symptoms of irritability or agitation in children with ASD. In addition to improvements in irritability and agitation, these two medications have also shown

reductions in these children's levels of aggression, self-injury, and other disruptive behaviours (Fung et al., 2016). Over the last two decades, successful results from several randomised control trials of antipsychotics use in children with ASD, such as those discussed, have led the US Food and Drug Administration (FDA) to approve risperidone and aripiprazole for the treatment of irritability in children with ASD (Shafiq & Pringsheim, 2018). Currently these two drugs remain the only approved treatments for irritability in children with ASD (Bartram, Lozano, & Coury, 2019). Both risperidone and aripiprazole are mixed dopamine-receptor and serotonin-receptor antagonists or partial agonists (Politte & McDougle, 2014) that help improve irritability and agitation symptoms in children with ASD by blocking these receptors. However, both drugs can also cause adverse events, including sedation and weight gain (Anagnostou et al., 2016) which in turn increase the risk of further health problems in later life.

In a recent systematic review and meta-analysis of three studies, Maneeton, Maneeton, Putthisri, Suttajit, Likhitsathian, and Srisurapanont (2018) concluded that aripiprazole also has efficacy in the treatment of behavioural disturbances in children with ASD, including irritability, hyperactivity/noncompliance, inappropriate speech and stereotypic behaviours found in children with ASD. McKinney and Renk (2011) suggested that such atypical antipsychotic medications ought only to be considered when behavioural interventions have been tried and failed and when the physical risks associated with disruptive behaviour exceed the risk of harm from medication and its' side effects.

3.1.2 Central Nervous System Stimulants

Central nervous system stimulants or psychostimulants are used to treat the comorbidity of ADHD often present in children with ASD. Psychostimulants such as methylphenidate and amphetamines have been demonstrated as effective treatments in reducing irritability and aggression in children with ADHD (Stuckelman et al., 2017) and, as such, they are also beneficial in managing these same symptoms in people with ASD (Davis & Kollins, 2012). While amphetamine-

derived psychostimulants increase irritability (whilst decreasing aggression), methylphenidate derivatives significantly decrease the risk of irritability (Stuckelman et al., 2017).

Nickels, Katusic, Colligan, Weaver, Voigt, and Barbaresi (2008) studied the impact of psychostimulants on a sample of 65 children with ASD and found that these medications can improve the target symptoms of hyperactivity, impulsivity, disinhibition and inattention. McCracken et al. (2014) studied the impact of methylphenidate on hyperactivity in 58 children ASD. The children with ASD were randomised to either a placebo or one of three different dose levels of methylphenidate during a 4-week blinded, crossover study. Although 14 children discontinued the study because of methylphenidate side effects, they found that methylphenidate was able to reduce hyperactivity in individuals with ASD. They concluded that ADHD symptoms are common in ASD and methylphenidate remains the best empirically validated treatment for reducing this hyperactivity although participants' variation in response to and tolerability of methylphenidate is large (McCracken et al., 2014).

3.1.3 Antidepressants

Currently, there are no antidepressant agents approved by any regulatory body for the treatment of ASD, although antidepressants, specifically Selective Serotonin Reuptake Inhibitors (SSRIs) are widely prescribed to ASD patients (Scahill & Boorin, 2011). These medications are typically employed when a child with ASD develops symptoms of depression, anxiety or obsessive-compulsive disorder (Baribeau & Anagnostou, 2014), however, results for children with ASD using these drugs are mixed.

The Cochrane Collaboration published an updated systematic review of antidepressant use in children with ASD (Williams, Brignell, Randall, Silove, & Hazell, 2013). They reviewed four Serotonin Selective Reuptake Inhibitors (SSRIs): fluoxetine, fluvoxamine, fenfluramine and citalopram. They found that SSRIs were prescribed for the treatment of conditions often comorbid with ASD such as depression, anxiety and obsessive-compulsive behaviours (Williams et al., 2013) but not for the

symptoms of ASD themselves. The authors concluded that there was not sufficient evidence for the efficacy of SSRIs in children with ASD and also limited evidence for the effectiveness of SSRIs in adults with ASD. Some smaller studies on the other hand, have reported improvements in children with ASD who were prescribed an SSRI (e.g., Branford, Bhaumik, & Naik, 1998; Buchsbaum et al., 2001; Hollander et al., 2012).

For example, Hollander et al. (2005) showed that fluoxetine was superior to a placebo in reducing children's repetitive behaviours and it was generally well tolerated by them. In their study, 45 children or adolescents with ASD were randomised into two acute 8-week phases in a double-blind placebo-controlled crossover study (Hollander et al., 2005). Although the sample size was small, the effect of fluoxetine was marginally superior to placebo. The results provided justification for further testing of fluoxetine in this population. Any such future testing will also need to ensure that neither social desirability nor a placebo effect have an impact on the final results. A larger sample size could be used to overcome these problems.

3.1.4 Adrenergic Agents

Agents which act on alpha- and beta-adrenergic receptors have been considered as possible treatments for both hyperactivity and irritability/aggression in patients with ASD (Baribeau & Anagnostou, 2014). Among these, alpha-2 adrenergic receptor agonists are used to reduce aggressive behaviour, sleep disturbances and anxiety symptoms which are present or are prominent comorbidities in children with ASD (Sharma et al., 2018). Guanfacine, another alpha-2 agonist which is FDA approved for treatment of ADHD in children has also been investigated for use in children with ASD (Baribeau & Anagnostou, 2014).

Handen, Sahl, and Hardan (2008) carried out a small placebo controlled cross-over trial of 3 mg/day of guanfacine, which showed reductions in children's levels of hyperactivity although three of the nine children with ASD showed drowsiness as a side effect. It is important therefore to monitor the dosage of this and other medication in these children.

Clonidine is another alpha-2 agonist which has been used to treat children with ASD. A small placebo controlled cross-over study was used to investigate the effect of clonidine on hyperactivity in eight male children with ASD (Jaselskis, Cook, & Fletcher, 1992). The results showed modest reductions in hyperactivity but as with guanfacine, the side effects included drowsiness as well as decreased activity levels. Again, monitoring of dosage is an important part of treatment so that the side effects of any medication do not outweigh any potential benefits.

3.1.5 Hormones

Hormones like oxytocin are also used in the treatment of the symptoms of ASD. Oxytocin is both a peptide hormone and a neuropeptide. Studies with adolescents and adults with ASD have revealed increased eye contact, increased saliency and learning around social stimuli, and better scores on tests of social cognition in response to treatment with intravenous or intranasal oxytocin (Baribeau & Anagnostou, 2014).

In one recent study, Bernaerts, Boets, Bosmans, Steyaert, and Alaerts (2020) used a double-blind, randomized, placebo-controlled, parallel design to study the effects of four weeks of intranasal oxytocin treatment (24 International Units were administered once daily in the morning) in 40 adult men with ASD. This study had two follow-up sessions, the first at four weeks and the second 1-year post-treatment. The authors concluded that no treatment-specific improvements were evident in terms of core social symptoms however, they observed some long-term beneficial effects in the reduction of repetitive behaviours and feelings of avoidance which were suggestive of the therapeutic potential of oxytocin in the treatment of ASD. Yamasue et al. (2018) found similar results in their randomized, parallel-group, multicentre, placebo-controlled, double-blind trial of 106 males (18-48 years old) with high-functioning ASD. While they also concluded that oxytocin was not recommended for the treatment of the core social symptoms of high-functioning ASD in adult men, they agreed that it could be used to treat repetitive behaviours in individuals with ASD. A meta-analysis of 16 randomised controlled trials comprising 520 individuals with ASD by Wang, Wang,

Rong, He, and Yang (2019), reported that oxytocin had a small but not-significant effect on participants social functioning.

The initial results with oxytocin have been encouraging and many clinical trials are currently underway (Guastella & Hickie, 2016; Higashida, Munesue, Kosaka, Yamasue, Yokoyama, & Kikuchi, 2019) to test further oxytocin in individuals with ASD across age ranges, with results anticipated over the next several years. In addition to these pharmacological treatments, several non-pharmacological interventions are utilised when working with children with ASD.

3.2 Non-Pharmacological Treatments

Typical non-pharmacological treatments include speech and language therapy, occupational therapy, and behavioural interventions, which can be administered in a variety of settings including clinics, schools, and in the home (Nguyen, Krakowiak, Hansen, Hertz-Picciotto, & Angkustsiri, 2016). Given the range of skills associated with spoken-language development, the majority of these interventions have taken one of two approaches to improve spoken-language: either a targeted or a comprehensive approach (Hampton & Kaiser, 2016).

Not all children with ASD will benefit from the same approaches (Lord et al., 2018). This difference in impact is due to individual child related factors. For example, although most children first learn words receptively (that is, productive language) and then expressively (that is, being able to express oneself), children with substantial delays might first learn words by saying them and only later come to understand their meaning (Woynaroski, Yoder, & Watson, 2016). Different types of therapies including both targeted and comprehensive treatments are discussed next.

3.2.1 Targeted Speech Therapies

Numerous intervention methods and frameworks have been developed to target communication and language impairments in individuals with ASD and to help improve these core symptoms of ASD (Manwaring & Barber, 2019). Interventions that specifically teach speech,

receptive/productive language, pre-linguistic communications or social uses of communication are considered targeted speech therapies (Hampton & Kaiser, 2016). Targeted speech therapies play an essential role in the treatment of individuals with ASD (Manwaring & Barber, 2019) and in the design and implementation of treatment programs for them (Manwaring & Barber, 2019). Method, dose, timing of the intervention, and content have been recommended as the four key factors that are likely to influence the outcomes of speech therapy (Kasari, Freeman, Paparella, Wong, Kwon, & Gulsrud, 2005).

Method refers to the approach used for delivery. The method is the framework designed to determine which interventions are most effective for the individual child's characteristics (Sherer & Schreibman, 2005). Targeted methods include the Picture Exchange Communication System (PECS) (Bondy, & Frost, 1994; Liddle, 2001), the use of speech generating devices (Charlop & Gumaer, 2019; Roche et al., 2014; Schlosser & Koul, 2015), self-management (Koegel, Park, & Koegel, 2014), Reciprocal Imitation Training (RIT) (Ingersoll & Schreibman, 2006) and typical speech therapies. These interventions are designed to enhance functional communication, emotion recognition, social skills, and to promote independence (Lai et al., 2014).

A dose of 25 hours per week of intervention with active engagement has been recommended in order to maximise positive outcomes (Manwaring & Barber, 2019; National Research Council, 2001). Timing of the intervention refers to the child's age at which the intervention begins and it is well established that earlier interventions lead to better intervention outcomes (Fein, Barton, & Dumont-Mathieu, 2017). Children with ASD who started their intensive behavioural program before age 4, 3, or even 2 years of age realised gains directly related to their age of intervention onset, with especially impressive gains in the youngest group (Fein et al., 2017). Furthermore, the largest effects are found when both parents and the clinician implement the intervention (Hampton & Kaiser, 2016) leading to consistency of application for the child.

The content of intervention refers to what is being taught, and how this relates to outcomes (Manwaring & Barber, 2019). For an intervention to be effective, the content needs to target pivotal areas, that is, areas that when changed will potentially bring about improvements in the targeted as well as, potentially, other areas of the child's life (Koegel, Ashbaugh, & Koegel, 2016). The pivotal areas that have been found to improve a child's response to treatment include pre-treatment levels of spoken language, object exploration, joint attention, and receptive language (Gordon, Pasco, McElduff, Wade, Howlin, & Charmanet, 2011; Paul, Campbell, Gilbert, & Tsiouri, 2013; Sherer & Schreibman, 2005; Yoder & Stone, 2006). These four factors constitute what is currently considered a typical treatment plan for children with ASD.

Once a treatment plan is designed and implemented, the child's progress is monitored. If the child is not progressing or not responding to a particular intervention method, treatment must be adjusted appropriately (DiStefano & Kasari, 2016). For non-verbal individuals, the Picture Exchange Communication System (PECS), a graphics based system, could be helpful at least in the short term (Maglione, Gans, Das, Timbie, & Kasari, 2012). A PECS approach is supported by the strong visual processing ability seen in many children with ASD (da Silva, Fernandes, & Grohmann, 2014). Augmented communication based approaches such as Speech Therapy with Augmented Reality (STAR) which focuses on communication improvement have also been shown to be effective in interventions for children with ASD (da Silva, Fernandes, & Grohmann, 2014).

In a recent systematic review of 17 studies on school based language interventions for children with ASD, Sedgwick and Stothard (2018) found that in nine studies significant positive effects were reported. The authors stressed that improvements in children's general language skills/specific vocabulary acquisition stemmed from sessions conducted by highly trained therapists which were reinforced by the child's adherence to the program.

3.2.2 Comprehensive Interventions

The comprehensive interventions are designed to cover a broad set of skills that are directly or indirectly related to spoken-language development (Hampton & Kaiser, 2016). These programs may include activities to target cognitive skills, motor skills, self-help skills, play, imitation, receptive language, with some focus on productive language skills (Hampton & Kaiser, 2016). Typically, these interventions combine speech and behavioural therapies. These treatments are time intensive (Rogers & Vismara, 2008) and are highly structured in nature, given over periods of 15-20 hours or more per week (Lord et al., 2018). The benefits of these intensive therapies have been shown to last beyond the child's early years into later childhood and adulthood (Pickles et al., 2016).

Landa (2018) reviewed the studies on early comprehensive interventions for children with ASD younger than 5 years of age published in the last 15 years. The author concluded that young children with ASD benefit from early interventions. Furthermore, when these interventions involved a 'parent-coaching' component, the parents learnt to implement child-responsive engagement strategies outside session times. The evidence supporting the combination of parent mediated and direct clinician implemented interventions to maximise the child's developmental gains comes from several studies, including Green et al. (2010), Oono, Honey and McConachie (2013), Pickles et al. (2016), and Landa (2018).

Additionally, the highly structured and predictable nature of these programs render them highly appropriate not only to target the core symptoms of ASD but also comorbid symptoms such as anxiety and depression (Hesselmark et al., 2014). However, these therapies do not show similar positive results at low-intensity/frequency (Carter, 2011; Rogers & Dawson, 2010), which highlights the importance of more therapy hours per week.

Many of these programs and approaches use cognitive behavioural therapy (CBT) as their underlying theoretical framework (Lord, Elsabbagh, Baird, & Veenstra-Vanderweele, 2018). Cognitive behavioural psychotherapeutic interventions have been shown to be effective for children with ASD

(Hesselmark, Plenty, & Bejerot, 2014) targeting a range of comorbid psychiatric disorders often present in children with ASD, such as anxiety, depression and OCD (Sharma, Gonda, & Tarazi, 2018). The most well-known form of behavioural treatment for children with ASD is Applied Behaviour Analysis (ABA) and there are many versions of this approach (Lord et al., 2018).

ABA interventions are recognized as the most effective evidence-based interventions for children with ASD (Fein et al., 2013). These interventions support a child with ASD in at least five ways: 1) to teach new skills (e.g., systematic instruction and reinforcement procedures to teach functional life skills, communication skills, or social skills), 2) to reinforce and maintain previously acquired skills, 3) to generalize behaviour from one situation to another (e.g., teaching and transferring social skills to natural settings), 4) to restrict or narrow conditions under which interfering behaviours occur (e.g., modifying the learning environment; antecedent modification), and 5) to reduce interfering behaviours by discontinuing their reinforcement and reinforcing competing replacement behaviours (Steege, Mace, Perry, & Longenecker, 2007).

The origins of ABA can be traced to the University of California at Los Angeles Young Autism Project that Lovaas (1987) developed and ran in the 1980s. Hayward, Eikeseth, Gale, and Morgan (2009) conducted a study to examine progress after 1 year of ABA treatment in children with ASD who received an average of 36 hours per week of one-on-one therapy at the University of California's Los Angeles Applied Behaviour Analysis (UCLA ABA) program. Two types of service provision were compared: an intensive clinic based treatment model with all treatment personnel (N = 23), and an intensive parent managed treatment model with intensive supervision only (N = 21). There were no significant differences between the two groups on any of the measures at follow-up. Between intake and follow-up, children in both groups improved significantly on IQ, visual-spatial IQ, language comprehension, expressive language, social skills, motor skills and adaptive behaviour (Hayward, et al., 2009). These results highlight the importance of family participation in programs

designed to assist children with ASD. Such familial participation requires the parents to attend training and be dedicated in its application.

Other similar interventions include Pivotal Response Treatment (PRT), Joint Attention Symbolic Play and Engagement Regulation (JASPER), Early Social Interaction (ESI) (Schreibman et al., 2015), Early Start Denver Model (ESDM) (Dawson et al., 2010), Responsive Teaching (Mahoney & Perales, 2005), and the Developmental Individual-Difference Relationship-based model (DIR/Floortime) (Greenspan & Wieder, 1997). These treatments differ from one another but share similarities in that they all follow typical developmental sequences and protocols such as they emphasise play, social interactions, and communicative initiation on the part of the child, and the natural consequences of the child's behaviours or actions as opposed to using rewards such as food (Lord et al., 2018). The other skills they focus on include cognitive skills, motor skills, self-help skills, play, imitation, receptive language and limited productive language skills development (Hampton & Kaiser, 2016).

Each of these programs comprise a core element of psychoeducation in combination with social coaching to enhance development of social skills, instruction on self-care skills, and highly structured worksheets and visual aids (Ung, Selles, Small, & Storch, 2015). Development of these skills are addressed by taking into consideration the difficulties the person experiences as a result of ASD such as problems in recognising and understanding thoughts and feelings (Danial & Wood, 2013). These programs are also designed to build the cognitive capacities of children with ASD, in particular their abilities to understand other people's cognitive and mental states as well as to build the social skills required for reciprocal interactions (Sharma et al., 2018).

Comprehensive interventions tend to be more time intensive than speech therapies alone because of the increased number of goals and proposed outcomes (Rogers & Vismara, 2008). These treatments are usually intended to be given intensively in periods of 15-20 hours or more per week (Lord et al., 2018). Comprehensive therapies can be administered individually, in group settings or

can involve parents/family members (Gates, Kang, & Lerner, 2017; Sharma et al., 2018). The increased flexibility and personalisation of these interventions to meet the specific needs and skill deficits of each individual make these individual treatments highly effective (White, Oswald, Ollendick, & Scahill, 2009).

Group based interventions also have specific benefits such as facilitating increased social interactions; sharing of experiences, promotion of self-acceptance and improved insights of both the strengths and impairments related to ASD among participants in the group (Gates et al., 2017; Hesselmark et al., 2014). White, Ollendick, Scahill, Oswald, and Albano (2009) introduced a group therapy component into their therapeutic intervention targeting anxiety and social competence. They utilised this group therapy version with four adolescents with ASD. The therapy was delivered over the course of approximately 11 weeks and helped to improve social skills in all four participants as well as reducing anxiety in three of the four participants (White et al., 2009).

Numerous studies have shown beneficial results from comprehensive therapies. Weston, Hodgekins, and Langdon (2016), in a review of 48 studies, found that these therapies led to significantly improved cognition, affective communication, facial emotion perception and social skills in children with ASD. Meta-analyses of interventions for children with ASD indicate that there is some evidence that these interventions improve language outcomes (Hampton & Kaiser, 2016). A meta-analysis of studies examining early intensive behavioural interventions by Reichow, Barton, Boyd, and Hume (2012) identified reductions in ASD symptomatology and improvement in overall language outcomes for children. Additionally, Reichow et al. (2012) reviewed five studies all of which identified early intensive behavioural interventions as an effective program for young children with ASD. Each of these programs in these five studies resulted in improved communication for participants compared with children with ASD enrolled in a special education setting. Early intensive behavioural therapies, for children under five years old, which decompose complex skills into more elementary subskills and teach these subskills individually, have increased intellectual functioning in

50% of young children with ASD (Cohen, Amerine-Dickens, & Smith, 2006; Sallows & Graupner, 2005).

An important aspect of these therapies is their capacity to concurrently treat comorbid disorders such as anxiety (Sharma et al., 2018). This comorbid anxiety often leads to or exacerbates several other problems including irritability, disruptive behaviours, inattention, and decreased functionality (Ung et al., 2015). Meta-analyses of studies which have used these therapies to treat the symptoms of anxiety in children with ASD have reported these therapies were effective in reducing anxiety in these children (Kreslins, Robertson, & Melville, 2015; Sukhodolsky, Bloch, Panza, & Reichow, 2013; Ung et al., 2015). A number of group therapy programs have also shown successful reduction of anxiety symptoms in children with ASD, usually when parent groups are conducted in parallel with the children's therapy groups (McConachie et al., 2014: Wood, Drahota, Sze, Har, Chiu, & Langer, 2009).

3.2.3 Occupational Therapy

Occupational therapy (OT) is used with children with ASD to enhance their fine motor and adaptive skills, including self-care, manipulation of toys, and handwriting (Hyman, Levy, & Myers, 2020) and to improve sensory and even gross motor skills in the short term compared with usual care in young children with ASD (Weitlauf, Sathe, McPheeters, & Warren, 2017). It is important to improve gross motor skills in children with ASD as impaired motor skills can impede social skills development and active learning and may also be a risk factor for overweight and obesity (Srinivasan, Pescatello, & Bhat, 2014). Toe walking, where children literally walk on the balls of their feet, with no contact between the heels and ground, is common among children with ASD and interventions to address this include passive stretching, orthotics and, in extreme cases, the use of casts (Hyman et al., 2020).

Sensory-oriented treatments are a considerable part of early interventions delivered via occupational therapists. These therapies are sometimes offered as one component of a

comprehensive program to address sensory-based problems (Lai et al., 2014). Various sensory-related components which have been used as adjuncts to OT practices such as weighted blankets (to help with sleep), swings (for sensory therapy), or brushing (as a part of the sensory therapy program) have not shown consistent positive effects (Silva, Schalock, Gabrielsen, Budden, Buenrostro, & Horton, 2015) although there may be some benefits for individuals.

Bilaver, Cushing, and Cutler (2016) examined the prevalence and correlates of educational intervention utilisation among preschool aged children with ASD in the United States via a nationally representative longitudinal survey. They found that 65% of the children with ASD included in their survey, were reported to be engaged in occupational therapy. They did not look at the results of therapy interventions in their study as this was not their focus.

Pfeiffer, Koenig, Kinnealey, Sheppard, and Henderson (2011) examined the impact of sensory integration and fine motor interventions based on occupational therapies in children with ASD aged 6-12 years. The children were randomly assigned to either a fine motor or a sensory integration treatment group. Pre-tests and post-tests measured social responsiveness, sensory processing, functional motor skills, and social-emotional factors. All participants received 18 treatment interventions of 45 minutes each over a 6-week period, except for one child who missed one treatment session. Results identified significant positive changes in scores on Goal Attainment Scaling for both groups: the sensory integration group also showed a significant decrease in autistic mannerisms.

Linderman and Stewart (1999) investigated the effects of home-based sensory integrative-based occupational therapy on the functional behaviours of two 3-year-old boys with ASD. The treatment phase was 11 weeks for Participant 1 and 7 weeks for Participant 2. Both boys displayed significant improvements in the areas of social interaction, approach to new activities, response to holding or hugging, and response to movement. Additionally, the authors noted a decrease in the frequency and duration of disruptive behaviours (e.g., aggressive behaviours), with an increase in

functional behaviours such as spontaneous speech, purposeful play, and attention to activities and conversation in both boys. However, Participant 2's functional gains appeared minimal compared with Participant 1 which might have been a function of the program's duration. The authors also noted that concurrent interventions that were not part of the study (e.g., initiation of speech therapy, preschool, vitamins) may have confounded these results. Occupational therapies, in conjunction with speech therapies and physical therapies, play an important role in early identification and intervention to address sensorimotor and social skills of high risk infants (Flanagan, Landa, Bhat, & Bauman, 2012) and children with ASD.

3.2.4 Physical Therapy

Effectively addressing the motor-related impairments of children with ASD is crucial, therefore access to early physical therapy interventions is paramount (Atun-Einy, Lotan, Harel, Shavit, Burstein, & Kempner, 2013). Holloway, Long, and Biasini (2018) found that motor skills and social function are related in young boys with ASD and they recommended increasing physical therapy interventions for children with ASD with the aim of correspondingly increasing social functioning. Aside from engagement in occupational therapy discussed above, Hartshorn, Olds, Field, Delage, Cullen, and Escalona, (2001) conducted a study in which they found a significant increase in attentive behaviours and a subsequent decrease in stress behaviours following physical activity involving dance movements in children with ASD. Torrance (2003) also reported a decrease in violent outbursts following a dance based therapy in children with ASD. In a systematic review of 13 studies, Bremer, Crozier, and Lloyd (2016) found that jogging, horseback riding, martial arts, swimming, yoga and dance all resulted in improvements in numerous behavioural outcomes among children with ASD. Among these improvements were reductions in stereotypic behaviours and enhanced levels of social-emotional functioning, cognition and attention. In sum, physical activity has numerous benefits for children with ASD, but they may also benefit from structured activities or exercise although neither physical activity nor exercise are mutually exclusive.

3.2.5 Exercise

Exercise may be defined as 'physical activity that is planned, structured, repetitive, and purposeful' (Caspersen, Powell, & Christenson, 1985). Exercise differs from other physical activities which can, for example, be incidental walking or other largely unplanned activities. Researchers have noted several health benefits of exercise in children with ASD. As a result of exercise, children with ASD are reported to achieve increases in desired behaviours (Petrus, Adamson, Block, Einarson, Sharifnejad, & Harris, 2008), decreases in inappropriate behaviours (Celiberti, Bobo, Kelly, Harris, & Handleman, 1997; Dillon, Adams, Goudy, Bittner, & McNamara, 2017), improved academic achievements (Nicholson, Kehle, Bray, & Heest, 2011), enhanced motor skills (Colombo-Dougovito, & Block, 2019) and it also had beneficial effects on metabolic indicators such as high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and total cholesterol (Toscano, Carvalho, & Ferreira, 2018). In one study, 15 minutes of running/jogging followed by a classroom task was sufficient to show improvement (e.g., task time and correct responses) in these children (Oriel, George, Peckus, & Semon, 2011). Consistent with findings in older children, these results indicate that aerobic exercise prior to classroom activities may improve academic responding in young children with ASD (Oriel et al., 2011). These activities can be part of the regular outdoor activities of all children in the school so that the child with ASD is not singled out.

Healy, Nacario, Braithwaite, and Hopper (2018) carried out a meta-analysis of 29 studies on the effects of exercise (physical education, sports, walking, hiking and playing at a playground) on youth with ASD. Their results showed that these activities had moderate to large effects on a variety of outcomes including the development of manipulative skills, locomotor skills, skill-related fitness, social functioning, and muscular strength and endurance. Clearly, exercise of many types is beneficial to the social, cognitive and health wellbeing of children with ASD. Music therapy is also emerging as an early intervention program to improve the quality of life of the children with ASD and indirectly their parents (Thompson, 2018; Thompson & McFerran, 2015).

3.2.6 Music Therapy

Children with ASD generally show a preserved and even heightened sense of musicality together with an ability to interpret and respond to the emotions conveyed by song or music even when unable to do so in speech (Heaton, 2009). This heightened sense of musicality extends well into adulthood (Heaton, 2009) and this ability or sense can be used to improve symptoms and social skills in the children with ASD.

Systematic reviews reveal that music therapy is very effective in improving developmental aspects such as social interaction, including nonverbal and verbal communicative skills (Rossignol, 2009; Wheeler et al., 2008), social-emotional reciprocity, social adaptation, joy, and the quality of the parent-child relationship (Geretsegger, Elefant, Mössler, & Gold, 2014). While there is little research on how music therapy affects and improves the skills of the children with ASD (Mössler et al., 2019), it does seem that music is a universal language that seems to "speak" to children with ASD and it can be implemented at convenient times and with little cost.

3.2.7 Animal Assisted Therapy

One type of complementary and integrated therapy that is gaining popularity for children and adolescents with or at risk of mental health problems is animal assisted therapy (Hoagwood, Acri, Morrissey, & Peth-Pierce, 2017). Animal assisted therapies are goal-directed and structured interventions that incorporate animals for the purpose of therapeutic gain in humans (Kamioka et al., 2014).

Animal assisted therapy programs have shown significant improvements in children with ASD, in their social functioning, distractibility and sensory seeking and sensory sensitivity (Bass, Duchowny, & Llabre, 2009), emotional functioning and physical functioning (Lanning, Baier, Ivey-Hatz, Krenek, & Tubbs, 2014), adaptive and motor skills (Gabriels et al., 2012), irritability, hyperactivity, social cognition, communication, and verbal communication (Gabriels, Pan, Dechant, Agnew, Brim, & Mesibov, 2015). All of the assisted therapies used in those studies were equine

based where children with ASD participated in horse-back riding (Bass et al., 2009; Gabriels et al., 2012; Gabriels et al., 2015; Lanning et al., 2014) and, in some, horse grooming (Lanning et al., 2014).

Martin and Farnum (2002) evaluated the effects of the interactions between children with ASD and dogs and also found improvements in the children's verbal communication and social interactions. Other researchers (e.g., Funahashi et al., 2014; Fung, 2015) have also noted improvements in children with ASD using dog assisted therapies. For instance, Fung (2015), although reporting on only one case study, noticed an increase in the social communication of a boy with autism. Stevenson et al. (2015) noticed a reduction in solitary or repetitive behaviours in three children with ASD. Two other studies have reported positive social behaviours increased and negative social behaviours decreased in children with ASD which they attributed to dog assisted therapy (Funahashi et al., 2014; Silva, Correia, Lima, Magalhães, & de Sousa, 2011). Funahashi et al. (2014) carried out sessions where the child with ASD interacted with small dogs under loose facilitation by a therapist, while Silva et al. (2011) organised one-on-one structured activities with a therapist and dog which involved the child touching, holding, and petting the dog under supervision.

Becker, Rogers, and Burrows (2017) built on these studies and conducted social-skills training using dogs with groups of youth with ASD with positive results. Activities included having the youth with ASD co-lead a dog with the handler, petting the dog, practice asking dogs to perform basic commands, practice grooming and appropriate ways to approach and engage with the dogs (Becker et al., 2017). Carlisle (2015) investigated the impact of untrained dogs with children with ASD and found that the amount of exposure to dogs was associated with increased social skills in these children. Overall, these findings indicate that animal-assisted therapy is beneficial for improving social skills and reducing ASD related affective symptoms. It seems from Carlisle's study that even exposure to the "family dog" may be beneficial to the child with ASD. Why this is so might be related to touch, interactions, and even unconditional engagement with or regard from the dog.

3.2.8 Parent Coaching

Early intensive parent training programs, the aims of which are to coach parents on ways to interact with their children with ASD, have shown positive effects for both the children (Weitlauf et al., 2014) and parents (Sealy & Glovinsky, 2016). These interventions are designed to coach parents on how to interact with their young children with ASD and they can result in immediate positive effects on their children's social behaviour and communication level (Weitlauf et al., 2014). These therapies also increase shared positive affect between the child and parent, children's adaptability (Mazurek et al., 2017), and reduce the disruptive behaviours of the children with ASD (Bearss et al., 2015). For parents these treatments help, to some degree, to alleviate the distress of families and give them something positive on which to focus (Sealy & Glovinsky, 2016). Parent-mediated interventions provide the additional benefit of carrying out therapies in the home and community environments thereby enabling skill transfer to real-life settings, and increasing parents' and caregivers' self-confidence in teaching and supporting their child (Dawson & Burner, 2011). These parent-related activities or coaching are often utilised in conjunction with, or an extension of, clinician run sessions.

In a review of 17 studies from six countries, Oono et al., (2013) concluded that all parent mediated therapies had shown some effectiveness although the effect sizes varied in intensity and duration. Another review of 18 studies (only three of which included participants with ASD), found that parent mediated therapies for language training significantly improved expressive language, vocabulary and syntax for children with a language impairment (Roberts & Kaiser, 2011). This improvement was also present in the three studies which involved children with ASD. Hampton and Kaiser (2016), in their systemic review of 26 studies on spoken-language outcomes for children with ASD, found that treatments delivered simultaneously by a clinician and a parent have resulted in greater improvements in spoken-language than therapies delivered by a clinician or parent alone. They attributed this to three reasons: 1) parents benefitted from the clinician modelling the intervention with their children, 2) children with ASD benefitted when there was a consistent

language and teaching strategy across communication partners that supports the generalisation of skills, and 3) parent stress may reduce when the clinician and parent are co-interventionists and parents can observe improvements in their child resulting in positive child outcomes through direct and indirect paths. Green et al. (2015) worked with parents of infants aged 7-10 months with a high familial risk of autism to develop a therapist created video of parental interactions with their child. These were then used to help parents understand the child's communication. Compared to a control group, children of these parents showed positive estimates across a wide range of behavioural and brain function risk-markers and developmental outcomes consistent with a moderate intervention effect to reduce the risk for later autism. It is important to note that strategies that work for most children might not work for everyone (Woynaroski, Yoder, & Watson, 2016). Therapies tailored to the individual child with ASD yield better results.

Overall, non-pharmacological therapeutic interventions for ASD focus on promoting functional independence and enhancing quality of life by targeting the development of key skills for socialisation, communication, emotions, cognition and behaviour. Clearly strategies to assist the child's positive development will also have flow on effects to reduce the burden, and enhance the life, of families living with a child with ASD.

3.3 Chapter Summary

There is no known cure for ASD but early interventions have been designed to help reduce the impact of the symptoms of ASD and help improve the overall lifestyle and wellbeing of individuals with ASD. In this chapter, a range of pharmacological treatments were discussed which typically focus more on the comorbidities of ASD than directly on its symptoms. Caveats were drawn about potential drug side-effects and the need to monitor dosages. A range of non-pharmacological treatments, such as speech and language, occupational and behavioural interventions were reviewed and the need for early intensive intervention stressed. The innovative use of music and animals as therapy have provided encouragibg results. It was also noted that not every child benefits

from all interventions but therapiesco-delivered by clinicians and parents afford the greatest consistency for change.

Chapter 4. Psychosocial Impact on Families and Coping Strategies

Living with a child with ASD can have an immense impact on the entire family (Kamio & Inada, 2014; Lavelle, Weinstein, Newhouse, Munir, Kuhlthau, & Prosser, 2014). Research has consistently pointed to ASD as having a more deleterious impact than other childhood disorders on the wellbeing of children with the disorder (Kamio & Inada, 2014), their parents (Altiere & von Kluge 2009; Baker-Ericzén et al., 2005; Cappe et al., 2018; Eisenhower et al., 2005; Gray & Holden, 1992; Lee et al., 2009; Paynter et al., 2018; Vasilopoulou & Nisbet, 2016; Yamada et al., 2012) and also their siblings (Macks & Reeve, 2007; Rivers & Stoneman, 2003; Tomeny et al., 2017; Tsai et al., 2018; Wigston, Falkmer, Vaz, Parsons, & Falkmer, 2017). This negative impact is significantly associated with the behaviours commonly associated with ASD which are often antisocial and disruptive.

These behaviours include unhealthy obsessions and repetitive behaviours (Calderoni, Bellani, Hardan, Muratori, & Brambilla, 2014; Kim et al., 2016; Wolff, Hazlett, Lightbody, Reiss, & Piven, 2013), tantrums and self-harm (Duerden et al., 2012; Minshawi, Hurwitz, Morriss, & McDougle, 2015; Soke et al., 2016; Wolff et al., 2013), and destructive/aggressive behaviour (Mazurek, Kanne, & Wodka, 2013; Hill et al., 2014; Kaat, & Lecavalier, 2013), which often reach extreme levels (Bauminger et al., 2010; Carroll et al., 2014; Carter Leno et al., 2018; Ghayan, A., & Ghayan, S., 2014; Yang et al., 2017). The management of these children's behavioural problems is compounded by their communication deficits (Brignell et al., 2018; Øien et al., 2016; Tager-Flusberg & Kasari, 2013; Zeedyk et al., 2014), dietary inflexibilities (Marquenie et al., 2011; Ooi, Ong, Jacob, & Khan 2016), sleep disorders (Liu et al., 2006; Marquenie et al., 2011; Reynolds et al., 2017; Richdale & Schreck, 2009; Richdale & Schreck, 2019; Tyagi et al., 2018) and sensory issues (Addo et al., 2017; DuBois et al., 2017; Kumazaki et al., 2018; Larsson et al., 2017; Linke et al., 2018; Rogers & Ozonoff, 2005) such as hypersensitivity and lack of multisensory integration (Addo et al., 2017) associated with ASD. These characteristics and behaviours disrupt all areas of family life (Khanna et al., 2011; Kuhlthau et

al., 2014; Tung et al., 2014) creating multiple challenges for both parents and siblings and leading to a range of negative outcomes for the family.

These behaviours and deficits related to the child with ASD have an impact on the social life of the family and, at times, the parent's relationship with each other. Parents report a scarcity of time for themselves as a couple (Hock, Timm, & Ramisch, 2012; Saini et al., 2015; Sim, Cordier, Vaz, & Falkmer, 2016), and for family activities, as well as a lack of spontaneity generally due to the stressors associated with raising a child with ASD (Hutton & Caron, 2005). Parents of children with ASD also report a constant need for vigilant parenting (Karst & van Hecke, 2012). It has been estimated that they spend 1,000 hours more per year in the care of their child with ASD than the parents of normally developing children (Järbrink, 2007).

A major impact of these additional child care hours and demands is a restriction on parents' ability to engage in paid employment (Fletcher, Markoulakis, & Bryden, 2012; Matthews, Booth, Taylor, & Martin, 2011; Sitimin, Fikry, Ismail, & Hussein, 2017) which, in conjunction with greater investment in health care (Fletcher et al., 2012; Järbrink, 2007; Karst & van Hecke, 2012), exacerbates the family's financial burden. These financial restraints may also limit family holidays and educational opportunities, as well as the purchase of material goods (Dyke et al., 2009). Furthermore, families often experience embarrassment and ostracism by others in public situations as well as from their peers due to their child's antisocial behaviours (Dyke et al., 2009). These experiences can act to further restrict the family's social life.

Zimmerman, Ownsworth, O'Donovan, Roberts, and Gullo (2016) carried out a systematic review of 27 studies on factors related to psychosocial outcomes for the family of a child with ASD. They found that having a child with ASD had an impact on family members' mental health, namely depression, anxiety, stress, self-esteem, and their social and adaptive functioning such as reduced levels of independence, vocational, academic, and interpersonal functioning. To address these formidable psychosocial challenges faced by the families of children with ASD, there is an emerging

body of work which recognises the importance of understanding the complex impact that living with a child with ASD has on families and family adaptation (Cridland, Caputi, Jones, & Magee, 2014; Morgan, 1988).

Family adaptation refers to the family's ability to manage change and stressors (Olson, Sprenkle, & Russel, 1979) which are considered ongoing for the family of a child with ASD. The extant literature shows families living with a child with ASD engage in a continuous process of adaptation throughout their child's development (Karst & van Hecke, 2012; Manning, Wainwright, & Bennett, 2011) with variability in the family's coping strategies and levels of social support linked to individual differences in outcomes (McStay et al., 2014; Paynter et al., 2013). Understanding variables related to these outcomes is important when developing support strategies for families as well as to support outcomes for the children affected (Pepperell et al., 2016).

The availability of social support is argued to moderate the impact of high levels of stress on psychological wellbeing (Cohen, 2004) and an individual's perception of the extent of the availability of this support is a fundamental aspect of its' buffering effect on outcomes (Cohen, 1988). Research has found that the quality of social support and its' perceived availability moderates the influence of high levels of stress on health outcomes in a range of clinical populations including the families living with a child with ASD (Payne et al., 2012; Steptoe, 2000). It is important therefore, that families living with a child with ASD are supported by the wider family, friends and the community generally.

4.1 Impact of the Child's Behaviours on Families

The pervasive and severe deficits often present in children with ASD are associated with an increase in the mental and physical health problems of their parents (Karst & van Hecke, 2012) and the more severe these behavioural problems, the greater the relationship with caregivers' psychological and physiological health (Lovell, Moss, & Wetherell, 2015; Shepherd, Landon, & Goedeke, 2018). Attempts to manage the child and their behaviour can lead to higher fatigue among caregivers (Giallo, Wood, Jellett, & Porter, 2013) as well as elevated levels of stress (Shepherd,

Landon, Taylor, & Goedeke, 2018). Parents of children with ASD also report significantly higher levels of parental anxiety and depression than parents of typically developing children (Padden & James, 2017; Yirmiya & Shaked, 2005) and the symptoms of ASD are positively associated with parental depressive and anxiety symptoms (Chan, Lam, Law, & Cheung, 2018). Parenting a child with ASD who has intermittent outbursts of aggression exposes parents and other family members to chronic hypervigilance and psychological distress in their attempts to avoid potential physical threats (Swaab, McCormack, & Campbell, 2017).

Cohrs and Leslie (2017) conducted a study using the MarketScan database of 42,649 children with ASD and an equal number of matched controls which resulted in a sample of 85,298 unique families for the study. Of the 42,649 families with a child with ASD, 1,533 (3.6%) reported they had more than one child with ASD. Cohrs and Leslie's analyses revealed that mothers of children with ASD were at 2.9 times greater odds of having a diagnosis of depression than the mothers of children who did not have ASD: the fathers of children with ASD had a 2.4 times greater odds of having a diagnosis of depression than the fathers of children not diagnosed with ASD. They also found that the odds ratio for depression increased when there was more than one child with ASD in the family (Cohrs & Leslie, 2017).

The characteristics and behaviours of the child with ASD disrupt all areas of family life (Khanna, Madhavan, Smith, Patrick, Tworek, & Becker-Cottrill, 2011; Kuhlthau et al., 2014; Tung et al., 2014) creating multiple challenges for parents and leading to a range of negative outcomes including scarcity of time for the parents as a couple (Hock et al., 2012; Saini et al., 2015; Sim et al., 2016), lack of time for broader family activities, as well as a lack of spontaneity for leisure time activities due to the stressors associated with raising a child with ASD (Hutton & Caron, 2005).

Researchers have indicated that the parents of children with ASD are vulnerable to relationship stress, lower marital satisfaction, and potential relationship dissolution (Ramisch, Timm, Hock, & Topor, 2013). Papp and Hartley (2019), in their study of 174 parents with a child with ASD

and a comparison group of 179 parents with a child without a neurodevelopmental disability, found that increased marital conflict, lower dyadic positivity and higher dyadic anger, were reported more by both the mothers and fathers of a child with ASD than either parent of typically developing children. Hartley et al. (2010) compared the occurrence and timing of divorce in 391 parents of children with ASD and a matched representative sample of parents of children without disabilities. They found that the prevalence of divorce was significantly higher for couples with children with ASD than the comparison group (23.5% vs. 13.8%). The rate of divorce for parents of children with ASD was spread across the child's lifespan while for the comparison group, the rate of divorce was highest when the child was less than eight years (Hartley et al., 2010). This finding suggests that all couples experience relationship difficulties following the birth of a child and its impact on the couple. However, for couples with a child with ASD these difficulties and challenges are lifelong, putting a continuous strain on the relationships.

Other direct impacts related to behavioural severity in the child with ASD include increased parental stress in managing activities such as bathing, toileting, dressing or eating (Shepherd, Landon, & Goedeke, 2018), as the child with ASD requires physical support in all everyday tasks. Greater symptom severity has a negative impact on the child's ability to self-care and perform other tasks of daily living which leaves the responsibility of simple caregiving tasks to the parents of the child with ASD (Shepherd et al., 2018). The child's behavioural problems can also have an indirect impact on parents. For example, parents of children with ASD are often called to the school due to their child's behavioural problems (Myers, Mackintosh, & Goin-Kochel, 2009). These problems may involve barricading themselves in rooms with other children, setting off fire alarms and, in extreme instances, destroying school property resulting in the school staff calling the police (Brede, Remington, Kenny, Warren, & Pellicano, 2017). Some further examples of parents being called to school for property destruction include pulling things off the wall and destroying other students' school materials (Farrugia, 2009).

Parents are often apprehensive to take their child with ASD to the majority of public places due to the fact that crowds and noise often precipitate negative behaviours in the child, thus forcing families to spend a great deal of time at home (Myers et al., 2009). Temper tantrums, high pitched screams and the child running away from parental control in public places are some of the behaviours that lead to parents' decisions to avoid public places (Myers, Mackintosh, & Goin-Kochel, 2009). In fact, the behaviours of a child with ASD can lead to the social isolation of the family from friends, school, the public, and in some cases, health care providers (Gorlin, McAlpine, Garwick, & Wieling, 2016).

As discussed earlier, sleep disorders are common in children with ASD (Klukowski et al., 2015; Rossignol & Frye, 2011) and Miano, Giannotti, and Cortesi (2016) reported this to be the case in up to 50% of these children. The exhaustion of both the child and the parent is associated with the child's demands for care during the night (Myers et al., 2009). The unrelenting stress derived from what is often a chronic lack of sleep (Gorlin et al., 2016) has an enormous impact on parents. This effect is especially noticeable when parents are unable to sleep more than an hour or two during the night (Myers et al., 2009) and this lack of sleep or fatigue subsequently affects their overall wellbeing and can lead to drastically declining health (Myers et al., 2009). The wakefulness of the child with ASD also disrupts siblings' sleep (Myers et al., 2009). In fact, the behavioural problems of the child with ASD have been found to increase depressive symptoms in siblings as well as the effect they have on parents (Lovell & Wetherell, 2016). Parents have also noted physical violence and assaults by the child with ASD towards their siblings and even self harm (Myers, Mackintosh, & Goin-Kochel, 2009). Overall, the emotional and behavioural difficulties of the child with ASD can lead to lower caregiver mental wellbeing (Salomone et al., 2018).

4.1.1 Lack of Time

Parents report having little time for family activities as well as a lack of spontaneity to engage in activities, due to the stressors associated with raising a child with ASD (Hutton & Caron, 2005).

Parents of the child with ASD report a constant need for vigilant parenting (Karst & van Hecke, 2012) due to behavioural problems such as intermittent outbursts of aggression (Swaab et al., 2017), running away (Myers et al., 2009) and their child having little or no understanding of danger and safety. A great deal of time (Järbrink, 2007) and energy are required to care for the child with ASD (McCann, Bull, & Winzenberg, 2012; Ooi et al., 2016). A child with ASD can require the attentions of a family member from the time of awakening until the time they fall asleep (DeGrace, 2004).

As discussed earlier, parents of a child with ASD typically spend 1,000 hours more per year on caring for and managing the child/s with ASD than the parents of typically developing children (Järbrink, 2007). This time is spent directly on the child with ASD and indirectly. Directly it is spent on constant vigilant parenting (Karst & van Hecke, 2012) both during the day and during the night time (Gorlin et al., 2016; Myers et al., 2009). Even simple routines like mealtimes can be extraordinarily challenging (Marquenie, Rodger, Mangohig, & Cronin, 2011) due to child with ASD's atypical food preferences and behaviours. Typical behaviours reported during mealtimes include temper meltdowns (Dickie, Baranek, Schultz, Watson, & McComish, 2009), gagging on food (Dickie et al., 2009; Marquenie et al., 2011), walking away from the table (Marquenie et al., 2011) and throwing food on the floor (Marquenie et al., 2011). Indirectly, parents need to constantly watch the child to make sure the child does not run out of the house or into a room with things that are harmful, such as the bathroom where a medicine cabinet and potentially dangerous implements need always to be secured, or stopping the child from hurting their siblings. All of these concerns make life difficult for the entire family (Myers et al., 2009) and can consumes the caregiver's entire day often leaving little or no time for the parents themselves.

Further time is spent in coordinating and financing services (Gorlin, McAlpine, Garwick, & Wieling, 2016), traveling to medical services (McAuliffe, Vaz, Falkmer, & Cordier, 2017), as well as the time spent at the therapies themselves (Myers, Mackintosh, & Goin-Kochel, 2009). These heavy time commitments around the child with ASD further restrict the family and couple's social

participation (Meny, Hayat, & Wright, 2018) and can have a deleterious effect on the couple's relationship. Research has shown that parents of children with ASD have voiced that they do not have enough time with their partners because of the demands of caring for their child (Myers et al., 2009; Saini et al., 2015). This loss of time spent together due to the high demands of taking care of the child with ASD, also puts strain on the parent's relationship (Matthews, Booth, Taylor, & Martin, 2011; Myers et al., 2009) which can often lead to separation or divorce (Hartley et al., 2010).

Often one of the parents, typically the mother, has to quit her job or curtail time spent working to devote the required time to the child with ASD (Myers et al., 2009). This reduction in, or absence of, work hours can place the entire family into financial hardship. Some families report that the father needs to maintain more than one job in order to meet the financial demands of the family (Myers et al., 2009). The monetary burden that the family of the child with ASD faces is discussed next.

4.1.2 Monetary Burden

Families with children with ASD have higher costs of living than families generally (Rogge & Janssen, 2019). Monetary issues such as greater investment in health care and associated therapies for the child with ASD as well as less opportunities for both parents to work impose a financial burden that can have a significant impact on the entire family's quality of life (Karst & van Hecke, 2012). Parents report that the amount of effort and time they devote to the work domain is significantly impaired by the demands of the child with ASD (Matthews, Booth, Taylor, & Martin, 2011), and this level of child care often leads to a restriction on parents' ability to engage in paid employment or gain promotion or advancement in their current roles (Fletcher et al., 2012; Matthews et al., 2011; Sitimin et al., 2017). Such a reduction in family income in conjunction with greater investment in health care (Fletcher et al., 2012; Järbrink, 2007; Karst & van Hecke, 2012) exacerbates the family's financial burden. These financial restraints often limit the possibility for

family holidays, educational opportunities for all family members, and the purchase of material goods (Dyke, Mulroy, & Leonard, 2009).

Lack of or restricted finances also contributes to a negative impact on the family's social participation (Meny, Hayat, & Wright, 2018). Siblings of the child with ASD are also affected, with research showing that these siblings have a lower rate of participation in extracurricular school activities (Wigston, Falkmer, Vaz, Parsons, & Falkmer, 2017), social activities with friends, hobbies and recreational classes compared to children with typically developing siblings (Rao & Beidel, 2009). These restrictions are due to the financial constraints and additional burden related to the care of the child with ASD (Dyke et al., 2009).

Rogge and Janssen (2019) in a recent literature review of 48 papers covering multiple countries (US, UK, Australia, Canada, Sweden, the Netherlands, Egypt and China), analysed the economic costs associated with living with a child with ASD. Five main categories of costs were identified for the families of the child with ASD: 1) medical and healthcare service costs; 2) therapy costs; 3) (special) education costs; 4) costs of informal care and lost productivity for family/caregivers; and 5) costs of accommodation, respite care, and various out-of-pocket expenses such as travel and time off work associated with care (Rogge & Janssen, 2019).

4.1.3 Stigmatisation and Discrimination

Parents of a child with ASD consistently report that they experience feelings of social stigma (Chan & Lam, 2017; Farrugia, 2009; Gray, 1994; Meny et al., 2018) and negative experiences of discrimination in their interaction with their wider community (Divan, Vajaratkar, Desai, Strik-Lievers, & Patel, 2012). Parents of a child with ASD have also reported intolerance in religious gatherings such as their child with ASD being made fun of by both adults and children in the congregation (Myers et al., 2009). These experiences of embarrassment and ostracism by their peers (Dyke et al., 2009) also contribute to the family restricting their social life. Parents of a child with

ASD often refrain from taking part in social activities out of fear they will be stigmatised by others for their child's *invisible* disability (Ooi et al., 2016).

Many parents also avoid social interactions with their relatives. Parents have reported that relatives sometimes blame the parent/s for causing the child's ASD while others believe the parents are using bad parenting practices which have caused or which maintain the child's symptoms of ASD (Myers et al., 2009). Parents who experience these unsupportive even punitive attitudes from family and friends, tend to value leisure activities that do not require any public exposure (Divan, Vajaratkar, Desai, Strik-Lievers, & Patel, 2012).

In the same vein, parents of the child with ASD also avoid inviting friends to their house for multiple reasons. Aside from fears of stigmatisation, criticism of their parenting, and fear that their child may be disruptive or aggressive during the visit, thus reinforcing others' perceptions, the parents of the child with ASD often do not have enough time or are too tired to clean the house or prepare for the visit to the standards they would wish. In sum, many parents avoid inviting anyone to socialise with them in their home as they are fearful that they or their child might be criticised, and they are often embarrassed that their house is untidy and not cleaned to their standards (Myers et al., 2009). This further restricts their social life.

Liao, Lei, and Li (2019) conducted a meta-analysis of 25 studies looking at parents of children with ASD and levels of stigma. They found that in all 25 studies, the parents of children with ASD reported that they had experienced significant levels of stigma. The experience of stigma and the negative perceptions of others were common problems for parents of children with ASD, despite the heterogeneity of the samples in each of the studies. Parents from different cultures (western to eastern) all struggled to deal with stigma related to their autistic children. Other than parents, siblings of the child with ASD were also often ostracised or teased about their sibling. In fact, parents have reported that siblings are often embarrassed by their sibling with ASD and/or often hear others laughing at the sibling with ASD (Myers et al., 2009). Their friends might say: "oh yes you can come

around but can you not bring… [the child with ASD], they're a bit loud", or "rolling their eyes at the siblings or parents when in the company of the child with ASD" (Farrugia, 2009). Liao et al. (2019) concluded that educational strategies to inform the public about ASD and its' symptomology may help to erase the stigma experienced by these parents and siblings and thereby reduce some of the hurt experienced by these families (Liao et al., 2019).

4.1.4 Extra Burden on Mothers

Compared with mothers of typically developing children, mothers of children with ASD reported significantly higher levels of fatigue (Giallo, Wood, Jellett, & Porter, 2013). Their fatigue was associated with poor maternal sleep quality, demands of support from their child, and the poor quality of their physical activity. Fatigue is also significantly related to other aspects of wellbeing, including stress, anxiety and depression, and lower parenting efficacy and satisfaction (Giallo et al., 2013). Fatigue might compromise mothers' cognitive and physical functioning, in turn contributing to their experience of stress (Seymour, Wood, Giallo, & Jellett, 2013). It can also lead to fewer opportunities to engage in self-care behaviours (e.g., engaging in relaxing and pleasant activities, healthy eating and exercise) (Giallo et al., 2013).

Family mealtimes are reported to be challenging due to demands from the child with ASD and they often involve exhaustive work especially by the mother (Ausderau & Juarez, 2013). Atypical food preferences and disruptive mealtime behaviours of the child with ASD increase the stress and workload associated with both meal preparation and mealtimes (Ausderau & Juarez, 2013; Curtin, Hubbard, Anderson, Mick, Must, & Bandini, 2015). Further, the stereotypical behaviours of children with ASD can make mealtimes as an occasion for family bonding difficult. Families in Ausderau and Juarez's (2013) study reported that mealtimes were important but challenging. As mentioned earlier, these challenges relate to the demands of the child with ASD, which involves exhaustive work especially by the mother during disruptive mealtimes. Atypical food preferences and disruptive mealtime behaviours further increase the stress of parents battling to give nutritious and healthy

food to the child with ASD (Ausderau & Juarez, 2013). Families attempt to cope with these difficulties using creative arrangements, such as presenting the child with one or more foods on a utensil and/or plate the child likes (Silbaugh et al., 2016), only giving the child's highly preferred foods (Silbaugh et al., 2016), repackaging healthy food into "acceptable" containers, and providing contingent rewards (Ledford, Whiteside, & Severini, 2018). Each of these strategies place an extra burden on the caregiver and don't always result in a balanced diet for the child.

4.2 Coping Strategies of Families

Research has highlighted the importance that coping strategies have on the well-being of the parents of children with ASD (Pepperell et al., 2016). Coping refers to how individuals respond cognitively and behaviourally to manage a stressful situation and the emotions that accompany these situations (Lazarus & Folkman, 1984). In their Stress and Coping Model, Lazarus and Folkman (1984) posited that coping strategies can be classified broadly as problem-focused and emotion-focused. Problem-focused strategies are aimed at solving or changing the problem or stressor, while emotion-focused strategies are aimed at reducing or managing the feelings of psychological distress associated with the stressor.

The main coping strategies of parents of children with ASD identified in the literature include 1) active avoidance/disengagement coping (Benson et al., 2010; Hastings et al., 2005) where parents focus on passive appraisals (Twoy, Connolly, & Novak, 2007) and escape-avoidance coping (Pisula & Kossakowska, 2010) where parents attempt to avoid dealing with the situation; 2) engagement/positive coping (Benson et al., 2010; Hastings et al., 2005) where parents seek social support and where they proactively attempt to deal with issues (Pisula & Kossakowska, 2010) and reframing (Twoy et al., 2007) with parents try to put a different perspective onto the issue; and 3) religious coping (Hastings et al., 2005; Tarakeshwar & Pargament, 2001) involving religious activities, including prayer and spirituality (Ekas, Whitman, & Shivers, 2009). Vernhet et al. (2018) carried out a recent systematic review of the coping strategies used by parents of children with ASD and found

similar results, that is: 1) parents of a child with ASD used more avoidance strategies and less social support-seeking strategies than parents of typically developing children; 2) where problem-focused coping strategies were used, they protected against parental stress and enhanced quality of life, and 3) the use of emotion-focused coping was a risk factor for partners' altercations and damage to their relationship. Gray (2006) conducted a longitudinal study of 28 parents (19 mothers and nine fathers) with a child with autism over a period of approximately a decade. The investigation was based on ethnographic methods that emphasised in-depth interviews and participant observation, concerning: the child's medical history and referral experience, the child's present symptomatology, the effects of the child's problems on the parent's well-being, the effects of autism on the family's social life, parental coping strategies, illness conceptualisation and the parents' expectations for the future. Results showed that coping strategies changed over time, as fewer parents coped through reliance on service providers, family support, social withdrawal and individualism and relatively more parents coped through their religious faith and other emotion-focused strategies.

The use of coping strategies and their impact on family adaptation are considered to be ongoing for the family of a child with ASD. The extant literature shows differences in the adaptation of the families of children with ASD, with variability in the coping strategies they employ and their levels of social support linked to individual differences in outcomes (McStay et al., 2014; Paynter et al., 2013). For example, a family's sense of coherence, that is their ability to cope with stressors, has been related to positive parental outcomes while an inability to deal with the externalising behaviours of the child with ASD had a negative impact (McStay et al., 2014). Additional non-coping strategies or attributes which have been found to help parents cope are a sense of self-efficacy or mastery (Hastings & Brown 2002; Weiss, 2002) and sense of resilience (Byrne, Sarma, Hendler, & O'Connell, 2018).

Resilience is an important protective and adaptive factor in people's ability to deal with stressors, no less so family stressors. Resilience is an individual's ability to cope with and adapt to

stress and adversity (Masten, 2009). It has been related to factors that promote well-being and protect against risk in the family (Fisman, Wolf, Ellison & Freeman, 2000; Giallo & Gavidia-Payne, 2006). Research has shown high correlations between the use of positive coping strategies, that is, the ways in which people proactively deal with life stressors, and resilience (Moore, Russell, & Bouchoucha, 2017). It may be that positive coping strategies, combined with support, may be what contribute to families' sense of resilience in the face of stressors. In some situations, such as living with a child with ASD, viewing any associated adversity as a challenge may be helpful in fostering adaptive coping and hence resilience (Moore et al., 2017). Overall, resilience or hardiness, social support and proactive coping have been shown to be predictive of successful adaptation among families generally, with social support also helping to ameliorate stress-related symptoms (Weiss, 2002). It might also be that good support and proactive coping contribute to building resilience in the families of children with ASD as they cope with ongoing change and challenges. Thus, it is important to understand both the coping mechanisms of parents of a child with ASD and the level of support available to them in order to improve their well-being.

4.2.1 Support

Social support can be received or perceived (Kaniasty, Norms, & Murrell, 1990; Norris & Kaniasty, 1996) and it seems that either can be important in helping people deal with stress. Social support can have a direct effect or a buffering effect (Cohen & Wills, 1985; Thoits, 1982). Social support, besides its effects on health behaviours exerts a direct effect on physical systems but it also acts as a buffer, especially under conditions of stress (Ditzen & Heinrichs, 2014). A direct effect is when social support is attributable more to an overall beneficial effect of support while buffering effect is when a process of support protects the person from the potentially adverse effects of the stressful event (Cohen & Wills, 1985).

Understanding variables related to family adaptation or conversely, a reduction in the impact of living with a child with ASD, is important when developing support strategies for families living

with a child with ASD as well as to support outcomes for the children affected by ASD (Pepperell, Paynter & Gilmore, 2016). The availability of social support has been found to moderate the impact of high levels of stress on psychological wellbeing (Cohen, 2004) and an individual's perception of the extent of availability of this support is a fundamental aspect of this buffering effect (Cohen, 1988). Research suggests that the quality of social support and its perceived availability moderates the influence of high levels of stress on health outcomes in a range of clinical populations (Payne et al., 2012; Steptoe, 2000) as well as parents of children with ASD. The benefits of social support for parents of children with ASD have been researched over the last three decades (Boyd, 2002; Bromley, Hare, Davison, & Emerson, 2004; Gray & Holden, 1992; Robinson, Weiss, Lunsky, & Ouellette-Kuntz, 2016) with positive results.

Boyd (2002) found that there is an association between the challenging characteristics of the child with ASD and a mother's inclination to seek social support. Those mothers under greater stress from their child's demands or behaviours were more prone to pursue social support; mothers who received low levels of social support were the most likely to experience depression and anxiety. Bromley et al. (2004) found significant psychological distress in mothers of children with ASD was associated with low levels of family support and with their child's higher levels of challenging behaviours. Robinson et al. (2016) found that informal support was negatively related to burden in parents of children with ASD, although it did not act as a moderator. Gray and Holden (1992) found similar results in parents of children with ASD who received more social support, had lower depression, anxiety and anger.

Benson (2012) examined the characteristics of the support networks of 106 mothers of children with ASD. He found that networks indirectly effected mothers' adjustment, mediated by perceived social support and measures of network structure (network size) and function (proportion of network members providing emotional support). Network size and portion of network members

providing emotional support predicted increased levels of perceived social support which, in turn, predicted decreased depressed mood and increased well-being of the mothers.

Hastings and Johnson (2001), in their quantitative study of 141 parents of children with ASD, found that the perceived availability of informal supports was significantly negatively related to parental stress. Conversely, Cantwell, Muldoon, and Gallagher (2014) studied 109 parents of children with ASD and found that the association between stress and physical health was moderated by perceived social support.

The chronic nature of ASD dictates that social support for parents needs to be pervasive and ongoing. Results of past research show that both perceived and received social support exert a substantial positive effect in reducing parental stress levels associated with living a child with ASD. Also, the quality of the support and its perceived availability moderates the influence of high levels of stress on health outcomes.

4.3 Chapter Summary

Research shows that ASD has a more deleterious impact than other childhood disorders, not only on the wellbeing of children with the disorder, but also their parents and their siblings. Families living with a child with ASD are affected by the child's antisocial and disruptive behaviours. Parents of children with ASD report they spend more time caring for their child with ASD than other parents; Mothers in particular, are often restricted in their ability to be part of the workplace which, in conjunction with the costs of the child's treatment, places a financial burden on the family. Families are often further burdened by social ostracism by the public and, regrettably, sometimes blame from family and friends. How families cope with living with a child with ASD, and the supports available to them, are imperative in determining the level of burden or adaptation of the family. Compared with mothers of typically developing children, mothers of children with ASD have reported significantly higher fatigue and stress. The main coping strategies used by parents of children with ASD identified in the literature include active avoidance/disengagement coping,

engagement/positive coping and religious coping. Additional non-coping strategies or attributes which have been found to help parents cope are a sense of self-efficacy or mastery and feelings of resilience. Social support has been found to be negatively associated with stress in parents of children with ASD. In sum, the symptoms of ASD have been shown to have a significant impact on the quality of life and the psychosocial wellbeing of the entire family (Lovell et al., 2015; Wigston et al., 2017).

Chapter 5. Models of Family Adaptation to Stress

Parenthood is a major life event (Mercer, 2004) and considered one of the most meaningful and complex of human experiences (Stern & Bruschweiler-Stern, 1998). As one's parental identity evolves, so too personal and work-related activities expand to include those of the new family (Christie et al., 2017; Genesoni & Tallandini, 2009; Rubin, 1967). The parenting role is a highly complex process (Berry & Jones, 1995) and includes parents' hopes and dreams for their children (Mercer, 2004; Rubin, 1967; Siddiqui & Hägglöf, 2000), as well as dealing with everyday behaviours and decisions. These family occurrences can influence and are influenced by parents' other roles (such as being a partner, a friend) and by roles outside the family (e.g., work, friendships) (Menaghan, 1983). Despite the joys of parenthood, this highly complex process of ebb and flow can result in parental role strain (Barnett & Baruch, 1985) involving both role overload and role conflict (Berry & Jones, 1995). According to Berry and Jones the obligations and responsibilities involved with the mother's parenting role might be the most significant source of stress in women's lives. While fathers also face the same challenges and difficulties, the literature has typically addressed mothers or, more generally, family.

Compared to other stressors such as job stress, parenting stress is unique and subjective (Roggman, Moe, Hart, & Forthun, 1994). In addition to the joys of parenthood, Ventura (1987) described the subjective feelings of irritation, annoyance, inadequacy, frustration and distress which parents can experience in response to the demands of, and daily interactions with, their children. Such parenting stress has been associated with insecure attachment of the child to the mother and father (Jarvis & Creasey, 1991). On the other hand, a sensitive, responsive caregiver is crucial to the development of secure attachments during infancy (Ainsworth, Blehar, Waters, & Wall, 2015). Parenting a child can be especially stressful if the child has health or behavioural issues outside the "norm".

Over the course of the last century, researchers have made efforts to understand and explain how families adapt to such stress (Angell, 1936; Burgess, 1926; Cavan & Ranck, 1938; Hill, 1949, 1958; Koos, 1946; Malia, 2006). A vulnerability to stress will have an impact on subsequent wellbeing and research has emphasised the uniqueness of stress people's responses to individual and specific stressors (Lazarus & Folkman, 1984). Researchers have studied parenting and family stress when faced with a child's prolonged hospitalisation or a physical or emotional disorder (Anastopoulos, Guevremont, Shelton, & DuPaul, 1992; Quittner, Glueckauf, & Jackson, 1990). This early work on family stress was conducted by Burgess (1926), Angell (1936), Cavan and Ranck (1938), and Koos (1946) during the Great Depression of the 1930s.

At that time, researchers took the construct of stress (borrowed from structural engineering), and introduced it into the field of family studies (Kingsbury & Scanzoni, 1993). Several theorists have proposed models to explain predictors of family stress and, more recently, the stress on families living with a child with a chronic illness. According to Malia (2006), it was Hill's (1949, 1958) ABCX Model of Family Stress that laid the foundations of contemporary family stress research. In the later part of the 20th century, the Contextual Model of Family Stress (Boss, 1987; 2002), and the Family Adjustment and Adaptation Response model (Patterson, 1988; 2002) were also developed and lead to the subsequent development of the Double ABCX model (McCubbin & Patterson, 1982; 1983) An important focus of these models was on parenting stress where children have special health or developmental needs (Hodapp, 1997; Roach, Orsmond, & Barratt, 1999). The historical development of these models is provided next in order to provide a theoretical context for an investigation into the impact on families living with a child with ASD.

5.1 ABCX Model

Hill's ABCX model (1949, 1958) laid the foundations for contemporary family stress research (Malia, 2006). Hill (1949), building on the initial studies from the Great Depression Era and his work on families' response to war separation and reunion, advanced the first major family stress

framework in the ABCX model. The ABCX model is focused on the factors preceding a potential crisis (or for some, adaptation) that determine the family's capacity to cope with the stressful event (Figure 5.1). In the model, factor 'a' represents the crisis-provoking stressor event and related hardships that interact with factor 'b' representing the family's resources for meeting a crisis and factor 'c' representing the family's perception or appraisal of the stressor: these three factors come together in an interactive manner to produce the amount of crisis or maladjustment, or conversely, the level of impact of the stressor, as factor 'x'.

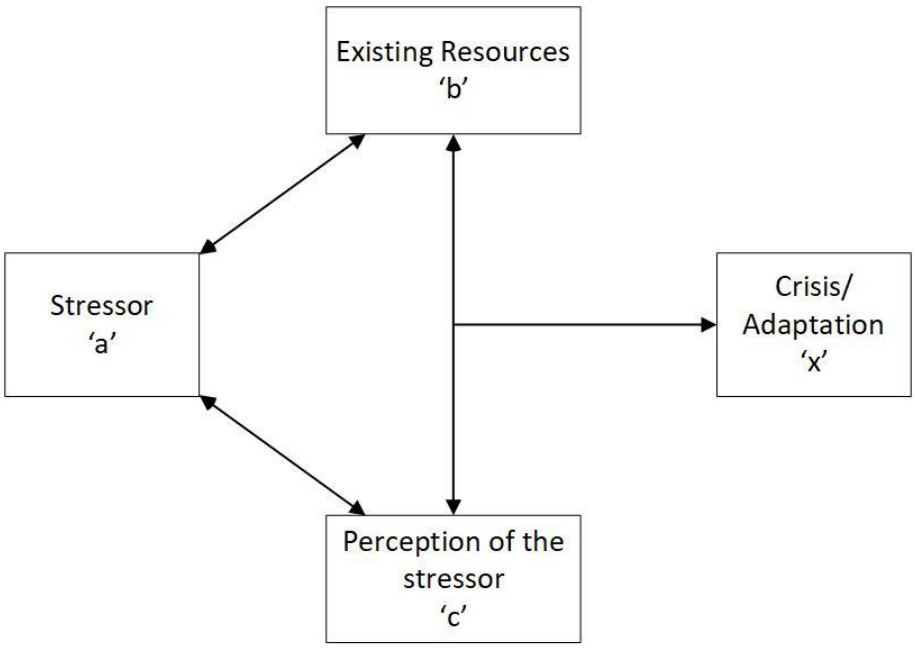

Figure 5.1: ABCX Model (Hill, 1958)

Hill (1949) defined stress as distinct from a crisis and predicted that resources and appraisals mediate the family's ability to adjust to the stressful event and prevent a crisis or, alternatively, their ability to adapt to the situation. In this context stress is considered a demand-capability imbalance in the family, while crisis is defined as a continuous variable denoting the amount of disruptiveness or disorganisation in the family social system (Burr, 1973). This disruptiveness occurs when the family is unable to restore stability following a stressful event (McCubbin & Patterson, 1983). Alternatively, if the family successfully manages the stress and restores stability, then they do not enter the state of

crisis but rather, one of adaptation. The Salutogenic Model of Antonovsky (1979) followed Hill's work.

5.2 Salutogenic Model

In his Salutogenic Model, Antonovsky (1979) sought to describe the process of maintaining health despite exposure to stress. The Salutogenic approach is focused on what moves people toward health and well-being in contrast to the traditional pathogenic paradigm that focuses on the cause of illness (Malia, 2006) or the level of crisis/adaptation (Hill, 1958). Antonovsky's argument was that people are exposed to stressors in daily life but some people are more or less affected by these than others. He acknowledged preceding factors such as genetic and psychosocial resistance resources, as well as life experiences, contributing to one's sense of physical or emotional ease/dis-ease, but his primary contribution was in relation to a sense of coherence (SOC). This SOC was said to intervene or mitigate between vulnerabilities, experiences, and people's health outcome (Figure 5.2). SOC refers to the extent to which one sees one's world as comprehensible, manageable, and meaningful (Antonovsky & Sourani, 1988). Comprehensibility refers to the extent to which events are perceived as making sense; meaningfulness involves a sense that the challenges faced are worthwhile to overcome; and manageability encompasses the ability to use coping strategies flexibly. SOC is a crucial determinant of an individual's position on the health continuum, where personal well-being depends on the individual's ability to successfully process stressors.

The Salutogenic Model has been used to study family adaptation (Antonovsky & Sourani, 1988) through its' three dimensions: comprehensibility, meaningfulness, and manageability (Antonovsky, 1987). It has also been used as a framework to explore the impact of a child's disability on parents, and to guide the implementation of methods of intervention (Oelofsen, & Richardson, 2006). While a SOC can contribute to managing tension and thus reinforcing a SOC and wellbeing, it is less clear why, in the Salutogenic Model (Figure 5.2), the path branches from tension to

unsuccessful management. It would be seemingly more usual to interpret high/low scores on constructs and their impact on an outcome variable than have two discrete paths.

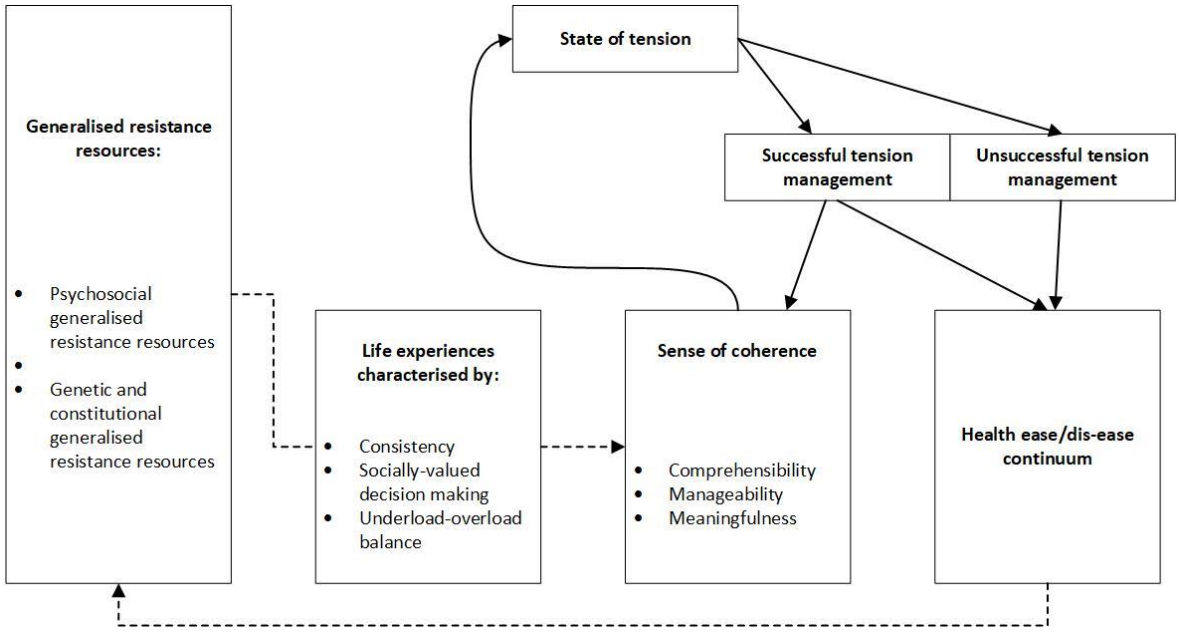

Figure 5.2: Simplified reproduction of the Salutogenic Model (Source Super et al., 2015)

Little research has used the Salutogenic Model and SOC in particular to understand the experience of parents of children with ASD and other developmental disabilities (Al-Yagon, & Margalit 2009; Oelofsen, & Richardson, 2006; Olsson & Hawang 2002). Those few studies that have been published based on these populations have focussed on the relationship between SOC and parental depression or between SOC and family social-environmental characteristics and these have been limited to samples from large urban areas (Margalit, Leyser, Avraham, & Lewy-Osin, 1998; Margalit, Raviv, & Ankonina, 1992; Olsson & Hwang, 2002).

Antonovsky's main outcome variable in the Salutogenic Model, namely health, was not studied in relation to parental SOC nor did those studies contain measures of parental stress, which has consistently been found to be higher for parents of children with a developmental disability (Oelofsen & Richardson, 2006). However, there is one study of interest which used this model to analyse the predictors of adjustment in parents of children with ASD (Siman-Tov & Kaniel, 2011). Their analysis showed that a sense of coherence, an internal locus of control, social support and the

quality of their marriage increased parents' ability to cope with the stress of parenting a child with ASD. They did not investigate the specific coping strategies of these parents or the level or type of impact on the family of living with a child with ASD.

A strength of the Salutogenic Model is that it can provide a useful theoretical framework, particularly in clinical situations, upon which to base interventions when used in conjunction with a cognitive behavioural approach (Oelofsen & Richardson, 2006). This approach has been illustrated in strategies designed to promote a sense of coherence in the treatment of adults with obsessive-compulsive disorder (Hastings & Beck, 2004; Joachim, Lyon, & Farrell, 2003). While a SOC reflects the individual person's coping and management of a stressor in order to cope with tension, it does not indicate any specific coping behaviours (Antonovsky, 1993), nor is it a comprehensive model of the factors which may have an impact on the family living with a child's illness.

5.3 Family Adjustment and Adaptation Response (FAAR) Model

The FAAR Model (Patterson, 1988) is an extension of the ABCX Model and attempts to describe the methods by which families achieve pre-crisis adjustment and post-crisis adaptation. In doing so, Patterson (1988, 2002) used the concepts of risk and protective factors, family resiliency, and adaptation at the individual, family, and community levels. According to Weiss, MacMullin, and Lunsky (2015, it extends the ABCX Model by suggesting that the family system uses its' capabilities (resources and coping behaviours) to balance its' demands (stressors, ongoing strains, and daily hassles) through a process of adjustment and adaptation. Specifically, the FAAR model emphasises four central constructs: family demands, family capabilities, family meanings, and family adjustment or adaptation, which interact with each other (Patterson, 2002). In this model, families engage in active processes to balance family demands with family capabilities as these interact with family meanings to arrive at a level of family adjustment or adaptation.

In Figure 5.3, the family demands are comprised of: (a) normative and non-normative stressors such as discrete events or change; (b) ongoing family strains that include unresolved,

insidious tensions; and (c) daily hassles consisting of minor disruptions of daily life. Family capabilities comprise: (a) tangible and psychosocial resources available to the family; and (b) coping behaviours that is, what the family does to manage demands or stressors. Both demands and capabilities can originate from three different levels of the ecosystem, they are: (a) individual family members; (b) the family unit; and (c) from various community contexts. For example, the diagnosis of a child with ASD would be an example of an individual level demand; marital conflict about how to manage the child's condition would be a family level demand; and community stigma about disability would be a community level demand.

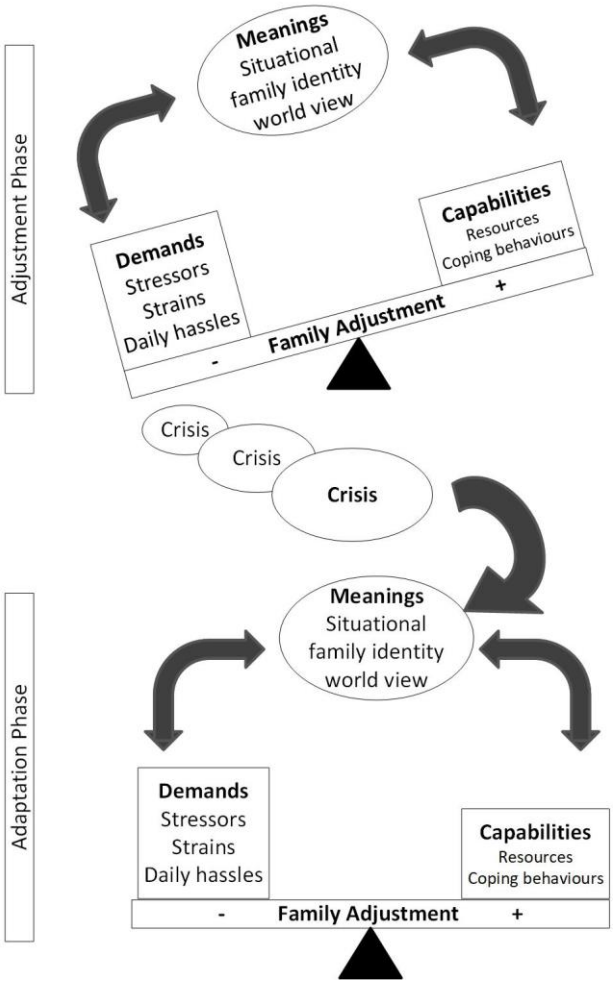

Figure 5.3: Family Adjustment and Adaptation Response (FAAR) Model (Patterson, 1988)

Parent education, family cohesiveness, and good health and education services are examples of capabilities at each of the three levels, which could be used to help manage the aforementioned demands (Patterson, 2002). In the model, crisis is defined as a subjective experience on a continuum of distress, from low levels of distress through to crisis (Patterson, 2002). A key factor of the FAAR model is the proposition that families utilise their capabilities (resources and coping behaviours) to balance their demands (stressors, strains, and daily hassles). Meaning is also an important factor in the FAAR Model which Patterson described across three levels: (a) the definition of demands (primary appraisal); (b) their identity as parents; and (c) their worldview. Patterson further argued that meaning influences the nature and extent of risk, as well as the protective capacity of the parent. Meanings (or appraisals) of an event as stressful or challenging are essential to the experience of that event as distressful, thus, stress and daily hassles may not be caused by the challenging experience itself, but by the interpretation of the experience as stressful by the parent (Patterson, 1988). The FAAR model has been used to understand the meanings parents place on their experiences. Understanding meaning is important in order to understand the effect that stressful experiences have on parental well-being and, to further understand the ways parents cope with these experiences.

For example, Weiss, MacMullin, and Lunsky (2015) used FAAR to examine experiences of distress in mothers of individuals with ASD. Their results indicated that greater child problem behaviours were related to less parental empowerment which, in turn, was related to greater maternal distress, indicating that empowerment was a partial mediator of the child's behaviour and maternal distress. Another study that used the FAAR Model as a theoretical framework to investigate the role of families' capabilities (coping strategies and resources of support) and positive meanings in raising a child with ASD in family functioning was that of Xue, Ooh, and Magiati (2014). They found that the parents of children with ASD who used family integration and optimism as coping strategies, followed by understanding the condition and developing esteem, reported greater psychological stability.

According to Weiss, Tajik-Parvinchi, Maughan, and Lake (2018), one important limitation of the FAAR Model is the focus on stressors and negative outcomes. In the FAAR Model coping exists before the encounter with a stressor (Patterson, 2002). And according to this approach, Weiss et al. argued that coping is like any other resource factor and determines whether individuals appraise a situation as stressful or not. This appraisal and use of coping resources then determines in part whether the individual is able to manage the stressful situation. This appraisal process is aligned with Lazarus and Folkman's (1986) premises in their Transactional Model of Stress and Coping, which emerged around the same period.

5.4 Contextual Model of Family Stress

The Contextual Model of Family Stress (Boss, 1987; 2002) includes the A, B, C and X factors from the ABCX model but for Boss, the X factor includes not just the crisis but the stress around it (Figure 5.4). Boss maintained that the primacy of resources (B) and assignment of meaning or perceptions (C) in handling the event or stressor (A) and in determining whether a stressor will precipitate a crisis or will lead to coping for any given family. In this model the factors are depicted as circles with an increasingly narrow focus or impact.

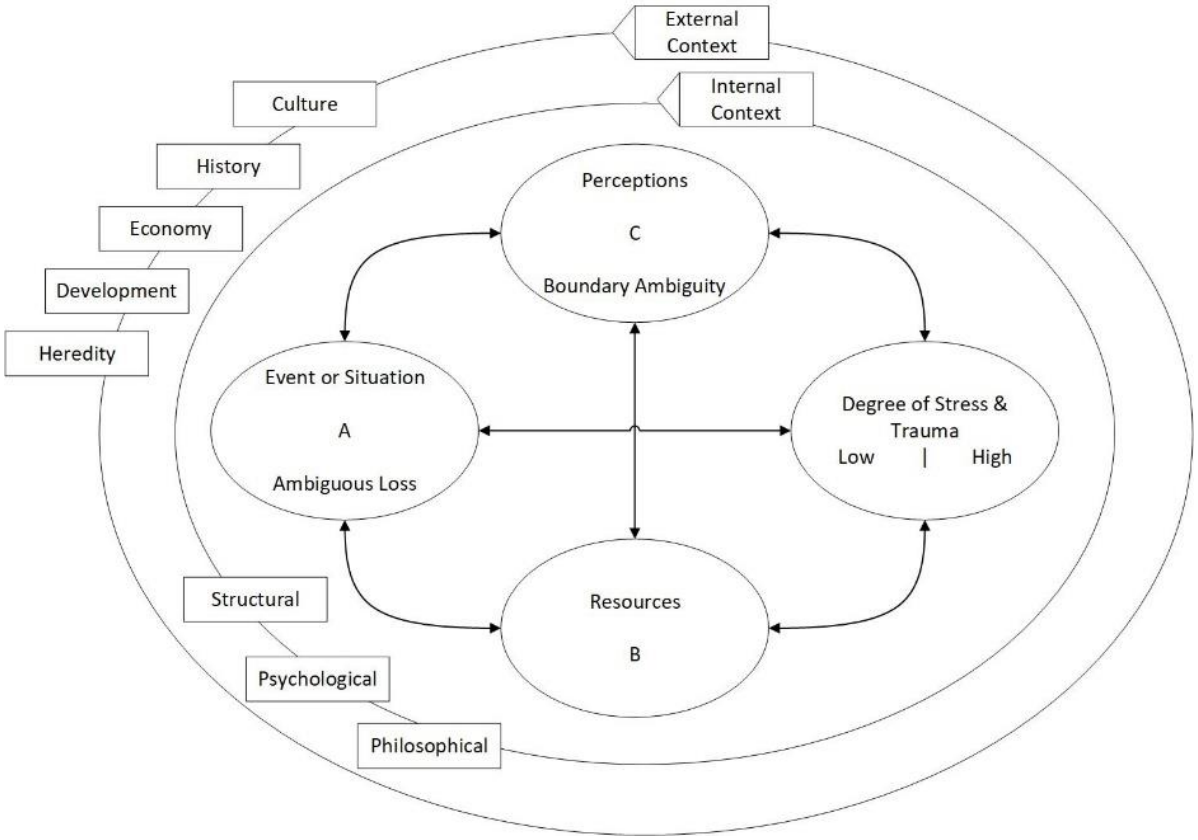

Figure 5.4: Contextual Model of Family Stress (Boss, 2002)

The outer circle represents the external contexts which are the components over which the family has no control, closer in is the internal context which the family is capable of modifying (Boss, 2002). External contexts that the family is rarely able to control comprise the constraints of genetics and development as well as economics, history, and culture. The internal context represents the elements that the family can control or change such as structural, psychological, and philosophical elements. The inner most component, or the heart of the model, relates to the event/stressor, the perceptions of it and resources to deal with it, and the impact of all. The Contextual Model of Family Stress model addresses some limitations seen in the ABCX by focusing on the ways in which families cope with stress and how these are influenced by external and internal contexts.

This model has been used to explore the experiences of parents living with a child with developmental disorders within different cultural backgrounds (Kim & Dababnah, 2019; Sullivan, 2015). Kim and Dababnah used the Model to explore the stress experience of Korean-American

immigrant parents of children with developmental disorders living in the United States of America (USA). They found four major themes: sources of difficulties, sources of support, perceptions of developmental progress, and personal transformations. The sources of difficulties reflected participants reported stress and frustration related to the child's developmental disability and the associated challenges such as the community's negative attitudes towards the child with a disability, and its lack of support. In sources of support, all participants stated they had received varying degrees of support from their family and the community. High levels of support helped alleviate some stress associated with caregiving demands but not all parents experienced adequate levels of support. Parents' perceptions of their aspirations for their child's developmental progress changed from the time prior to the child's diagnosis to that post diagnosis. Almost half the parents of older children with development disabilities revealed that they had unrealistic hopes for their child's development during childhood. These expectations included hopes the child would be "cured" or make substantial developmental progress with extensive training or treatments. Parents of younger children with developmental disabilities reported that, although they had hope for their child's future development, they accepted their child's special needs and probable limitations of their child's development. In terms of the last theme, participants reported they underwent positive personal transformations because of their caregiving experiences. These changes included increased persistence, spousal relationship cohesion, and a commitment to family life.

Sullivan (2015), in a case study, used this Family Stress model to understand the experiences of a family of a child with ASD in order to develop appropriate treatment plans for family therapy. She found the model was effective when working with diverse populations as it encompassed the richness of a family's lived experiences and offered clinically useful targets for both prevention and intervention. She indicated that she selected this model specifically as it encompasses the effect of the family's race and culture on the stressors that people experience. These last were particularly important for Sullivan as she studied families from diverse ethnic backgrounds. Sullivan also studied the coping strategies of mothers. She found that when mothers assigned negative meaning to the

father's presence in the home, this was a stressor which had the potential to precipitate a crisis in the family. Furthermore, any conflict between the family and the extended family coupled with the lack of support provided by the extended family resulted in an increase in the family's stress. Sullivan argued in was important to work with all available family members to address these stressors associated with living with a child with ASD. She saw this as a reasonable approach to ameliorate the presenting problems and possibly avert a crisis in the family system. Sullivan argued, on the basis of her case study analysis, that the Contextual Model of Family Stress is a suitable tool for effectively guiding treatment. It must be acknowledged however, that her recommendation was based solely on the case study of a single family.

In comparison to FAAR and ABCX Models, the Contextual Model of Family Stress offers a parsimonious set of critical factors that can be easily assessed and which can guide intervention and treatment protocols (Sullivan, 2015). It has also addressed the effect of culture, which is particularly relevant in diverse populations. Despite its strengths, the model has limitations. Like other models, it views all families through one theoretical lens and ignores aspects of their presentation that do not fit into that particular orientation. This approach may cause clinicians to miss salient features of an individual family's experience (Sullivan, 2015). Additionally, the model inherently does not promote information collection via validated assessments. As suggested by Sullivan, the use of a theoretical framework to organise clinical information is only valid if a thorough assessment has been conducted and the information gleaned is accurate. The Double ABCX model is an extension of Hill's (1949) model and is discussed next for its depiction of families' adaptation to a crisis or to a chronic illness within the family.

5.5 Double ABCX Model

The original ABCX model (Hill, 1949) considered only the factors preceding the stressful event (depicted as lowercase letters in Figure 5.5). McCubbin and Patterson (1982; 1983) expanded that model into the Double ABCX Model and included factors that occur after the stressful event such as

stress proliferation and seeking additional social supports (McCubbin & Patterson, 1983). By redefining pre-crisis variables and adding post-crisis variables, the double ABCX Model describes: (1) the accumulation of life stressors and strains that is, stressors prior to those following the crisis-producing event, which result in a pile-up of demands (A); (2) the range of family outcomes in response to this pile-up of stressors (maladaptation to bonadaptation) (X); and (3) the intervening factors that shape the course of adaptation: family resources; both existing and new, perception, and the related coping strategies.

In sum, the Double ABCX model comprises: severity of the stressor (a) (e.g., severity of family member illness); pileup demands and additional life stressors (aA) (e.g., additional illness in the family, divorce); family's internal resources (b) (e.g., locus of control); family's new resources (bB) (e.g., social support, finances); family's perception of the situation (c) (e.g., positive or negative, threat or challenge); coping strategies used (BC); family's perception and coherence (cC); the crisis (x); and the outcome (xX) in the form of type of adaptation.

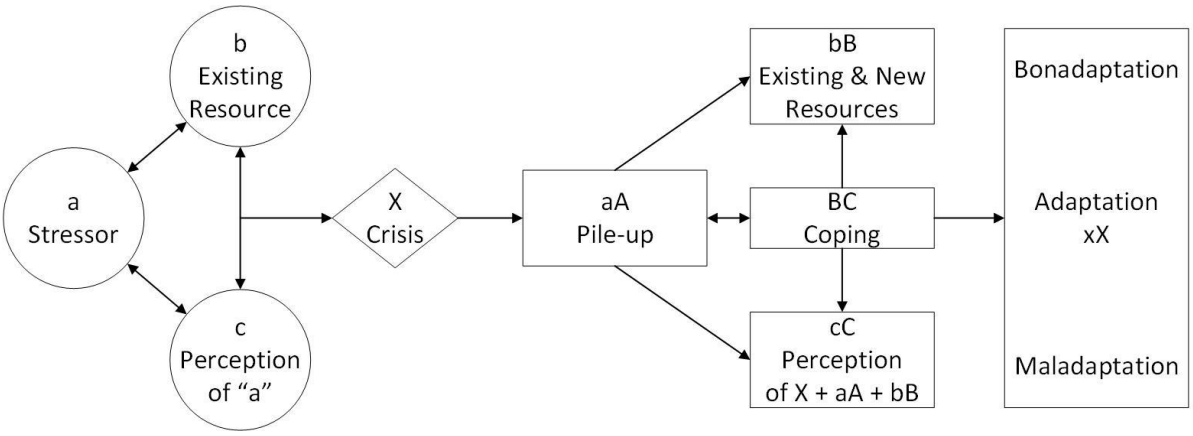

Figure 5.5: Double ABCX Model (McCubbin & Patterson, 1983)

To elaborate further, in the model, the initial stressor/s (a) represents situation or event for which the family had minimal preparation. It is seen as problematic and can precipitate a crisis. The existing resources (b) represent the qualities, capacities, or entities that were already in place or available that help the family to meet the demands of the initial stressor. Initial perception (c) is the

interpretation or meaning the family members ascribe to the initial stressor (a). Crisis (x) relates to the situation or feelings emanating from the stressor that overwhelm or incapacitate a family. The pile-up (aA) factor denotes the cumulative effect of all stressors on the family occurring prior to, during, and immediately following the current stressor over crisis (a). This Pile-up of stressors is regarded as a process with a complex set of changing conditions that have a history and a future, and where the family is viewed as dealing with a cluster of normative and non-normative events rather than with a single stressor (Lavee, 2013). This clustering effect of normative and non-normative events over time led Patterson and Mccubbin (1983) to incorporate them into their model. These cumulative sources of stress may place additional demands on the family while it struggles with a major stressor event such as the chronic illness of a family member, or parenting a child with a disability (McCubbin, & Patterson, 1983). Dealing with this pile-up of stressors will require some form of change from the individual or family in order to reach a state of stability (McCubbin, 1998). These changes might involve use of resources to help with the problem (B), the use of coping strategies to manage the situation (C), or changing one's perceptions about the issue (C).

Resources refers to the qualities, capacities, or entities that help the family meet the demands of the crisis, including intra-family qualities, individual psychological capacities, and the presence or availability of social support (Lavee, McCubbin, & Patterson, 1985). It also includes the resources developed and strengthened in response to the stressor (a). In combination these resources help families to tackle the pile-up of demands by either reducing the impact of demands on the family and/or helping the family adapt to the required changes (Lavee, McCubbin, & Patterson, 1985). These resources are considered adaptive and can be categorised as personal resources, family system resources or social support (Lavee et al., 1985). Personal resources are the characteristics of individual family members such as self-esteem, knowledge, and skills which are potentially available to the family in times of need (Pearlin & Schooler, 1978). Family system resources include the internal attributes of the family unit such as cohesion, flexibility (adaptability) and communication

(Olson, 2000). Social support refers to the capabilities of people or institutions existing outside the family system on which the family can draw or a network in which the family is cared for and loved, is esteemed and valued, and where family members feel that they belong (Cobb, 1976; Lavee et al., 1985; Pilisuk & Parks, 1981).

Perception and Coherence (cC) is the ongoing interpretation or meaning family members ascribe to the crisis in the intersection of the crisis, pileup demands, and old and new resources. Perception and Coherence also refer to the family's general orientation to the overall situation (Lavee et al., 1985). This orientation has been described as pervasive and enduring with a dynamic feeling of confidence that internal and external environments are predictable (Antonovsky & Sagy, 1986). It reflects a sense of acceptance and understanding of the situation, a framework within which a definition of the situation is made and within which perceptions are judged (Antonovsky & Sagy, 1986; Lavee et al., 1985). In the double ABCX Model coherence is dynamically influenced by the internal and external experiences of the family. Internal experience relates to the family's internal environment such as perceived strengths: the external environment relates to the cumulative effect of positive and negative experiences from with the external environment (Lavee et al., 1985). This coherence, in turn, shapes the meaning the family gives to the total crisis situation. This crisis situation encompasses the stressor event, added sources of strain (pile-up), and the resources the family has for meeting the demands. Within such a definition coherence becomes a facilitator of the family's adaptive power.

Coping (BC) represents the coping strategies used by the family in response to the stressor. It is the ways in which families integrate family resources (bB factor) and perceptions of the situation (cC factor) to adapt to stressful situations (McStay et al., 2015). These may be proactive or avoidant and again, influence levels of adaptation or maladaptation such as seeking out additional supports or looking at the stressful event in a more positive light (McCubbin et al., 1981).

The outcome of dealing with stressors, perceptions and crisis leads to family adaptation (xX). Family Adaptation (xX factor) is the collective outcome in response to the antecedent factors of stress (a), resources (b), and perceptions (c) which predate the crisis (X). The crisis leads to what McCubbin and Patterson (1983), termed Pileup (aA) which, in turn, leads to further perceptions, use of coping strategies and resources, culminating in some level or adaptation or maladaptation (xX). Maladaptation, the negative end of the continuum, is defined as the continued imbalance between the pile-up of demands and the family's capabilities for meeting those demands (Lavee et al., 1985). On the adaptation/maladaptation continuum, maladaptation may be characterised by deterioration of family integrity, of family members' sense of well-being, and of their physical and/or psychological health (Lavee et al., 1985). Bonadaptation, the positive end of the continuum, is defined as a minimal discrepancy between the pile-up of demands and the family's capabilities to achieve a balance in family functioning (Lavee, 2013; Lavee et al., 1985). Bonadaptation is characterised both by maintenance or strengthening of family integrity and by family members' sense of well-being (Lavee et al., 1985). It is important to note that adaptation does not imply that there has been no disorganisation or change in the system (Burr, 1973): it merely denotes the family system resuming its routine level of operation after having to cope with the change (Burr, 1973).

5.5.1 Double ABCX Model in ASD Research

Lavee et al. (1987) suggested that the Double ABCX model is suited for the dual purposes of integrating research and guiding clinical practice with families. It has a number of strengths over other models that make it suitable for utilisation in research with families living with a child with ASD. In comparison to the Salutogenic Model, in the Double ABCX model the researcher can determine the specific coping styles of the parents of children with ASD. Additionally, McCubbin and Patterson (1983) incorporated the SOC construct into their Double ABCX model in Perception and Coherence (cC) (Oelofsen & Richardson, 2006) giving it the strength of both the Salutogenic and ABCX models.

According to Pickard and Ingersoll (2017), the Double ABCX model includes a number of factors to explain family adaptation to stress that also make it suitable for use in research. They argued that the model is comprehensive and includes several variables that work together to determine adaptation to stress rather than considering each factor in isolation. Another strength suggested by Lavee (1997), is the flexibility within the model in how specific variables are operationalised. This flexibility allows for its application in different stressful situations and populations and is also applicable to ongoing stressors, such as those associated with raising a child with ASD. The elements of the Double ABCX model explain a significant proportion of the variance in how parents adapt to a life with children with ASD (Bristol, 1987; Manning et al., 2011; McStay et al. 2014; Saloviita et al., 2003) as discussed next.

For instance, Bristol (1987), in a study of 45 families of autistic and communication-impaired children found that the model accounted for 55% of variance in in-home family adaptation, 33% of variance in depressive symptoms and 53% of variance in marital adjustment. The family adaptation was positively predicted by adequacy of social support and active coping patterns. Poorer adaptation was predicted by other family stresses, unwarranted maternal self-blame for the handicap, and maternal definition of the handicap as a family catastrophe. Finally, resources and beliefs were more predictive of adaptation than severity of the child's handicap.

McStay et al. (2014) studied 98 mother-father dyads of children with autism who were aged three to 16 years. They explored the potential predictors of maternal and paternal stress and family quality of life. The Double ABCX model indicated that 57% of the variance in maternal stress was explained by factors in the model. Their child's externalising behaviours explained a significant amount of variance (13%) in maternal stress and SOC negatively predicted a further 8% of the variance in maternal stress. Their findings showed the negative impact of child externalising behaviours and the importance of family SOC on positive parental outcomes.

Pozo, Sarriá, and Brioso (2014) evaluated the Double ABCX model in a sample of 118 parents (59 mothers and 59 fathers) with a child with ASD for the impact on family quality of life and psychological well-being. The 59 children with ASD were aged four to 38 years, and the adult children still lived at home. The results showed that children with more behavioural problems and higher levels of ASD severity, as well as a lower parental sense of competence and less social support, were associated with lower family quality of life for both mothers and fathers. Coping strategies were variously related to adaptation, where active avoidance coping strategies were negatively correlated with psychological well-being while positive and problem-focused coping were related to psychological well-being for the parents.

Although they did not specifically address parents of children with ASD, Saloviita et al. (2003) found that the model was helpful in explaining parental stress of fathers and mothers of children with an intellectual disability. In their study, multiple regression equations explained 72% of the variance in maternal stress and 78% of the variance in paternal stress, with the single most important predictor of parental stress being parents' negative definition or perception of the situation. In fact, perceptions were different in mothers and fathers. For mothers, the negativity was associated with the child's behavioural problems while for fathers it was associated with the lack of social acceptance of the child. Experienced social acceptance of the child was measured with a three-item, four-point Likert scale constructed specifically for their study. The three items were whether they perceived neighbours and acquaintances of the family would accept the child as: 1) the friend of their children of the same age, 2) the classmate of their children of the same age and 3) a neighbour when the child reached adulthood. The results indicated that the way in which parents perceived their situation, and perceptions of the child's acceptance by others were more important in the prediction of parental stress than other challenges such as the child's illness related behaviours. This finding is important when considering the possibilities for supporting family adaptation such as changing the attitudes or perceptions of the parents but perhaps more

importantly, those of the community. Spousal support was shown to be an important factor in family adaptation for both fathers and mothers.

Overall the model has been successfully used as a theoretical framework for detecting the degree of family stress and how parents cope with children with ASD (McStay et al., 2014; Pakenham et al., 2005; Pozo et al., 2014). It has been widely used to study the adjustment and adaptation process in families of children with disabilities, and has been used specifically in families of children with ASD (Manning, Wainwright, & Bennett, 2011). Manning et al. studied the relationships between ASD symptoms (A), problem behaviours (A), social support (B), coping (BC), and cognitive appraisals (C) in a large sample of 195 families living with a school-age child with ASD. Using these variables from the Double ABCX model they predicted adjustment (X) assessed as the level of parental distress and the level of family functioning. Family functioning was defined as the level of family cohesion, expressiveness and conflict with elements including life stress, autism severity, child behaviour severity, informal support and reframing. Their findings suggested that child behaviour problems and reframing were most strongly associated with family outcomes. Child behaviour problems were related to both family functioning and parental distress. Reframing is a cognitive appraisal strategy that can lead to positive affect by allowing one to see a situation in a more positive light (Folkman & Moskowitz, 2000) and it is linked to perception in the Double ABCX Model. The results of Manning et al.'s study showed that reframing allowed parents to view the situation of having a child with autism in a more positive light and related to lower parenting stress.

In other studies, involving families living with a child with ASD, Siman-Tov and Kaniel (2011) found a significant positive correlation between the severity of a child's symptoms and parents' stress. Others (e.g., Lecavalier et al., 2006) have reported contrary results related to the association between the child's behaviour and parental stress and Hastings, Kovshoff, Brown, Ward, Espinosa, and Remington (2005) found that the child's adaptive behaviour was unrelated to parental stress where an inverse relationship was expected.

Although the Salutogenic Model (Al-Yagon, & Margalit 2009; Oelofsen, & Richardson, 2006; Olsson & Hawang 2002), FAAR Model (Weiss et al., 2015; Xue et al., 2014), Contextual Model of Family Stress (Sullivan, 2015) and the Double ABCX Model (Bristol, 1987; Stuart & McGrew, 2009; Paynter et al., 2013) have been used successfully in several studies of adaptation and outcomes in families of children with ASD, the existing research has been limited by the tendency to focus on a single outcome or family burden and a failure to utilise situation specific measures, the importance of which was suggested by Bandura (1986).

Additionally, Bandura (1986) although referring to self-efficacy, argued that it is important to utilise domain specific scales where possible. Although scales used in past research typically have good psychometric properties, none are domain-specific to families living with a child with ASD. It is important therefore that such instruments be developed in line with Bandura's (1986) argument for domain specificity in assessment to better understand the impact on families living with a child with ASD.

5.6 Proposed Model

A review of the models presented in this Chapter has revealed several commonalities in the factors comprising each model as well as those found to be significant predictors by past researchers.

Based upon this review, it seems that the availability of resources (ABCX Model (Hill, 1949); Contextual Model of Stress (Boss, 1987); Double ABCX Model, (McCubbin, & Patterson, 1982; 1983) and coping (SOC, Antonovsky, 1993; FAAR, Patterson, 1988; Double ABCX Model McCubbin, & Patterson (1982; 1983), which together are called capabilities in the FAAR model, are integral elements in how families manage their stressors. Interestingly, Masten (2009) considered coping as an element of resilience while others (e.g., Fisman, Wolf, Ellison & Freeman, 2000; Giallo & Gavidia-Payne, 2006) have seen both coping and resources as protective factors against risk in the family.

The result of stress, typically mediated by coping and support, has variously been called crisis/adaptation (ABCX Model, Hill, 1949), tension (Salutogenic Model, Antonovsky, 1979); adjustment/adaptation (FAAR, Patterson, 1988; Double ABCX Model, McCubbin & Patterson, 1983). From these models and the extant literature relevant to the impact of living with a child with ASD, the following model is proposed (Figure 5.6). The model is couched within the à prior framework of a family living with a child with ASD and the potential extant stressors that these families experience. As such no assessment is made of perceived or actual stressors or their pileup. Rather the focus is on the resources and coping strategies, that is, the capabilities, families used to reduce the impact of living with the demands associated with living with a child with ASD.

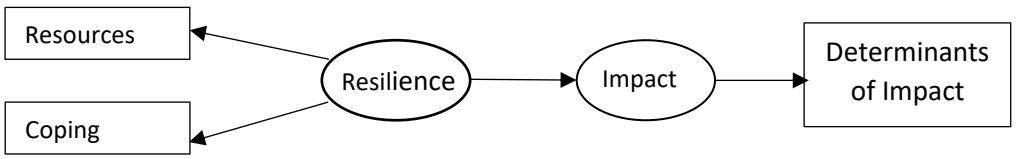

Figure 5.6: Proposed Model of the Impact on Families Living with a Child with ASD

Resilience is included in the proposed model as it is argued that proactive coping strategies combined with resources contribute to a sense of resilience in the face of stressors. As such, resilience is an important protective and adaptive factor in people's ability to deal with stressors, no less so family stressors. Research has also shown that resilience is predictive of successful adaptation among families of children with ASD (Weiss, 2002).

Furthermore, it is important to examine the impact of living with a child with ASD using specific measures derived from the experience of families whose child has ASD. The essential items to be considered in a hypothesised model of living with a child with ASD are the roles of support and coping as indicators of family resilience, and its relationship with impact, as defined by factors such as relationship satisfaction (Baker, Seltzer, & Greenberg 2012) and financial costs (Rogge & Janssen, 2019) which may emerge from interviews with family members. Prior to this, it is important to review extent measures for their specific utility in such an exploration.

5.7 Summary

In summary, a number of models of the impact of family stress or family adaptation to stress have been proposed and several of these have been used, in whole or in part, to study the impact of living with a child with ASD. Hill's (1949) ABCX model is the earliest model used to study family stress and subsequent others include the Salutogenic Model (Antonovsky, 1979), the FAAR Model (Patterson, 1988), the Conceptual Model of Stress (Boss, 1987) and the DABCX Model (McCubbin & Patterson, 1983). There are several commonalities across these models including the role of family support and coping, with both of these factors contributing to people's capabiities which, in turn, reduce the impact of the stressors or demands on the family thus leading to greater adaptation or less tension. A composite model of the Impact of Living with a Child with ASD is proposed based on the commonalities across the models reviewed. The importance of reviewing measures that assess the elements of these models and appropriate assessment measures is imperative but particularly, identifying domain-specific measures as recommended by Bandura (1986).

Chapter 6. Measures of Family Impact

Much research has focused on the aetiology (Cannell, 2008; 2017; Emberti Gialloreti et al., 2019; Hughes, Rose, & Ashwood, 2018; Sandin et al., 2014; Volk et al., 2013), prevalence (Baio, 2012; Baron-Cohen et al., 2009; Wingate et al., 2014), comorbidities (Díaz-Román, Zhang, Delorme, Beggiato, & Cortese, 2018; Frye & Rossignol, 2016; Mannion & Leader, 2013; Miano et al., 2016) and the functional challenges (Calderoni et al., 2014; Condy, Scarpa, & Friedman, 2019; Minshawi et al., 2015) associated with ASD as well as assessing the impact of ASD on those who experience it (Hill et al., 2014; Kozlowski, Matson, & Rieske, 2012; Rattaz, Michelon, Munir, & Baghdadli, 2018). Among these assessments is the Autism Spectrum Disorders-Behavior Problems for Children checklist (Matson, Gonzalez, & Rivet, 2008) which parents rate in terms of their child with ASD. Higher scores are indicative of more behavioural problems exhibited by the child and the greater severity of ASD. While this measure focuses on the child's behaviour it does not assess the impact that these behaviours have on the family, per se. In fact, scant research to date has focused on the development of specific scales to assess the impact on families who are living with a child with ASD. Most scales used in past research, even research specifically related to families with a child with ASD, are of a more general nature as discussed below.

6.1 Unsupportive Social Interactions Inventory (USII)

Each of the models reviewed in the previous chapters included support or resources which can include support, as an integral component of how families deal with stressors. In contrast to this, Ingram, Betz, Mindes, Schmitt, and Smith (2001) developed the 24-item Unsupportive Social Interactions Inventory (USII) to measure unsupportive social interactions perceived by people experiencing an illness or stress. The USII has four subscales: distancing (e.g., people did not seem to want to hear about it), bumbling (e.g., people did not seem to know what to say), minimising (e.g., people said that I should look on the bright side), and blaming (e.g., other people 'should' or 'shouldn't have comments about my role in the event) which are rated on a scale from 0 = *none* to 4

= *a lot*. The internal reliability for the total scale is α=.86 and for the subscales, distancing α=.78, bumbling α=.73, minimising α=.76 and blaming α=.85 (Ingram et al., 2001).

The USII has been used to assess the interpersonal interactions between a person experiencing a stressful life event and how the people in his/her social network respond to the stressful circumstance. An example of a stressful life event is the onset of a disease in the individual or a member of their family. Ingram, Jones, Fass, Neidig, and Song (1999) used the scale with 271 individuals diagnosed with Human Immunodeficiency Viruses (HIV) to investigate the level of unsupportive or upsetting responses that a person with HIV perceives from those in their social and work networks. They tested the hypothesis that unsupportive social interactions would account for a significant amount of the variance in depression beyond the variance explained by physical functioning and social support. The factors of the USII were correlated, ranging from r = .53 to .80 and its factors demonstrated adequate internal consistency (α ≥ .93). The multiple regression analysis revealed that scores on the USII predicted significant variance in depression. Specifically, physical functioning explained 6% of the variance in depression, with lower levels of physical functioning being associated with increased depression; social support accounted for 25% of the variance with higher levels of satisfaction with support being related to decreased depression while unsupportive social interactions accounted for an additional 15% of the variance in depression beyond that explained by physical functioning and social support. Together, the factors in the model explained 46% of the variance in depression experienced by individuals diagnosed with HIV.

McInnis, McQuaid, Matheson, and Anisman (2017) conducted a cross-sectional study among 476 undergraduate students to examine associations between the oxytocin receptor (OXTR) rs53576 and CD38 rs3796863 variant polymorphisms and unsupportive social interactions and mood states. Saliva samples for DNA were collected and genomic DNA was extracted for OXTR and CD38 sequences. The original sample size was 476 but there were 13 individuals for the OXTR polymorphism and 14 individuals for the CD38 polymorphism for whom the genotype could not be

determined. As such, subsequent analyses involving the OXTR and CD38 polymorphisms comprised 463 and 462 individuals, respectively. They used the USII to investigate the participants' received negative or upsetting responses when talking to peers or parents about events in their life in recent weeks. In their study the Positive and Negative Affect Schedule scale of Watson, Clark, and Tellegen (1988) was used to examine the mood states of participants.

The OXTR polymorphism was not associated with perceptions of unsupportive social interactions, a finding consistent with an earlier report from McInnis, McQuaid, Matheson, and Anisman (2015) indicating that this polymorphism was not directly related to perceived levels of unsupportive interactions. Also consistent with an earlier report from Tabak et al. (2016) was that the genotype of the CD38 polymorphism promoted greater social sensitivity to chronic interpersonal stress. McInnis et al. concluded that, despite the observation that CD38 polymorphism was associated with higher perceptions of peer unsupportive responses, their findings did not suggest a stronger association between perceptions of unsupportive social interactions and current negative affective states. Neither was a moderating effect for either polymorphism (OXTR and CD38) apparent when assessing unsupportive social interactions from parents as the predictor, or positive affect, as the outcome. McInnis et al. stated that their findings were entirely correlational and it was unclear whether negative mood was a result of experiences of unsupportive interactions or whether mood states influenced participants' perceptions of their social interactions.

Talebi, Matheson, and Anisman (2016) investigated perceptions of negative social interactions using a shortened version of the USII. Participants were first year students experiencing the transition stressor associated with entry to university operationalised in terms of stigma. Students rated the extent to which they encountered each of eight behaviours from their peers (e.g., my partner/close friend refused to provide the type of help or support I was looking for) on a five-point scale ranging from 0 = *never* to 4 = *all the time*. The findings revealed that peer social support negatively loaded on each of three variables to assess stigma: self-stigma academic (β = -.15), other

stigma (β = -.13), and other stigma academic (β = - .26). Conversely, unsupport loaded positively on each of these three measures of stigma (β = .13; .19; and .23, respectively). Talebi et al. also investigated peer support and unsupport on self-stigma for mental health but only unsupprt was predictive of this factor (β = .12). The role of parental support on the four stigma variables was also tested but there was no effect.

In a second study, Talebi et al. (2016) also found that individuals who reported depressive symptoms were most sensitive to perceiving stigma, and that the impoverished social experiences and ineffective coping styles associated with depressive symptoms played a key role in predicting perceptions of stigma. Although positive support perceptions facilitated problem-centred coping and lower self-stigma, students' unsupportive encounters with their peers were expected to trigger a cascade of maladaptive responses, including emotion-focused behaviours that serve to heighten the tendency to self-stigmatise the need for help. Overall, they found that impoverished social support resources and coping strategies associated with depressive symptoms were predictive of greater perceived stigma of help-seeking which, they stated, likely creates obstacle to accessing appropriate professional care by those who are most in need. The focus with each of these studies using the USII, has been on the person experiencing the stress or illness and not their family or their carers. USII has been used with parents of children with ASD as well (e.g., Jones, 2018; Pottie, Cohen, & Ingram, 2008).

Pottie et al. found that unsupportive interactions were negatively associated with positive mood. Jones (2018) found that unsupportive social interactions were not significantly predictive of Quality of Life in families with a child with ASD, but in those parents with poor coping strategies there was an effect.

While these studies provide some support for the utility of the USII for use with families with a child with ASD, the items are not domain specific.

6.2 Multidimensional Scale of Perceived Social Support (MSPSS)

The Multidimensional Scale of Perceived Social Support (MSPSS) is a 12-item self-administered scale which aims to evaluate people's perceived social support (Zimet, Dahlem, Zimet, & Farley, 1988; Zimet, Powell, Farley, Werkman, & Berkoff, 1990) from three different sources: family, friend, and significant other. There are four questions relating to each source (e.g., there is a special person who is around when I am in need; there is a special person with whom I can share joys and sorrows; my family really tries to help me, and I have a special person who is a real source of comfort to me). The reliability of the total scale is excellent, Cronbach's α=.88 and for each of the subscales alphas were ≥ .85 (Zimet et al., 1988). The MSPSS has primarily been used to assess individuals' perceptions of support suggesting that it could be suitable to assess support perceived by carers or families living with a child with ASD, although the items are generic.

Several studies have supported the psychometric properties of the MSPSS in diverse populations. Dahlem, Zimet, and Walker (1991) examined its psychometric properties in a group of 154 students at an urban college. They found that the MSPSS had good internal reliability: the coefficient alpha for the total scale was α = .91; for the Family, Friends, and Significant Other subscales the alphas were .90, .94, and .95, respectively. Also factor analysis confirmed the three factor structure of the measure: family, friends, and significant other. For students who scored high on stress, social support and depression were significantly negatively correlated (r = - .345, p < .002); for students who scored low on stress, the social support-depression correlation was not significant (r = .016, p = ns). The former result is congruent with the loneliness and negative perceptions prevalent in people with depression. The lack of correlation for those scoring low on stress might be because social support exerts either a direct or a buffering effect. Neither of these propositions were tested by Dahlem et al. They did however, indicate that there was no social desirability bias effecting students' responses and further acknowledged that their correlational data do not allow for attributions of causality (Dahlem et al., 1991).

Canty-Mitchell and Zimet (2000) also investigated the psychometric properties of the MSPSS in 222 adolescents, predominantly African-American (68%). Factor analysis confirmed the three subscale structure and they demonstrated high internal consistency ($\alpha \geq .89$). The correlations among the three factors ranged from $r = .53$ to $r = .66$. The validity of the MSPSS was assessed using the Adolescent Family Caring Scale. The Adolescent Family Caring Scale correlated significantly with the MSPSS Family subscale ($r = .76$), Friends subscale ($r = .33$), and Significant Other subscale ($r = .48$). The stronger correlation between the family scales suggested good convergent validity while the lower correlations of friends and significant other with the Adolescent Family Caring Scale suggest their discriminant validity.

Alon (2019) used the MSPSS to investigate the relationship between social support and post-crisis growth among mothers of children with ASD and mothers of children with Down's syndrome. They used the type of disability as a moderating variable between support and growth and, in more detail, the subtypes of support and growth. Participants were 99 mothers of children with ASD and 119 mothers of children with Down's syndrome. Reliability for the questionnaire in their study was high, Cronbach's $\alpha = 0.91$ for overall score, $\alpha = 0.91$ for family support, $\alpha = 0.89$ for friends' support, and $\alpha = 0.80$ for support from other important individuals. They found that social support directly predicted maternal post-crisis growth ($\beta = .02$, $p < .05$) although the significance of this small Beta-weight is likely to be a function of sample size ($N = 219$). When Alon examined the interaction of social support by type of disorder (ASD vs Down's syndrome) they found that the impact of social support for the mothers of children with ASD was significantly more predictive of parental growth post crisis than for the mothers of the other children.

Paynter et al. (2013) used the Double ABCX model to study the factors underlying family outcomes of living with children with ASD. MSPSS was used to measure social support from a significant other, family and friends as indicators of the factor, Resources (bB), in the Double ABCX model. Participants included 43 parents (18 males, 25 females) of children aged 2.5 to 6 years.

Participants' scores on the MSPSS indicated that greater social support was associated with lower levels of individual distress across all individual level variables except anxiety, including depression ($r = -.47$, $p < .001$), stress ($r = -.53$, $p < .001$), and parenting stress ($r = -.60$, $p < .001$). Greater social support was also associated with better relationship quality ($r = .51$, $p < .001$) and lower negative impact on family ($r = -.63$, $p < .001$).

The MSPSS is clearly valid for assessing perceptions of support by individuals and by caregivers, however, the entire family may be affected by the illness or disability of a child. The following scales were designed to focus on the family.

6.3 Family Crisis Oriented Personal Evaluation Scales (F-COPES)

McCubbin, Olson, and Larsen (1981) developed the 30-item Family Crisis Oriented Personal Evaluations Scales (F-COPES) to identify problem solving and behavioural strategies utilised by families in difficult or problematic situations. Questions are answered on a 5-point Likert scale from 1 = *strongly agree* to 5 = *strongly disagree*. There are five subscales: Acquiring Social Support (e.g., sharing concerns with close friends), Reframing (e.g., accepting stressful events as a fact of life), Seeking Spiritual Support (e.g., having faith in God), Mobilising Family to Acquire and Accept Help (e.g., seeking information and advice from the family doctor), and Passive Appraisal (e.g., believing if we wait long enough, the problem will go away). Higher scores represent an increase in the number of coping strategies used by the family. Reliability for the F-COPES total scale's Cronbach's $\alpha = .86$, and the test-retest reliability over 4-5 weeks is $r = .81$ (McCubbin et al., 1981).

Nabors, Cunningham, Lang, Wood, Southwick, and Stough (2018) used F-COPES to assess family ($n = 61$) coping when a child with a chronic illness was hospitalised for procedures related to his or her illness. Parents used cognitive strategies to see the hospital stay as positive for the child or to accept it as the possibility for improving the child's life. Some of the mothers mentioned financial stress was a difficulty experienced by the family at this time. Several studies have used the F-COPES with samples drawn from parents of children with ASD.

Lee (2009) measured coping differences in relation to psychosocial adjustment, that is levels of depression, anxiety, and marital adjustment between parents of children with (48) and without (26) ASD using F-COPES. They found significant differences between the ASD and control groups on depression, anxiety, one subscale (General Relational Satisfaction) of the marital adjustment measure (Dyadic Adjustment Scale), and coping approaches as measured by F-COPES and the Coping Health Inventory for Parents. Parents of children with ASD exhibited less adaptive coping skills compared to parents of children without any disability. A comparison of parents of children with ASD revealed mothers exhibited more adaptive coping skills compared to fathers.

McStay et al. (2014) explored the potential predictors of maternal and paternal stress and family quality of life in 196 parents (98 mother–father dyads) of children with ASD via a questionnaire package based on the Double ABCX model. F-COPES was used to assess the coping (BC) factor for predictors of stress and proactive coping predicted 2% of the variance in family quality of life of parents.

Manning et al. (2011) used F-COPES to assess the Coping Strategies, again as a component (BC) of the Double ABCX model. Their study included 195 primary caregivers of children with ASD who completed three subscales of the F-COPES. The first subscale measured the family's ability to actively engage in acquiring support from relatives, friends, neighbours and extended family (Coping by Relying on Family and Friends). The internal consistency for this subscale was α =.79. The second subscale was for the family's coping by utilising spiritual support (Seeking Spiritual Support). Internal consistency for this subscale was α = .89. The third subscale measured the family's ability to seek out community and professional resources and to accept help from them during times of stress (Coping by Seeking Community Support). This last subscale was not included in analyses due to low internal consistency. Manning et al. (2011) used the reframing subscale from the F-COPES to examine appraisal of the stressor (factor cC). In the sample, internal consistency for the reframing subscale was α = .78. These subscales were used in order to identify problem solving and behavioural

strategies utilised by families in difficult or problematic situations. They also measured the degree to which families utilised various sources of social support as a means of coping with stressful situations. The authors found that outcomes of both family functioning and parental distress were significantly predicted by Coping by Relying on Family and Friends. Parents who reported using their family and friends as a means of coping with stress also reported higher levels of family functioning and reduced parental distress. Families who reported higher levels of coping through spiritual support (e.g., attending religious services, prayer) also reported lower parental distress. Reframing was a significant predictor of both family functioning and parental distress. Manning et al. concluded that reframing may allow parents to view the situation of having a child with ASD in a more positive light. In their study, a large percentage of parents endorsed reframing strategies including 65% of parents who reported that they defined the problem in a more positive way, that is they may have seen it as a challenge, and 71% of parents who reported the belief that their family had the power to solve major problems (Manning et al., 2011). These percentages were not reported as mutually exclusive.

6.4 Family Support Scale (FSS)

Dunst, Jenkin, and Trivette (1984) developed the 18-item Family Support Scale (FSS) which family members answer with respect to different sources of support they have received while raising a young child. Questions are answered on a 5-point Likert scale to rate the perceived helpfulness of supports drawn on over the past 6 months. If the source of support is unavailable, then the option of NA (not available) is given. Participants addressed 18 sources of support (e.g., my parents; my spouse/partner's parents; my friends; my spouse/partner's friends) regarding "how helpful has each of the following been to you in terms of raising your child"?

The sources of support are grouped into informal supports such as partner, friends, co-workers and formal supports such parent groups, church, early childhood programmes. This scale

has acceptable internal consistency, Cronbach's α = .77, and test-retest reliability ranges from *r* = .41 to .75 across subscales (Dunst et al., 1984).

Hanley et al. (1998) investigated the construct of family support using the FSS among 244 low-income families of children who participated in Head Start programs. They extracted five subscales which they labelled: (1) community; (2) spouse and in-laws; (3) friends; (4) specialised/professional; and (5) own parents and extended family. Factor analysis shows that these five factors accounted for 61% of the variance as follows: community (30.6%); spouse and in-laws (11.5%); friends (7.1%); specialised/professional (5.7%); and own parents (5.7%). The internal consistency and test-retest reliability were moderately high for each of the five subscales. Cronbach's alphas for the total score was α = .85 and a test-retest reliability of *r* = .73 for its total score.

Searing, Graham, and Grainger (2015) examined the perceived availability and helpfulness of supports used by 92 caregivers of children with ASD in New Zealand using the FSS. They found that spouses were rated as the most helpful source of support and professional helpers were rated as somewhat helpful. Helpful support emphasised caring, knowledge and accessibility. They also found ethnic (Māori) differences in perceptions of formal supports (e.g., parent groups, church, early childhood programmes) and informal supports (e.g., spouse/partner, friends, co-workers) compared with Caucasian participants where Māori participants reported support was more highly valued.

In addition to F-COPES discussed earlier, Manning et al. (2011) used FSS in their adaptation of the Double ABCX model's resources (bB) factor for two types of parent social support. These were Informal Support (family, friends, parents and other non-professionals) and Formal Support (agencies, and other professionals). Internal consistency in this sample was α = .74 for the Informal Support Scale but the authors failed to include the Formal Support Scale in further analyses due to its low internal consistency in their data.

Clearly, family support is a central construct related to a number of important research and clinical issues, such as overall family functioning, family well-being, parental perceptions of child functioning, and family integrity (Dunst, 1985).

6.5 Family Environment Scale

Family Environment Scale (FES) (Moos & Moos, 1981; Moos, 1990) has been used to assess the social climate of families (Lanz & Maino, 2014) that is, the measurement of the interpersonal relationships among family members, the directions of personal growth which are emphasised in the family, and the basic organizational structure of the family (Moos, Insel, & Humphrey, 1974). This scale is comprised of 90 true or false items which are organised into 10 subscales: Cohesion (the extent to which family members are concerned and committed to the family and the degree to which family members are helpful and supportive of each other), Expressiveness (the extent to which family members are allowed and encouraged to act openly and to express their feelings directly), Conflict (the extent to which the open expression of anger and aggression and generally conflictual interactions are characteristic of the family), Independence (the extent to which family members are encouraged to be assertive, self-sufficient, to make their own decisions and to think things out for themselves), Achievement Orientation (the extent to which different types of activities are cast into an achievement oriented or competitive framework), Intellectual-Cultural Orientation (the extent to which the family is concerned about political, social, intellectual and cultural activities), Active-Recreational Orientation (extent to which the family participates actively in various kinds of recreational and sport activities), Moral-Religious Emphasis (the extent to which the family actively discusses and emphasized ethical and religious issues and values), Organisation (measures how important order and organization is in the family in terms of structuring the family activities, financial planning and explicitness in regard of rules and responsibility) and Control (the extent to which the family is organized in a hierarchical manner, the rigidity of family rules and procedure). These subscales form three conceptual domains: relationship dimension (comprising cohesion,

expressiveness and conflict); personal growth dimension (independence, achievement orientation, intellectual cultural orientation and active recreational orientation); and system maintenance dimension (moral-religious emphasis, organisation and control) (Lanz & Maino, 2014). The FES subscales have good 8-week test-retest reliabilities varying from *r* =.73 to .86 (Moos, 1990). The internal reliabilities are good and the overall alpha is .79 (Moos, 1990).

Baker, Seltzer, and Greenberg (2012) used the FES to measure the association between behavioural problems in the children with fragile X syndrome with maternal internalising (i.e., depressive and anxiety symptoms) which would, in turn, relate negatively to both marital satisfaction and family cohesion. They measured these family variables in 115 married mothers of adolescents and adults with fragile X syndrome with 33 individuals having both fragile X syndrome and ASD. The mothers were asked to rate each statement as true or not true of their family. Items included statements such as: "Family members really help and support one another" and "There is very little group spirit in our family". Items reflecting low cohesion were reverse coded and added to remaining items such that potential scores could range from 0 = *low cohesion* to 9 = *high cohesion*. Results showed that behavioural problems were positively related to maternal internalising symptoms which were, in turn, negatively associated with both family cohesion and marital satisfaction. They found no direct associations between behavioural problems and family relationships.

Rao and Beidel (2009) used the FES to assess the family environment from the perspective of all family members along three dimensions: relationship (the degree of support families provide for their members), personal growth (the degree to which family members are self-sufficient and make their own decisions), and system maintenance (the degree of importance placed on family organization and structure). A sample of 15 parents of children with ASD and 15 parents of children with no known disability were matched according to their child's age, race, IQ, and family income. In addition, the siblings of each child in both groups were also assessed. Rao and Beidel used the FES

for the parents, and the siblings under the age of 12 completed a children's version of the FES. The findings indicated that parents of children with ASD experienced significantly more parenting stress than parents of children without ASD and with no other psychological or physical disorder. They also found that for siblings, the behavioural problems of the child with ASD were associated with internalising problem behaviours in the siblings.

Kelly, Garnett, Attwood, and Peterson (2008) used the FES to examine the potential impact of family conflict and cohesion, and peer support/bullying on children with ASD to determine whether these factors exacerbated or ameliorated ASD symptomatology and anxiety/depression in these children. They measured conflict and cohesion, two subscales of the FES, to assess the impact on children with ASD. These two subscales each consist of nine items (e.g., conflict subscale: "We fight a lot in our family"; cohesion subscale: "Family members really help and support one another") and items were rated as true or false. The sample consisted of 322 parents of children with ASD who completed the questionnaires on-line in terms of their child with ASD. The key findings were that anxiety/depression and ASD symptomatology were significantly related, and family conflict was more predictive of ASD symptomatology than positive family/peer influences. Of course, these data may not represent the child's actual view, rather they reflect the parents' perceptions.

6.6 Relevance of Existing Scales

Several researchers have used various scales to assess elements of the Double ABCX and other models, including levels of social support, family environment and family coping with varying results. It is important to note that none of these scales have been specifically designed to address the strategies and issues faced by families living with a child with ASD. Bandura (1986) although referring to self-efficacy, argued that it is important to utilise domain specific scales where possible. Accordingly, it is important that such instruments be developed in order to better understand the impact on families living with a child with ASD.

Several researchers have used various scales to assess elements of the Double ABCX and other models, including levels of social support, family environment and family coping with varying results. Furthermore, the Salutogenic Model (Al-Yagon, & Margalit 2009; Oelofsen, & Richardson, 2006; Olsson & Hawang 2002), FAAR Model (Weiss et al., 2015; Xue et al., 2014), Contextual Model of Family Stress (Sullivan, 2015) and the Double ABCX Model (Bristol, 1987; Stuart & McGrew, 2009; Paynter et al., 2013) have been used successfully in several studies of adaptation and outcomes in families of children with ASD, but the existing research has also been limited by the tendency to focus on a single outcome of family burden and a failure to utilise domain specific scales. The scales discussed above while having good psychometric properties are not domain-specific to families living with a child with ASD. It is important therefore that such instruments be developed for, although referring to self-efficacy, Bandura (1986) argued for the need for specificity in assessment.

6.7 Chapter Summary

In this chapter a review of scales to assess families and the impact of a disorder/stress on them was discussed. Among these scales was the Unsupportive Social Interaction Scale, Multidimensional Scale of Perceived Social Support, Family Crisis Oriented Personal Evaluations Scales, Family Support Scale and Family Environment Scale. For the most part, these scales have been of a general nature assessing perceptions of support and coping among families who have a family member with a disability, a family under stress, and across ethnicities. Several of these scales have been used in research involving families of children with ASD with varying results. Although these scales have good psychometric properties, none are domain-specific to families living with a child with ASD. It is important therefore that such instruments be developed for, although referring to self-efficacy, Bandura (1986) argued for the need for specificity in assessment.

Chapter 7. Thesis Aims and Hypotheses

ASD is a debilitating lifelong condition which in 2010 was found to effect approximately one in 68 persons in the USA (Wingate et al., 2014) with the most recent figures from Australia showing one in 90 4-year-olds experience ASD (May & Williams, 2018). Much is known about the comorbidities associated with ASD (Frye & Rossignol, 2016; Mannion & Leader, 2013) but little is known of its aetiology. Cannell (2008; 2017) put forward the proposition that Vitamin D may be implicated in the onset and the maintenance of ASD. There is some literature which has shown that both maternal deficits in Vitamin D (Chen et al., 2016; Dealberto, 2011; Magnusson et al., 2016) and the administration of Vitamin D to those with ASD (Jia et al., 2015; Stubbs et al., 2016) provide some support for his hypothesis. Vitamin D is synthesised through the body's absorption of UVB through natural sunlight. It may be therefore, that as people's exposure to sunlight and hence UVB decreases with the distance from the equator, so too the prevalence of ASD increases. This proposition is worthy of investigation as, indirectly, it may suggest strategies to ameliorate the symptoms of ASD.

Not only are the children with ASD effected but so too are their families. Living with a child with ASD has an impact on the family which can restrict parents' work and social relationships as well as the family's finances. The impact on families may be greater for those living in rural and regional areas as they are often at a distance from medical and other specialist services. It is important to understand and assess family's perceptions of the impact of living with a child with ASD and the strategies they use to cope. This information will contribute to medical practitioners and other health care providers to develop strategies to assist families to mitigate or reduce the impact on the families. Several models have been proposed to assess families affected by stress, trauma or illness but typically these use scales of a generic nature rather than ones that are domain-specific as recommended by Bandura (1986).

The aims in this thesis were: 1) to explore the prevalence of ASD by latitude and 2) determine the impact on families living with a child with ASD. In order to achieve these aims, three studies

were undertaken: Study 1) a review of the extant literature to explore the prevalence rates of ASD by latitude to determine if, at the broadest level, there is a relationship between sunlight/UVB and the prevalence rates of ASD; Study 2) a qualitative study where families who have a child with ASD were interviewed in order to understand their concerns, the impact of their child's disability on the family and their coping strategies; and Study 3) to develop and psychometrically test measures related to living with a child with ASD derived from the interview data collected in Study 2, and finally, as an extension of Study 3, to test a synthesised model of Living with a Child with ASD using the domain-specific scales developed from parents' input.

From the literature, it is generally hypothesised that:

1. The incidence of ASD across the globe will increase as latitude increases, that is, as levels of UVB decrease with distance from the equator;
2. Reports from a qualitative study will reveal the difficulties faced by parents of children with ASD, who live in a regional area, and the coping strategies they used to address these difficulties; and
3. The Model of Living with a Child with ASD will provide indicators of the impact on families but also the factors which may mitigate these impacts using domain-specific scales derived from the qualitative study.

Chapter 8. Study 1: Prevalence of ASD by Latitude[1]

The aetiology of ASD is unknown but in 2008 Cannell proposed that a deficiency in Vitamin D could contribute to its aetiology as Vitamin D regulates dozens of proteins involved in brain development. Since then several researchers have reported low levels of Vitamin D in individuals with ASD (Want et al., 2016). In line with investigations into Vitamin D deficiency, recent research has found a link between solar irradiance and ASD (Grant & Cannell, 2013; Grant & Soles, 2009; Mazahery et al., 2016). Solar irradiance is the measure of the sun's electromagnetic spectrum over all wavelengths per unit area, usually described in watts per meter^2 (W/m^2) units (Kimlin, 2008). The earth's atmosphere filters the sun's electromagnetic radiations leaving Ultraviolet (UV), Visible, and Infrared radiations as the main biogenically relevant components to reach the earth's surface. UV is further classified into UVA, UVB and UVC (Kimlin, 2008; Kulms & Schwarz, 2002). UVA reaches the earth's surface throughout the year, UVB reaches the surface only when the sun is high in the sky, while UVC is completely blocked by the earth's atmosphere (Kimlin, 2008).

When people are exposed to UVB radiation, 7-dehydrocholesterol present in the skin is converted to pre-Vitamin D3 and provides the main source of natural Vitamin D to the body (Grant & Cannell, 2013). Some research has reported a correlation between low solar UVB and high prevalence rates of ASD (Grant & Cannell, 2013; Grant & Soles, 2009; Mazahery et al., 2016) with these higher prevalence rates attributed by these researchers to lower levels of sunlight based Vitamin D production (Grant & Cannell, 2013) These studies were however, limited in scope, as, they focused only on the UVB based Vitamin D part of solar irradiation, commonly referred to as sunlight.

The aim of this study was to provide support for Cannell's (2008; 2017) hypothesis that a deficiency in Vitamin D, which is synthesized in the body from sunlight, is implicated in either the onset or maintenance of ASD. Sunlight, otherwise referred to as solar irradiance, contains the

[1] Syed, S., Moore, K. A., & March, E. (2017). A review of prevalence studies of Autism Spectrum Disorder by latitude and solar irradiance impact. *Medical Hypotheses, 109*, 19-24. (See Appendix A)

important UV needed to process Vitamin D. As sunlight decreases with distance from the equator, it is suggested that the prevalence rates of ASD will increase correspondingly. That is, the prevalence of ASD will be higher at higher latitudes.

8.1 Design

A review of the literature was conducted in order to identify studies reporting on the prevalence of ASD. These prevalence rates were then plotted by latitude.

8.2 Method

The major databases of PubMed, Google Scholar, ScienceDirect, Medline, Psychinfo and a non-content database (Web of Knowledge) were searched to access reports on the prevalence of ASD in children and adolescents. The search terms of "Autism" and "incidence/prevalence/epidemiology" were used. Subsequent searches included the name of countries/regions where gaps in geographical areas were identified.

8.2.1 Inclusion Criteria

In order to minimise the impact of any unknown factors and diversity in earlier assessments, only the latest reports based on comparable diagnostic criteria were reviewed for this study.

Each study revealed by the search was examined individually and included in the analysis if it met the following criteria: 1) Original publication between 2011 and 2016; 2) reported on samples less than 18 years of age; 3) used recognised diagnostic criteria (i.e., DSM-IV or ICD-10) or nationally modified diagnostic criteria for determining the prevalence of ASD; 4) was representative of a reasonably defined geographic area, that is, the spread of data collection was written 10 degrees of latitude (for determination of latitude and solar irradiance levels); and 5) represented the largest sample size.

In sum 25 studies met all the inclusion criteria. A further study from Australia that did not meet the inclusion criteria due to its wide geographical coverage exceeding 10 degrees of latitude will be discussed separately.

8.3 Results

The locations of the 25 studies reviewed are identified on the map in Figure 8.1. Although prevalence rates from some regions that is, Russia and southern Africa, are not represented, with respect to street level solar irradiance at latitudes, the entire globe is represented (Figure 8.1). An overview of the studies extracted for use in this study is listed in Table 8.1. This table includes age of participant, the prevalence of ASD and the diagnostic criteria used in each study. The prevalence rates were plotted by the respective latitude of the sample source and solar irradiance level are shown in Figure 8.2.

Figure 8.1: Black dots depicting the location of prevalence studies cited in this review

Table 8.1: Worldwide reports of prevalence of ASD (%) by latitude

Latitude	Area	Age	ASD Prevalence (%)	Sample Size	Criteria	Reference
00°14′	Quito, Ecuador	5-15	0.11	51,453	DSM-III, DSM-IV	Dekkers et al., 2015
8°-10°	Costa Rica	1-5	0.05	290,335	M-CHAT, VBAS	Schelly et al., 2015
10°77′	Shoranur, India	1-15	0.31	8,362	DSM-IV-TR	Poovathinal et al., 2016
21°07′	Leon, Mexico	8	0.11	4,431	DSM-IV-TR	Fombonne et al., 2016
21°26′	Taif, Saudi Arabia	7-12	0.04	22,950	AASQ	Al-Zahrani 2013
23°07′	Atibaia, Brazil	7-12	0.27	10,503	ASQ	Paula et al., 2011
17°-24°	Oman	0-14	0.01	798,913	DSM-IV-TR	Al-Farsi et al., 2011
22°-25°	Taiwan	0-17	0.29	372,642	ICD-9	Chien et al., 2011
28°9′	Las Palmas, Canary Islands	1-3	0.61	1,796	M-CHAT/ES	Fortea et al., 2013
29°-33°	Israel	8	0.49	2,431,649	DSM-IV-TR	Raz et al., 2015
32°26′	Himachal Pradesh, India	<11	0.09	11,000	ISAA	Raina et al., 2015
32°54′	Tripoli, Libya	1-10	0.31	38,508	DSM-IV	Zeglam & Maouna 2012
33°-35°	Fukuoka-Tokyo, Japan	3	2.54	2,516	DSM-IV-TR	Kamio et al., 2015

33°53′	Beirut, Lebanon	1-4	1.5	998	M-CHAT	Chaaya et al., 2016
34°03′	LA County, USA	3-5	0.46	1,626,354	DSM-IV-R	Becerra et al., 2014
37°34′	Goyang, South Korea	7-12	2.64	55,266	ASSQ	Kim et al., 2011
39°55′	Beijing, China	6-10	1.19	737	CAST	Sun et al., 2015
40°45′	Utah, USA	4,6,8	0.65	226,391	DSM-IV	Bakian et al., 2015
43°25′	Haute-Garonne, France	7	0.37	307,751	ICD-10	van Bakel et al., 2015
49°-59°	Manitoba, Canada	0-14	1	307,900	DSM-IV-TR	Vehling et al., 2016
53°18′	South Dublin, Ireland	6-11	1.15	5,457	EPAP	Boilson et al., 2016
55°-60°	Scotland, United Kingdom	4-18	1.6	684,415	ICD-10, DSM-V	O'leary et al., 2016
59°19′	Stockholm, Sweden	0-17	1.44	495,864	DSM-IV, ICD-10	Idring et al., 2015
62°00′	Faroe Islands	7-16	0.94	7,128	DISCO-11	Kočovská et al., 2012
63°-66°	Iceland	11-15	1.2	22,229	ADI-R or ADOS	Saemundsen et al., 2013

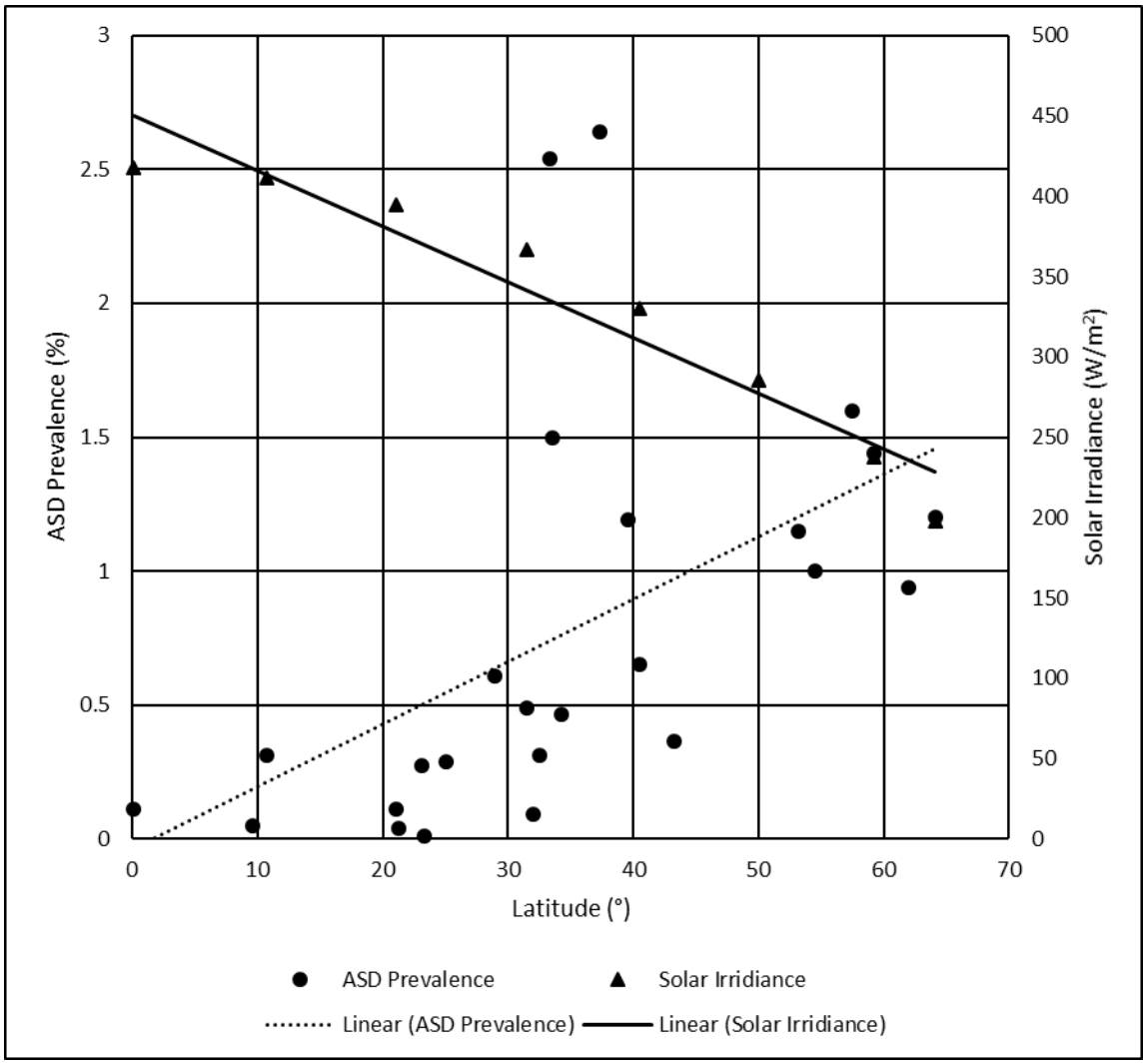

Figure 8.2: ASD prevalence and solar irradiance by latitude

8.4 Discussion

Cannell's hypothesis (2008), wherein he linked low levels of Vitamin D with ASD, was explored with respect to solar irradiation as a natural source of Vitamin D, and the results provide partial support for the exploration of this relationship. In general, the prevalence of ASD is shown to increase with increasing latitude while decreasing with increasing altitude. Fluctuations in both factors may be attributable to differences in elevation, pollution levels, annual solar irradiance, individual Body Surface Area (BSA) exposure, use of sunscreen protections, and skin pigmentation.

Overall, there is a tendency for ASD prevalence rates to increase and solar irradiance to decrease as the distance from the equator increases as shown in Figure 8.2. There are peaks in prevalence rates, which diverge from the line of best fit with Japan, South Korea and China having considerably higher prevalence rates of ASD while France has a lower rate (Table 8.1).

A notable finding is the increase in ASD prevalence above 25° latitude which maybe attributable to the decrease in solar irradiance which, until that point shows only a minor decrease. The other main finding is the continual increase of ASD prevalence above 33° latitude. During the winter months for those living above approximately 33° latitude very little if any Vitamin D_3 can be produced in the skin from sun exposure (Wacker & Holick, 2013) due to an increase in blockage of UVB by the atmosphere. Support for this premise can be seen in reports from Boston, USA (42 °N), Edmonton, Canada (52 °N), and Bergen, Norway (61 °N) where residents are reported to be unable to produce sufficient quantities of Vitamin D in their skin for four, five, and six months of the year, respectively (Holick, 2004; Wacker & Holick, 2013). Similar findings have been reported elsewhere (O'Neill et al., 2016; Stalgis-Bilinski, Boyages, Salisbury, Dunstan, Henderson, & Talbot, 2011; Seckmeyer, et al., 2013).

From the studies reviewed, Oman (Al-Farsi, Al-Sharbati, Al-Farsi, Al-Shafaee, Brooks, & Waly, 2011) has the lowest prevalence of ASD followed by Taif in Saudi Arabia (Al-Zahrani, 2013), Costa Rica (Schelly, González, & Solís, 2015), Himachal Pradesh in India (Raina, Kashyap, Bhardwaj, Kumar, & Chander, 2015), Quito in Ecuador (Dekkers, Groot, Mosquera, Zúniga, & Delfos, 2015), Leon in Mexico (Fombonne et al., 2016), Atibaia in Brazil (Paula, Ribeiro, Fombonne, & Mercadante, 2011), Taiwan (Chien, Lin, Chou, & Chou, 2011) and Shoranur in India (Poovathinal et al., 2016) in that order. All of these regions lie below 25° latitude except for Himachal Pradesh (India) and the prevalence rates were all less than 0.31%.

The Oman study was based on the nation-wide coverage of participants diagnosed at one centre. From 798,913 children aged 0–14 years, only 113 cases of ASD were found based on DSM-IV-

TR criteria. In addition to being close to the equator, Oman has a hot desert climate. Both of these may play a role in higher exposure to solar irradiance. The study from Saudi Arabia shows a prevalence rate of 0.035% from a sample population of 22,950 7–12 year-old students in the primary schools of the Taif district. The Costa Rican study was based also on a nation-wide sample (Rosero-Bixby, 2004; Savedoff, 2009) where, in the 1–5 year-old population, the prevalence of ASD was 118 children from a sample size of 290,335 that is 0.04%. The Himachal Pradesh study was carried out at 32° latitude in the Himalayan Mountains in India (Raina et al., 2015) and the only study beyond 25° latitude to have a prevalence rate of less than 0.3%. The reported low prevalence rate of 0.09% may be attributed to the elevation at which this study was carried out as at high elevation, solar irradiance (including UVB) is higher than at sea-level due to reduced travel-distance through the earth's atmosphere. Lower ASD prevalence at higher elevation further supports the argument for its association with solar irradiance. In this Indian study (Raina et al., 2015) 11,000 children aged 1–10 years in the identified region were surveyed by trained investigators using a Hindi language version of the Indian Scale for Assessment of Autism (ISAA) based on the Childhood Autism Rating Scale (CARS).

The authors of the Ecuadorian study (Dekkers et al., 2015) reported a prevalence rate of 0.11% in school going children of 5–15 years. The Leon (Mexico) study also found a low prevalence of 0.11% (Fombonne et al., 2016). This Mexican study has reported on ASD rates in mainstream school going children and special education school children. Paula et al. (2011) looked at ASD prevalence in a less urbanized region of Atibaia (Brazil), where they found a prevalence rate of 0.27%. The report from Taiwan is based on a sample of 372,642 children and adolescents aged younger than 18 years (Chien et al., 2011). The cumulative prevalence of ASD was found to be 0.287%. The other Indian study is from a semi-urban Indian region and reported a prevalence of 0.3% for 1–15 years old age-group (Poovathinal et al., 2016). Overall, the reported prevalence rates of these studies strengthen the findings with respect to the current interpretation of the data.

The rest of the studies show a continuous rise in the ASD prevalence corresponding to their increasing latitudes. In view of the fact that there is reduced UVB during the winter season above 33° latitude, an increasing trend beyond this latitude may indicate that the entire solar irradiance spectrum has a role in the ASD prevalence, otherwise, it could be expected that there would have been more or less uniform prevalence rates beyond 33° latitude.

In the regions above 33° latitude the data peaks for Japan, South Korea and China which all diverge from the line of best fit. They each have considerably higher prevalence rates of ASD while France, at 43° north has the lowest rate. South Korea has the highest prevalence in the reports reviewed in this study. The target population in that report included all children 7–12 years old (N = 55,266) in the Ilsan district of Goyang City near Seoul in South Korea (Kim et al., 2011). The Autism Spectrum Screening Questionnaire (ASSQ) was used and a prevalence of 2.64% was found. Data from two prospective community cohorts, namely Fukuoka Cohort and Tokyo Cohort were used in the study from Japan (Kamio, Haraguchi, Stickley, Ogino, Ishitobi, & Takahashi, 2015). Fukuoka Cohort comprised 1851 children and Tokyo Cohort comprised 665 children with 51 and 13 of them being confirmed as having ASD, respectively, using DSM-IV-TR. The prevalence rate across the combined samples was 2.54%. In the Chinese study, the Mandarin Childhood Autism Spectrum Test (CAST) was used with 737 pupils aged 6–11 in two mainstream primary schools in Xicheng District in Beijing (Sun et al., 2015). The prevalence rate was 1.19%. If low solar irradiance reaching the street level due to high pollution and smog levels in Beijing is considered, the high prevalence may be explained. South Korea and Japan do not have such high levels of pollution, yet their ASD prevalence rates are the highest in the reports reviewed. Weather might play a role, such as the longer and harsher winters experienced in these regions which lead to less BSA exposure or longer rainy seasons with cloud cover over 5.5 octa (Parisi, Turnbull, & Turner, 2007) which would further reduce UVB exposure days. The additional clothing required to keep warm in these harsher climates reduces the skin area exposed to sunlight, and may reduce vitamin D levels (Alagöl et al., 2000), however, this and other conjectures need to be investigated before drawing any conclusions.

It is important that future studies heed these factors in order to accurately judge the likely impact of solar irradiance on ASD. Since these countries are generally populated by the same race, it might also be that their race is more susceptible to ASD than the rest. This suggestion would need to be studied in comparison with Taiwan's ASD prevalence which is much lower (0.287%) at 25° latitude (Chien et al., 2011). Its subtropical climate with year-round high solar irradiance is also important to consider in future comparative studies.

The other noticeable exception from the increasing trend line is Haute-Garonne in France at 43° latitude. It has lowest prevalence of 0.365% (van Bakel et al., 2015) in regions above 33° latitude. This low rate merits further exploration and may relate to these children engaging more in outdoor activities and the local climatic/geographic factors. While van Bakel et al. (2015) cited ICD-10 as the diagnostic criteria used to assess prevalence of ASD their figures were drawn from a French area data base. This database itself may have contained inaccuracies or, of some concern, is Danielle Langloys' (the Presidente of Austisme France) suggestion that France in general lacks appropriate diagnostic criteria for ASD and its incidence is underrated.

The five studies from northern Europe are from above 50° latitude. All of them have a prevalence rate of 1% or higher with Faroe Island having the lowest rate followed by South Dublin (Ireland), Iceland, Scotland (United Kingdom) and Stockholm (Sweden) in that order. Faroe Island study (Wang et al., 2016) revealed a prevalence rate of 0.94% from all 7–16 year-olds living on the island who were followed up till the age of 15–24 (Kočovská et al., 2012). The report from South Dublin was based on the new European Protocol for Autism Prevalence (EPAP) for ASD prevalence measurement (Boilson, Staines, Ramirez, Posada, & Sweeney, 2016). The authors reported a prevalence rate of 1.15% in 6 to 11-year-olds in a sample size of 5,457. The Icelandic study is based on a nation-wide database of ASD children aged 11–15. A total of 267 children were diagnosed with ASD from 22,229 children recorded in the birth cohort with a prevalence rate of 1.2% (Saemundsen, Magnússon, Georgsdóttir, Egilsson, & Rafnsson, 2013). The investigation from Stockholm (Sweden)

reported a prevalence of 1.44% in 0–17 year-olds (Idring et al., 2015). A total of 7121 children were diagnosed with ASD in a cohort of 495,864 using DSM-IV until 2008, and ICD-10 since 2009. The Scottish study reported a prevalence rate of 1.6% (O'leary, Henderson, Jacobs, & Cooper, 2016) based on data from the annual Scottish Pupil Census which covers all publicly funded primary, secondary and special schools, some 684,415 students aged 4–18 (National Statistics, 2016). In the UK ICD-10 and DSM-V are used as the main diagnostic criteria for ASD (NSH, 2017).

Overall, countries nearer the equator that is, below 25° latitude, the approximate latitude of the Tropics of Capricorn and Cancer, have prevalence rates below 0.35% while the European and North American countries have reported rates above 0.35%. This contrast in prevalence rates which it is proposed is linked to exposure to solar irradiance merits further investigation.

8.5 Study Limitations and Future Work

A comprehensive review of the published literature was conducted to establish a global picture of ASD prevalence rates across different latitudes. However, prevalence studies give only a cross-sectional view of the number of the people suffering a disease at a specific point in time. Although attempts were made to use comparable criteria, there is always a challenge in comparing prevalence studies from different regions, with differences in case definitions, case identification (or case ascertainment), and case evaluation methods. The range of diagnostic criteria used, albeit internationally recognized, pose another challenge to ideal comparisons. Ideally, one criterion is needed to get an accurate picture of the change in ASD prevalence. Additionally, solar irradiation and hence sun exposure and more particularly Vitamin D levels, had to be inferred based on the reported location of the study but it is possible that those living near the equator could avoid the sun while those at higher latitudes might take advantage of all available daylight.

Small sample sizes in some studies is another weakness as is the diversity in the ages of the children assessed. Due to the remote locations of some studies from developing countries, there is a high probability of the sample having an overall similar genepool so that if a specific genepool has a

low or high ASD susceptibility, this would distort the results. This proposition then raises questions about the impact genes have on ASD prevalence, as for instance, the studies of Chinese samples reveal the highest rates of ASD which gives some credence to this factor for future studies. It is highly probable that children in colder climates remain indoors more where poorer air quality (Jung, Lin, & Hwang, 2013) and possibly more time spent watching television (Shane & Albert, 2008; Chonchaiya, Nuntnarumit, & Pruksananonda, 2011) may contribute to, or compound the effects of lack of vitamin D_3, in the onset of ASD.

Universal healthcare systems in some developed countries like that of Japan, Australia and United Kingdom posed their own constraints. Due to the availability of national databases of children with ASD, small regional prevalence studies are scarce in such countries. Lastly, there is a shortage of ASD studies from countries below 20 ° latitude. These two factors pose further barriers to any similar study.

An accurate assessment of ASD prevalence with respect to solar irradiance necessitates studies across a range of latitudes from diverse regions. It may be that the highlighted limitations could be overcome if a study were undertaken in Australian cities as it is a country that stretches from latitude 10 °S to over 45 °S. This latitude range provides year-round maximum solar irradiance in the north to the minimum irradiance in the south within one political and medical system. While Bent Dissanayake and Barbaro (2015) reported ASD prevalence rates in Australia, this was by state each of which stretch over several degrees of latitude. Even so, the Northern Territory (approximately 11° to 25°S) had the lowest reported ASD rate of 0.376% and Tasmania (approximately 41° to 45°S) had 0.752%. This change between two Australian states strengthens the argument that solar irradiance has an impact on ASD.

8.6 Overview

In this review, the aim was to explore the link between solar irradiance/sunlight and the prevalence rates of ASD. This study for the first time has shown, theoretically, a relationship

between low solar irradiance and ASD, particularly noted by the low rates in countries near the equator and Northern Territory (Australia) and high rates in Europe and Tasmania (Australia). The results of this review provide some support for Cannell's hypothesis (2008; 2017) that lack of Vitamin D, which in its natural form comes from exposure to UVB in sunlight, is implicated in ASD.

In conclusion, the findings of this review add support to the hypothesis that lack of exposure to full spectrum of solar irradiance increases the rate of ASD prevalence. These finding may contribute to understanding the aetiology and pathogenesis of ASD. Perhaps more importantly, it indicates that exposure to sunlight might be an inexpensive and effective intervention technique in the prevention of or to reduce the impact of, the symptoms of ASD on the child and family. Further studies are required to support this hypothesis, but in terms of the families living with a child with ASD, exposure to sunlight may assist in the management of symptoms.

8.7 Chapter Summary

The aim in this study is to expand on Cannell's (2008; 2017) hypothesis that linked ASD with low Vitamin D levels by investigating the link between solar irradiance/sunlight and the prevalence of ASD by latitude. As sunlight based UV is the main source by which the body produces natural Vitamin D, it was hypothesized that prevalence rates of ASD would increase as latitude increases. This relationship is due to the fact that solar irradiance and UV decreases with distance from the equator. A review of studies from across the globe provides tentative support for this hypothesis.

Chapter 9. Study 2[2]: A Qualitative Study

Raising a child with Autism Spectrum Disorder (ASD) is challenging and has an impact on parents and the rest of the family. The pervasive and severe deficits often present in children with ASD are associated with a plethora of difficulties and an increase in the mental and physical health problems of parents (Karst & Hecke, 2012). Compared with mothers of typically developing children, mothers of children with ASD report significantly higher fatigue (Giallo et al., 2013) and the severity of behavioural problems has been found to account for much of the variance in caregivers' poorer psychological and physiological health (Lovell et al., 2015). Consequently, the family has to cope with several stressors not experienced by the family of normally developing children.

Coping refers to the family's ability to manage change and stressors (Olson et al., 1979) and stress is considered to be ongoing for families of a child with ASD. The extant literature shows differences in the adaptation of the families of children with ASD with variability in coping strategies and social support linked to individual differences in outcomes (McStay et al., 2014; Paynter et al., 2013).

Despite a growing literature on psychosocial studies of children with ASD, the research on coping with and family adaptation to living with a child with ASD is still limited (Paynter et al., 2013). Understanding the experience of families living with a child with ASD and how it relates to family outcomes is important when developing support strategies for these families as well as benefiting the child with ASD (Pepperell et al., 2016).

Research has identified many economic, social and psychological difficulties affecting the family of a child with ASD, as discussed in previous chapters, but little is known about any compounding difficulties faced by families living in rural/regional areas. Exploration of such areas is

[2] Syed, S., Moore, K., & March, E. (2018). Coping strategies of parents of children with Autism in regional Australia. In P. Buchwald, K. Moore (Eds.), *Stress and anxiety: Theories and realities* (pp. 119-127). Logos Verlag. (See Appendix B)

important as facilities are often limited and time and travel costs associated with accessing services may be prohibitive. Identifying and understanding any additional stressors associated with families living in these areas will contribute to tailoring healthcare delivery. As such, the aim of the current study was to 1) explore the experiences of families in regional Victoria, Australia, who live with a child with ASD and 2) assess their perceptions of the impact of ASD on the family.

9.1 Method

9.1.1 Design

A qualitative study was used to collect and collate data from families living with a child with ASD in the Latrobe Valley, Victoria, Australia.

9.1.2 Participants

The 16 participants in this qualitative research were parents/guardians of children diagnosed with ASD. The sample included 12 mothers, three fathers and one paternal grandmother. Five mothers and two fathers indicated that they were separated or divorced. Aside from the two fathers who were divorced, each participant reported that the child with ASD lived with them. The grandmother was caring for her grandson as her daughter-in-law had divorced her son and moved to the city. All parents were aged between 30-45 years, and the grandmother was in her 60s.

9.1.3 Procedure

Approval for the project was provided by the Human Research Ethics Committee of Federation University (Project #A16-152, Appendix C). Participants were recruited from local ASD support groups in the Latrobe Valley of Victoria, Australia following agreement by each local organiser. The secretary of each group sent an email to members inviting interested persons to contact the researchers to participate in one-on-one interview at a time to be arranged (Appendix D). All interviews were held at a time and at a location preferred by each participant (which included home environments, the University and the support groups' offices). Interviews were around 45

minutes each, and were audio-recorded with participants' consent. Notes were also taken with participants' consent and the overall content of the interview confirmed with each participant at the conclusion of the session. Two researchers were present at each interview, and these were later transcribed by the current author and the audio files deleted. These transcripts were used for the subsequent analysis.

9.1.4 Materials

The semi-structured interview questions were designed to elicit responses relating to the participant's family situation and the impact of living with a child with ASD on themselves and their family (Appendix E). Open ended questions were related to the impact on their relationships, social activities, health status, school-time, support level and their concerns for their child going forward as well as any difficulties experienced by living in a regional area. Participants were free to raise any further issues that they considered relevant.

9.1.5 Analysis

A thematic qualitative research design (Braun & Clarke, 2006) using an inductive approach was employed to identify the common themes related to the psychosocial impact on the family of a child with ASD. The following realist theoretical framework was used in order to reflect the meaning of participants' reality. Themes were identified at a semantic level based on the explicit meaning of the data. There was no attempt to go beyond what had been said during the interviews. The six stepped process guiding this analysis was: (1) the transcribed accounts were reread and studied a number of times to familiarise with the data, (2) potential themes and common codes related to psychosocial impacts were identified, (3) codes that appeared in three or more of the accounts were categorised as a 'theme' (category), (4) themes were reviewed, (5) themes were defined and categorised in super-ordinate and sub-ordinate themes, and (6) themes were used to prepare the report (Braun & Clarke, 2006).

9.2 Results

The data generated by the semi-structured interviews revealed many commonalities with seven themes on psychosocial impact extracted from the data: Regional living; financial impact; participant's personal and family lives; child's health and behaviour; child's school and school-time; concerns about child's future, and limited support (Table 9.1). The themes extracted from the data show both variety and commonalities in the coping strategies adopted by the parents of children with ASD. There was a noticeable gender bias in the range of coping strategies with female being more emotional when discussing how they manage life and their child with ASD than males. The thematic analysis also yielded five coping themes each with sub-themes (see Table 9.2) which are discussed below.

Table 9.1: Psychosocial Impact Themes and Examples

Theme	**Examples**
Regional living	Additional time and monetary demands due to increased travel distances for appointments Lack of specialised support at the local schools Lack of specialised healthcare and other facilities in region Need to relocate to a larger town with better facilities
Financial impact	Four of six working mothers needed to quit their jobs to manage child and associated travel to appointments Lack of money for leisure and social activities
Participants' relationships and family lives	Child's ASD was a factor in six of eight divorces Little leisure, relaxing time

	Little time or opportunity for entertainment/outings Little or no time for partner Little time and money for siblings, extracurricular activities and outings Siblings feel neglected as child with ASD is the focus
Impact of child's health and behaviour on the family	Child's behaviours effect the entire family Parents' stress level associated with the severity of aggressive/disruptive behaviour Losing friends and a social life due to child's ASD Socially ostracised due to child's ASD Public incidents arouse feelings of embarrassment/humiliation Disruption of siblings lives for families who relocated Children picky eaters and implementing healthy diet is important but difficult Some parents used innovative tactics to trick their child into eating healthy food Mealtimes frequently stressful and usually not a positive family experience
Impact of child's school and school-time on the family	Full-time school attendance acknowledged as important Provides time for parents to carry out household chores and part-time jobs Important for child's social and intellectual development Friends and good social life at school helps

	Child being bullied at school is a problem Child disruptive or misunderstood Parents often called to school, even for minor incidents or misunderstood behaviours
Concerns about child's future	Child's incapacity to live independently in future Worry child might fall into bad company Concern for child when parents old/deceased No participant wanted any sibling to bear the burden of care
Limited support	Participants received some support from partner (n=10), friend or family support (n=5) or no support (n=1) Physical help allows free-time for part-time jobs Supportive partner critical to relieve caregiver's burden and help with home therapies Four participants (divorced (n=2) and married (n=2)) reported child's sibling giving a helping hand at home or at school (same mainstream school) These parents reported feeling overwhelmed and dissatisfied with life in general Family support available to some in small community or where grandparents moved in Overall a lack of "system support"

	Access to support groups identified as critical in coping with stress

Table 9.2: Coping Themes and Examples

Theme	**Examples**
Time management	Mothers gave up their jobs to be available for their child's needs Travelling to and time spent at appointments interfered with other commitments Parents sacrificed social lives Parents had to ask friends/family members to help with the child with ASD to get some free time Often easier to stay at the school then be called in
Social withdrawal	Parents avoided social events due to child's antisocial behaviours Uncomfortable inviting friends to house Parents felt ostracised
Emotional coping	Mothers heavily relied on the support from the ASD support groups, for some, family, friends and partners were also important Fathers relied less on support from others

Medication, meditation and physical activity	Exercise, walking the dog, yoga, tennis and swimming were being used as a coping strategy Psychological services Medication was being used by some to cope
Problem solving strategy	Parents reported engaging in problem-focused coping strategies for: • Child's behavioural and sensory management-medication, reward system, distractions, exercise • Lack of resources at schools-step in to assist when no aide • Child's disturbed sleep-medication, music, exercise to tire • Child's picky eating-alternate presentations of food • Outdoor and social activities-avoiding potential stimulants to child's tantrums, removal of media

9.2.1 Psychosocial Impact

9.2.1.1 Regional Living

A shortage of specialised medical support structures in the region forced parents to travel long distances for appointments which was costly in terms of parents' time and the price of fuel. This increased travel time also increased parental stress which affected their social lives, their work, and accentuated their monetary burden. One participant reported that she and her family had to move from the family farm because of unavailability of support at the local school. While this move to a larger town was beneficial for the child with ASD, it had a negative impact on the siblings who were settled in their school and had friends nearby. Another participant reported that she had moved to a

larger town due to the unavailability of medical practitioners in her local area. This move also had a negative impact on the siblings.

> We were on a farm and we had to move when our girl with autism started school because our girl had very minimal early intervention. So here we are and moving had a big impact on our children, on the other kid. (Participant 5)

9.2.1.2 Financial Impact

All three fathers were working full-time before and after their child's diagnosis and the grandmother's son was also working full-time. Of the mothers who were working before the diagnosis, two working part time reported no change in their work status but four mothers reported that they had given up work due to the hours needed to attend frequent ASD related therapies and medical appointments for their child. This reduction in family income imposed a financial burden on the entire family.

> I used to work full time. Then I gave up work after child's birth. [Then I] go and work casual at a bottle shop to get income as well. But then I decided I can't work. (Participant 7)
>
> Gave up work because of appointments and therapies. (Participant 9)

A lack of finances, especially in one income families, and the additional costs of therapies as well as less time both perceived and actual, by parents for the other children had a negative emotional impact on the siblings.

9.2.1.3 Participant's Relationships and Family Lives

A recurring theme was disturbances in the personal, family and social lives associated with raising a child with ASD. Participants reported long lasting and continuing psychosocial consequences on "me-time", and notably, their personal relationships and social activities. Six of the eight separated/divorced participants stated that ASD was a factor in the breakdown of their marriages.

[Child with] Autism had an impact on our relationship. Puts extra burden, stress, no free-time for ourselves; a strain and burden. (Participant 6)

Separated because of personal and child's autism reason. [It] had a huge impact on us. (Participant 10)

Other participants also reported difficulties associated with maintaining a good relationship with their partner.

It [autism] does impact the whole family. Each and every member. Relationships. It is hard… [We] don't get much time together. (Participant 8)

Parents also spoke of the impact on siblings.

My husband is on work and when other kids are going for football, netball, I will take them there but I will stay in the car with this [autistic] girl and I would miss out their game and they missed me probably. It's hard to spread yourself out and you can't just leave them with anybody at all. (Participant 5)

Parents noted that siblings sometimes showed resentment towards the child with ASD as they saw that the child being given a "lion's share" of the parents' attention.

He [sibling of the child with ASD] actually feels pretty bad though. He thinks that the whole family actually revolves around what [the child with ASD] does and wants. I speak with him. I let him know he is different. (Participant 10)

Younger one [sibling] missed out on a lot of outings. Once he actually asked me, am I adopted mum. Out of nowhere. You don't try to treat them different. (Participant 4)

9.2.1.4 Impact of Child's Health and Behaviour on the Family

The health and behaviours of the child with ASD were directly related to the stress and burden on the family. Parents' attempts to provide a healthy lifestyle for their child with ASD was a

challenge and contributed to the overall stress. They found it difficult to deal with the child's picky eating habits and often rebellious behaviour at mealtimes.

> He would eat anything by how it felt in the mouth; sensory. At one stage he was living on sausages, party pies, potato crisps and milk. (Participant 3)

> Eats a lot of junk. Potato and strawberry only fruit and vegetable he eats. (Participant 14)

Parents came up with innovative approaches to include healthy foods in the child's diet. One example was to re-use the packaging of the child's favourite snacks and repack it with fruits or to mix vegetables with chocolate.

> He had a particular banana dessert he liked. Then they changed the carton and he wouldn't touch it. I actually wrote to the company and explained my dilemma and they sent me a big box of the old cartons. In the kitchen when he wasn't looking I would get the new dessert pack or fruit or vegetable, fill it in and he would eat it all up. (Participant 4)

> Fruits and vegies were a no, no, when she was younger. But then at school there was dipping chocolate with fruits and she started liking it and gradually less chocolate and more fruit and now good to go. (Participant 5)

A major source of the parenting challenge stemmed from the child's aggressive/disruptive behaviours. Some parents reported increased stress and having to give up their social lives to the extent that they confined themselves and their child with ASD to the homes. Participants recalled anti-social incidents in public which left them with a sense of humiliation or embarrassment.

> We don't go out together as a family, together maybe once a year. If we all go out together, one of us has to go back home because he is not a social kid. He used to run off and throw tantrums… [We had planned for a week and] we went out once long time back for our anniversary. So we all went out, gave him some chips. And that's it, he wants to go home.

Hardly there for ten minutes. So I drove back home with him. It was a big deal for his dad. But we changed, basically our lives for him. (Participant 7)

Some of the participants felt socially ostracised and described an inability to attend social events due to the child's anti-social behaviours.

Lost a lot of good friends [due to child's autism]. (Participant 11)

Definitely [autism] impacts your social life, relationships, and friendships. Lost a lot of friends. They don't understand… Nobody invited to anything. And you just didn't get invited to anything. It was awful. (Participant 10)

Parents identified wide-spread ignorance of autism and society's incapacity to differentiate between the children with moderate and severe Autistic traits as major contributing factors towards social isolation.

9.2.1.5 Impact of Child's School and School Time on the Family

Of the children with ASD who attended school full time, 12 children were at a mainstream school and four attended special schools. Three children, who attended school part time were enrolled in mainstream schools and each of these had a full-time Integration Aide.

School time was very important for two main reasons. Parents considered the school time necessary for the child's development, intellectual and social, but it also provided them with free-time to carry out other essential tasks, such as a part time job, shopping, house-work and some opportunity for socialising. Parents of the three children attending school part-time commented that they were called to the school on numerous occasions due to the issues related to their child. Two of these children were non-verbal and calls to the school often involved the school staff's inability to understand the child.

Parents considered school friendships as an important part of the child's development. Nine parents reported that their child had a friend at school and only three of the 12 children attending school full time were not taking part in any extracurricular activities.

> If they do not have friends, they will not progress. (Participant 5)

Bullying of the child at schools was reported by six parents.

> She was bullied by other children. Her reaction was she became quiet, disturbed, introvert, which is opposite of her personality… She would hide under bed and scream: Don't let them hurt me mummy. (Participant 1)

> Everyone picks on him. Call him names. I know parents have been bitching [SIC] about him. He can be really naughty and rough and physical. He is big and strong and little kids go away. He is not in any kind of team sports, hell no. (Participant 13)

Bullying at school had a negative impact on the child with ASD which parents believed resulted in behavioural outcomes at home. Support at school was considered important to help with the child's development but also to reduce bullying. Interestingly, children with a full-time Integration Aide did not encounter any bullying incidents.

9.2.1.6 Concerns about Child's Future

All participants expressed great concern about their child's future; how could their child live independently in future without them. Nine participants were hopeful that their children would improve with time, learn to cope with the world around them and be able to live independently.

> As she matures, she will learn how to behave and will definitely improve to live independently. (Participant 2)

However, there was also a worry that they might fall into bad habits due to immaturity.

> Worry about him getting involved in drink, drugs or sex. Can live independently, most likely as an adult. (Participant 10)

None of the participants wanted the siblings of the child with ASD to act as the caregiver.

> Hate that she would be a burden, worry about her future a lot, cannot be independent. I don't want [siblings] to be bothered. (Participant 5)

> She [older sister] lives locally and she is not interested in him [child with autism] at all. I know how she feels so I don't ask. (Participant 8)

9.2.1.7 Limited Support

Participants clearly felt they were overworked and did not get enough support from family, friends or "the system". Among those in a relationship, 10 participants reported receiving some form of partner support, five received some support from both their friends and the family, but one participant reported not receiving any form of partner, friend or family support.

Partner's help was most appreciated with routine household activities as well as assistance with the child's developmental activities. This help allowed the primary carer some "time-out".

> [My child] goes for a walk with his dad every day... My husband helped me develop his speech. (Participant 7)

The five single parents had to rely either on family or a friend and reported feelings of depression and lower life satisfaction compared to the participants living with partners. One participant was on anti-depressants and regularly visited a psychologist. Another participant was relying on financial help from a friend to cope with the high demands of the child with ASD. A third participant was relying on grandparents to help raise the child.

Four participants mentioned that their child's siblings provided some level of support. Two noted that the siblings helped the child with ASD at school, another praised the sibling for helping

the child with ASD to learn and develop his skills. One participant mentioned that the sibling often acted as the voice of the child with ASD as the child was non-verbal.

> She would speak for [the child with ASD] when she was non-verbal. Siblings love each other. Share same bond as in any other normal families' kids. She is very protective of her sister at the school. (Participant 1)

> Elder brother helped me raise the boy as [father] works full-time and shift-work at night. Very close bond, loves him and cares for him. He has helped him learn a lot, skill development and plays with him. (Participant 3)

The positive impact of family support was expressed frequently by the seven participants receiving either physical or financial help or both.

> [We] Live in a small town, all are friends, very close and helpful. My family and [husband]'s family are very helpful and supportive. (Participant 15)

> Mum since my father passed away has been living with us, sold her home and came. She is 87. She is with me for last 10 years. Since then she has been full on. Looking after home, kids. Mum is very independent. Mind is always switched on. Body may be fragile. Mum wants kids to be more independent, autism or not. She says [child] should be able to this and this and this. Mum is working on [child]'s social skills. (Participant 1)

Physical help was also important to enable some participants to do part-time work.

> Sometimes my mother or sister would pick him up from the school and look after him till I'm back from work. I usually work part-time so I'm free at the time he goes school and comes back. (Participant 13)

The majority of the participants (n =12) were supported to some extent physically or financially by friends. Some participants were of the opinion that families were not as helpful as

friends. All participants had access to and were recruited through support groups, although five were not attending meetings due to constraints.

> Don't have much time [for the support group]. (Participant 2)

The participants who attended regular support group meetings identified this support as critical in helping them cope with the stress.

> Support group is such a relief. Makes me feel strong because it is great for sharing especially when feeling anxious. We can't expect ordinary people to understand what our lives are. Definitely made good friends here. (Participant 8)

9.2.2 Coping Strategies

Some of the results of this study are in concert with the existing literature, however, this study has also revealed new coping strategies such as parents staying at school with their child as a problem solving strategy in the absence of a teacher's aide in the school. These and the other coping strategies identified in this study are discussed below.

9.2.2.1 Time Management

Caring for a child with autism is not only physically and psychologically demanding but also time consuming. The demand of extra hours created by the care of their child and frequent therapy and medical appointments was one of the biggest stressors for the parents. This led to a number of coping strategies which were also gender biased. All three fathers were working full-time before and after the diagnosis and the grandmother's son was also working full-time. Mothers on the other hand often had to cope by giving up their work. Those mothers who were working before diagnosis, two working part time reported no change in work status however, four mothers reported that they had given up work following their child's diagnosis.

> "[I] stopped working after diagnosis." (Participant 3)

"[Left job.] Couldn't find time as children living with me. Put pressure after diagnosis." (Participant 6)

Fathers on the other hand coped with the time demand by not availing themselves of ASD support group help. All three fathers and the grandmother's son were not attending ASD support groups, which could be considered avoidant coping, or their use of that time for other activities could be considered "me time".

"No I don't have time for the support group. Happy to have some time for myself." (Participant 2)

Another strategy for coping with the stress caused by the limitation of time was by cutting down on social activities. All participants had done this to varying degrees.

"We don't get much time alone or to go for a dinner or to meet friends or family." (Participant 8)

Participants clearly felt they were overworked and relied heavily on partner, friends and family support to cope with the stress and get some free-time. Ten participants reported receiving some form of partner support, five received some support from both friends and family, and only one participant reported not getting any form of partner, friend or family support.

"My mum often visits our family and helps us." (Participant 10)

"My mum understood the struggle and comes to stay on kids' holiday. That is my holiday." (Participant 7)

9.2.2.2 Social Withdrawal

Factors leading to the coping strategy of social withdrawal were both circumstantial and cultural. For the child's meltdowns and tantrums parents found social withdrawal as the best coping

style. Parents were either avoiding social activities for the fear of the child's anti-social behaviours in public or leaving the social event at the onset of a meltdown.

> "Worry about when a meltdown can come, whenever we go out. Hard to control outdoors as I am not very strong. We just leave the place and go back home." (Participant 6)

The coping strategy of social withdrawal was also a reflection of parents' reaction to society's insensitive attitude towards "autism". Their opinion was that others don't understand ASD and can't differentiate between autistic behaviour and bad behaviour.

> "People don't understand, not even family and [they] ignore and pretend [he] doesn't exist. I don't take him around anymore." (Participant 10)

Some of the participants felt socially ostracised due the child's ASD and it was another factor leading to social withdrawal.

> "At school I feel parents ignore me. I feel stigmatised." (Participant 8)

9.2.2.3 Emotional Coping

Overwhelmingly, mothers described their reliance on emotional support from others including friends and family, and ASD support groups. As stated earlier, none of the fathers were availing themselves of ASD support group help. On the other hand, except for one, all mothers and the grandmother were accessing support from their ASD support groups. All of them identified it as an important part of their emotional coping strategy.

> "I feel very strong especially because of the support group. Great for sharing. We can't expect average people to understand what our lives are." (Participant 8)

The stark difference between fathers and mothers related to the emotional coping strategies was also noticeable in friend support. Only one father mentioned having a close friend to discuss and

share matters related to his child. Eleven out of twelve mothers on the other hand had friends to help with emotional coping. Although, nearly all of these friends were made through support groups.

> "Have good understanding friends. Some friends don't understand but some do. I plan to meet friends at lunch time or morning… Call them anytime and they would listen." (Participant 6)

9.2.2.4 Medication, Mediation and Physical Activity

Four participants noted physical activity as a coping strategy to manage stress. These activities ranged from walking the dog and yoga to swimming and tennis. For the participants it had become a part of their routines. This strategy also had a gender bias as all three fathers were using this strategy while only one mother had turned to these strategies.

> "When I'm off, I would clean the house, walk the dog and exercise." (Participant 2)

One participant visited a psychologist weekly and her GP has prescribed antidepressants for her. She was the only mother with no close friends to share her feelings and to discuss her child's issues.

9.2.2.5 Problem Solving Strategy

By far, the most utilised coping strategy was problem solving which all the parents were implementing, including the grandmother. Techniques for coping with the antisocial and aggressive behaviours of the child were a major part of their strategies. Different parents adopted different strategies. Four parents were giving their children medications for their behaviours while a fifth child had been prescribed one and was scheduled to start taking it in the week after the interview.

> "She was put on medication because of behaviour. Hard to teach her anything. [Medicine] masks the symptoms. Doesn't cure the problem." (Participant 2)

Three parents were countering the behaviours by a reward system for controlling aggression and meltdowns. This proactive strategy was in contrast to, avoiding social situations and leaving social gatherings at the onset of the meltdown, a strategy that was being practised by all the parents. Another strategy that was implemented by nearly all the parents was the use of iPads and tablets. Except for one mother all parents were relying heavily on tablets and other electronic devices to keep their children busy and to avoid meltdowns. This one mother had removed the TV, tablet and internet access from their home in attempts to force her child to play outside. Her strategy had worked and her child was spending a considerable part of the day playing outside. Lastly, swimming was being used as a sensory and behavioural control strategy by the parents. Parents had noticed calm behaviour in their child after swimming. Consequently, they made swimming a part of the child's routine in addition to other activities like bike riding and trampoline. Eleven parents were using swimming as a strategy.

Problem solving strategies also helped four parents whose children lacked adequate support at their schools. Although a more radical solution to the problem, the lack of good support structure in the rural areas had forced two parents to move with their family to a city in search of a better school and facilities for their child with ASD.

> "No special school in our town; public school wasn't good enough. It wasn't working… so we moved to the city." (Participant 7)

Another two parents would routinely go to school and spend time in the school as a replacement for their child's aide, during days when no aide was available.

> "I go to school, excursions and camp with my kid when there's no aide." (Participant 8)

Problem oriented coping was also visible in dealing with picky eating habits of the children. Parents came up with innovative approaches like using child's favourite snack's packaging to repack with fruits or they mixed chocolate with vegetables.

Disturbed sleep was another problem forcing parents to find solutions. Two parents were giving their children sleep medications while the rest had come up with innovative solutions like playing calming music, giving the child time on trampoline to tire him out and so forth.

> "I put him in the pram and walk around the home. As soon as the pram stops he would scream. I would walk around the home until he goes to sleep." (Participant 12)

> "I put on music to put him to sleep as it calms him down." (Participant 8)

Parents were mindful of the development of social skills of their children and employed different strategies. One parent realising the social ostracism associated with her child's behaviour in public had resorted to organising parties and inviting guests to her home. Two parents were using a reward system. They would take their child out in public and for good behaviour the child would be rewarded with something the child liked. Two parents were using child's favourite sport to make the child engage socially. For example, one child liked surfing and opportunities to go surfing were being used as rewards to teach him socially acceptable behaviours.

Finally, some parents were also actively looking for treatments for their children. Six participants clearly said that they wished to take autism away from their child. Of these, three parents reported actively searching for new therapies and interventions that would reduce autistic traits.

> "I have been doing my own research since the diagnosis. I want to find therapies and interventions that will reduce the severity of [autistic] symptoms." (Participant 16)

9.2.2.6 Barriers to Coping Strategies

In addition to the success in employing problem and emotion focused strategies, a number of parents discussed the barriers they experienced. Monetary issues were the biggest barrier in accessing coping strategies. Time was the second major factor. All three fathers identified time being a major constraint in accessing ASD support groups: they had to choose between physical activity

and “me-time” and support group and all three chose the former. A major cause of this lack of the availability of the free-time was due to regional living. Parents had to drive considerable distances to access the healthcare services and this placed an extra time and financial burden on them. Drastic measures like moving with the entire family to the nearest larger town was one of the solutions for some families but it was contingent upon the availability of jobs and the acceptance of the entire family to help with the move. Finally, social ignorance of ASD was another factor which influenced the coping strategies of the parents particularly when scorned by others.

9.3 Discussion

The aim of this study was to identify the ways in which living with a child with ASD has an impact on the psychosocial wellbeing of families living in regional Victoria, Australia and to identify their coping strategies. Semi-structured interviews were conducted with 16 participants and despite the relatively small sample size, the content of interviews reached saturation. In addition, it should be noted that the sample was homogenous, largely Caucasian, despite Australia being a multicultural country. This sample bias is attributed to the demographics of the region where the majority of the residents (78.37%) are Australian born and for most (75.93%) their ancestry is British (Australian Bureau of Statistics, 2016).

The findings from the current study corroborate previous research where parents of children with ASD have reported wide ranging physical (Ausderau & Juarez 2013; Ghayan, A. & Ghayan, S., 2014; Giallo et al., 2013; Hutton & Caron, 2005; Karst & van Hecke, 2012), social (Chan & Lam, 2017; Farrugia, 2009; Gray, 1994; Rao & Beidel, 2009), psychological (Karst & van Hecke, 2012; Lovell et al., 2015; Rao & Beidel, 2009) and financial (Dyke et al., 2009; Karst & van Hecke, 2012; Lovell et al., 2015) effects of living with a child with ASD. All participants in the current study recounted wide ranging effects, some with a long lasting impact on themselves, their partner and the child’s siblings. Such wide ranging effects indicate that living with a child with ASD affects the entire family.

The unique themes elicited in the current study are the impact of regional living and the impact of school going. Regional living contributed to an increase in the burden on the families due to a lack of nearby facilities and inadequate access to healthcare providers. Additional time required for traveling to therapy sessions and medical appointments led many mothers to quit their jobs, extenuating the monetary burden on families and reducing their social activities and ability to engage in entertainments.

Fletcher et al. (2012), Matthews et al. (2011) and Sitimin et al. (2017) also reported that the additional time to care for a child with ASD restricted parents' ability to engage in paid employment. The restrictions these financial restraints imposed on the family's social activities, as well as educational opportunities, were also reported by Fletcher et al. (2012) and Dyke et al. (2009).

One obvious solution to the inadequate availability of health care providers is to encourage suitably qualified specialist practitioners and therapists to relocate to these regional areas but, despite government initiatives, this need remains unfulfilled. A possible alternative is to have practitioners travel into regional areas on a regular, consultancy basis. While this occurs in some areas, the more remote a district, the less likely it is to occur. This approach might be more successful if Government increased financial incentives (fee/rebates) to the practitioners traveling to these areas, or, as is the case for practitioners coming from overseas, practitioners were required to spend some time in rural and regional areas post registration.

Six of the eight participants who were divorced (40% of the sample) stated that their child's ASD was a major factor in their relationship breakup. Several other researchers have reported a high incidence of divorce, lower marital satisfaction and even depression (Hartley et al., 2010, Ramisch et al., 2013), where living with their child's ASD was a major factor.

Parents reported little time, and money, for family activities and social outings, which supports past research from Hutton and Caren (2005). In fact, past research has suggested that mothers of children with ASD can spend up to 1000 hours per year more in the care of their child

than mothers of normally developing children (Järbrink, 2007). Naturally, this leaves less time for the partner relationship and for siblings, and for both partners to hold down outside work, as previously described by Hock et al. (2012); Saini et al. (2015) and Sim et al. (2016).

Further, results of the current study corroborate previous studies that have found behaviours of children with ASD are strongly associated with parental stress (Lecavalier et al., 2006). The level of this stress has been associated with the severity of disruptive behaviours (Karst & van Hecke, 2012; Lovell et al., 2015).

Participants reported that they had lost friendships as a result of their child's disorder and were frequently socially ostracised due to the behavioural problems demonstrated by their child in public places. Myers et al. (2009) also described parents' loss of friendships and their decisions to avoid public places due to fear of being stigmatised if or when their child screamed or engaged in temper tantrums.

Parents benefited greatly from their child spending part if not a full day at school, with some level of productive learning outcomes and supportive peers. With the child in school, this free time helped parents to carry out home chores and to work. Bullying at school, however had a negative impact on the child with ASD. As children with a full-time Integration Aide did not encounter bullying incidents, this strengthens the argument in favour of full-time Integration Aides for these children. It is also important that all children are encouraged to demonstrate tolerance for those who are different in some ways, whether because of ASD or any other physical or emotional difference. Past research has reported on the child with ASD's behavioural problems at school, such as barricading and, occasionally destroying school property (Brede et al., 2017; Myers et al., 2009) and the child with ASD being bullied at school (Kelly et al., 2008).

All parents were concerned about the ability of their child with ASD to live independently in the future. They did not want this burden to fall on the siblings. Past research has shown that siblings of children with ASD, often bear a greater burden than others, less social participation (Meny

et al., 2018) and hobbies (Rao & Beidel, 2009) due, not just to financial constraints, but also to the time and attention directed by parents to the child with ASD. Kirby, Bagatell, and Baranek, (2019) also noted that parents of a child with ASD have concerns about the future of their child

Support is an important element for families with a child with ASD, but many participants reported this was limited. This support could be physical (e.g., helping with chores, travel, babysitting) or friendship (e.g., sharing stories, burden). Partners were considered an invaluable source of support for those still in a relationship. Unfortunately, the social stigma (Chan & Lam, 2017; Farrugia, 2009) and negative experiences of discrimination reported in past research also, in part, prevented the current participants maintaining strong support networks. However, the availability of ASD support groups was considered by many participants to be critical in helping them cope.

Aside from the difficulties associated with living with a child with ASD, participants were also forthcoming about strategies they used to cope more generally. While several mothers relinquished their jobs, this also made time available to deal with their child's appointments, travel, and contributions to home therapies. Some mothers were able to ask a friend to help, allowing them brief respites for themselves, and this approach is in accord with the findings of Pisula and Kossakowska (2010). Mothers used their local support group for emotional support and coping, a strategy outlined by Benson et al. (2010) and Pisula and Kossakowska (2010).

Another finding of the study was the society's lack of understanding of ASD in general and of severe autism specifically. Thus, it is important to increase awareness of ASD and the differences between moderate and severe autism. Electronic and social media campaigns along with training of personnel in service provider organisations may also be used in this respect. This awareness would encourage social inclusion of the families of children with ASD.

Results of the current study suggest that interventions addressing a variety of parent and child needs are necessary in order to positively affect psychosocial wellbeing of the family. These include

social and healthcare support, good schools with qualified integration aides, as well as behavioural interventions, aimed at reducing the stressors and reducing the adverse impact of the stressors on the family. The therapeutic salience of social support and its beneficial effects on the parents receiving it was quite clear in this study. Health professionals should also be aware that families of children with ASD in regional areas may be at increased risk for physical and mental health difficulties. Greater understanding of these burdens may facilitate appropriate solutions by the relevant professionals, such as healthcare providers tailoring their care to match the demands of families of children with ASD. The data from this study and the extant literature will be used to develop a scale to assess the impact of living with a child with ASD on families. The cross-sectional survey will be designed with input from the results of the Study 2. Based on the results following themes were identified as being of key importance for inclusion in the next study, namely: supports, social life, relationships, finances, feelings and strategies.

9.4 Chapter Summary

The aim in Study 2 was to investigate the impact on families living with a child with ASD. The results indicate that families were affected socially and financially. Seven main themes were extracted from the data: regional living, financial impact, relationships, impact of child's health and behaviour on the family, impact of child's school on the family, child's future and limited support. Families experienced varied levels of social support, relationships were effected and child's negative behaviour and reduced school hours contributed to the family's impact on living with a child with ASD. It is suggested that health practitioners be aware of the needs of families living with a child with ASD especially in rural and regional areas. Although past research has investigated the impact of living with a child with ASD, few studies have used specific scales designed for this purpose. In order to have a more comprehensive appreciation of the impact on families it is important to develop such specific measures. The data from the current study will be used for this purpose.

Chapter 10. Study 3A: Scale Development

Previous research on the impact of living with a child with ASD has typically used scales that are generic in their composition to assess the impact of the child's ASD on families (see Chapter 6.6). In fact, scant research to date has focused on the development of domain-specific scales to assess the impact on families who are living with a child with ASD. Several researchers have developed domain-specific scales including Maltby, Day, Hall, and Chivers (2017); Wallston, K, Wallston, B., and DeVellis (1978), and Weber, Blais, and Betz (2002) although Bandura (1986) was possibly the strongest advocated for the need for such specigicity in measurement.

In view of this lack of specificity of assessment measures in this field, it is imperative to develop scales that specifically address the impact on families living with a child with ASD and which are drawn inductively from parents' own views.

10.1 Aims

The aims of Study 3 were to develop and evaluate the psychometric properties of a series of new scales designed to assess: 1) the factors influencing the impact of living with a child with ASD on families and 2) the coping strategies used by these families, based on the constructs identified by participants in Study 2 (see Chapter 9.2). This research will provide standardised, domain-specific scales to measure parents' perceptions of the impact on the family, their coping strategies and support, to enable better understanding of the impact of a child with ASD on the family.

10.2 Method

10.2.1 Design

A cross-sectional design was used to explore the psychometric properties of scales designed in this study to specifically to assess the impact on families living with a child with ASD.

10.2.2 Procedure

This study was approved by the Human Research Ethics Committee Federation University Australia (Project-A18-072) (Appendix F) and conducted according to the Ethical guidelines. ASD support groups in the local region were approached and asked to invite their membership to participate in this study. A Plain Language Statement (PLS) (Appendix G) and a link to the online survey were provided along with an invitation to group members (Appendix H). The support groups posted these details on their Facebook pages but it was not possible to determine a response rate by group.

The opening page of the questionnaire, which resided on "SurveyMonkey", provided a further copy of the Plain Language Statement with details on the study. The PLS informed readers that participation was voluntary, their data anonymous, and that their submission of the completed questionnaire would constitute their informed consent. Participants were also advised that they could exit the study at any time by simply closing their web browser. In addition to completing the scales designed for this study the participants were required to provide their age, gender, relationship to the child with ASD who lived with them, and the age of the child. No incentives were offered for participation.

10.2.2.1 Insertion Criterion

Participants were required to be a parent or carer living with a child with ASD.

10.2.2.2 Participants

One hundred and seventy-eight volunteers living with a child with ASD agreed to participate in this study. Of these participants, 167 were the mother of the child, six the father, three a grandmother, and two were in guardianship roles. Of the children with ASD, 146 were male (80%) and overall the mean age of the children was 9.19 years (SD=4.88).

10.2.2.3 Scale development

A series of questions were written for this study based on the themes derived in Study 2 that were identified from interviews with families living with a child with ASD. These questions addressed the participants' levels of perceived support; the impact on their relationship, social life and finances; their feelings and the specific strategies they used to deal with issues around living with a child with ASD (see Chapter 9, Tables 9.1 and 9.2). Questions were answered on a 5 point Likert scale from 1 = *A little* to 5= *Extremely*. All questions were presented in a random order to reduce a possible order effect, fatigue, and any primacy effects. Participants were asked to answer the questions focusing on their child with ASD and any possible impact their child's disability might have on the family.[3]

10.3 Results

Data were analysed using SPSS (Version 25). Principal Components Analyses (PCA) with oblique rotations were used to determine the underlying factor structure of each questionnaires written for this study. Cronbach's Alpha was used to assess the internal reliability of the scales.

10.3.1 Scales

10.3.1.1 Social Support Scale

The Kaiser-Meyer-Olkin Measure of Sampling Adequacy (.839) and Bartlett's Test of Sphericity (χ^2_{136} 791.30, $p < .001$) both indicated the factorability of the correlation matrix for these 13 items. PCA revealed one factor with an eigenvalue greater than one and Cattel's Scree Plot, Tabachnick and Fidell's (2013) criterion of choice, also suggested the presence one factor which explained 38.7% of the variance. Reliability analysis indicated strong internal reliability, Cronbach's $\alpha = .79$. Factor loadings and descriptive statistics are presented in Table 10.1.

[3] The actual scale items are included in their relevant table (see Tables 10.1 to 10.5).

Table 10.1: Factor Loadings and Descriptive Statistics for the Social Support Scale

Item	**Loading**
I get emotional support from others	.758
My family provides emotional support	.757
I am satisfied with the help I get from others	.740
I am satisfied with all the help I get from everyone else	.678
My friends were supportive after my child was diagnosed	.656
I get emotional support from my friends	.654
My family was supportive after the diagnosis of my child	.627
My family visits in school holidays	.624
My family looks after my child when required	.595
I find it easy to find someone I can talk to when I am stressed	.529
I am satisfied with the help my child gets at school	.454
My friendships are negatively affected (R)	.451
My friends are able to assist when needed	.440
Eigenvalue	5.03
% Variance Explained	38.70%
M	27.66
SD	6.72
A	.79

Note. R = reverse coded item

10.3.1.2 Impact on Relationship Scale

The Kaiser-Meyer-Olkin Measure of Sampling Adequacy (.767) and Bartlett's Test of Sphericity (χ^2_{15} 306.36, $p < .001$) both indicated the factorability of the correlation matrix for these five items. PCA revealed one factor with an eigenvalue greater than one as did Cattel's Scree Plot, Tabachnick and Fidell's (2013) criterion of choice. This unifactorial solution explained 50.98% of the variance. Internal reliability of this scale was strong, Cronbach's $\alpha = .82$. The factor loadings, eigenvalue, percent of variance explained and descriptive statistics are shown in Table 10.2.

10.3.1.3 Social Impact Scale

The Kaiser-Meyer-Olkin Measure of Sampling Adequacy (.763) and Bartlett's Test of Sphericity (χ^2_{136} 553.45, $p < .001$) both indicated the factorability of the correlation matrix for the seven items written to assess the social impact. PCA revealed one factor with an eigenvalue greater than one and Cattel's Scree Plot and Tabachnick and Fidell's (2013) criterion of choice, also suggested the presence one factor which explained 51.58% of the variance. Reliability analysis indicated strong

internal reliability, Cronbach's α = .84. Factor loadings and descriptive statistics are presented in Table 10.3.

Table 10.2*: Factor Loadings and Descriptive Statistics for the Impact on Relationships Scale*

Item	**Loading**
My child's diagnosis has placed my relationship with my partner at risk	.848
My child's diagnosis led to my failed relationship with my partner	.818
My relationship with my partner is negatively affected	.820
I get emotional support from my partner(R)	.682
My child's diagnosis made my relationship with my partner stronger(R)	.630
Eigenvalue	2.92
% Variance Explained	50.98%
M	14.23
SD	4.85
A	.82

Table10.3: Factor Loadings and Descriptive Statistics for the Social Impact Scale

Item	**Loading**
I restrict our outings as a family because of my child's behaviour	.803
I have to leave social gatherings because of my child's behaviour	.792
My social life is affected	.780
Overall my family is affected	.723
I have little time for social activities	.677
I have limited free time	.651
I have little time for entertainment	.571
Eigenvalue	3.61
% Variance Explained	51.58%
M	24.4
SD	6.07
A	.84

10.3.1.4 Feelings Scale

The Kaiser-Meyer-Olkin Measure of Sampling Adequacy (.751) and Bartlett's Test of Sphericity (χ^2_{10} 165.36, $p < .001$) both indicated the factorability of the correlation matrix for these four items. PCA revealed one factor with an eigenvalue greater than one and Cattel's Scree Plot, Tabachnick and Fidell's (2013) criterion of choice, also suggested the presence one factor which explained 59.05% of

the variance. Reliability analysis indicated strong internal reliability, Cronbach's α = 76. Factor loadings and descriptive statistics are shown in Table 10.4.

Table 10.4: *Factor Loadings and Descriptive Statistics for the Feelings Scale*

Item	**Loading**
I feel fatigued	.829
My sleep is disturbed	.819
I feel depressed	.719
I feel ostracized	.698
Eigenvalue	2.36
% Variance Explained	59.05%
M	14.01
SD	3.59
α	.76

10.3.1.5 Financial Scale

The Kaiser-Meyer-Olkin Measure of Sampling Adequacy (.683) and Bartlett's Test of Sphericity (χ^2_{28} 299.52, $p < .001$) both indicated the factorability of the correlation matrix for these eight items. PCA revealed two factors with eigenvalues greater than one and Cattel's scree plot, Tabachnick and Fidell's (2013) criterion of choice, also suggested the presence of two factors in the current data. Together these factors explained 53.71 % of variance. These two factors were labelled financial costs and financial support. Factor loadings and descriptive statistics for these two factors and their correlation are shown in Table 10.5.

Table 10.5: Factor Loadings and Descriptive Statistics for the Financial Scale

Item	Loadings Costs	Loadings Support
My child's therapies are a financial burden	.794	
My financial situation has been affected	.790	
Appointments for my child are expensive	.769	
My child's appointments involve a lot of travel time	.621	
My child's ASD affected my ability to hold a job	.607	
I rely on financial support from others		.835
My family provides financial support when required		.826
My friends help me financially		.327
Eigenvalue	2.6	1.57
% Variance Explained	33.13%	20.58%
Correlation	1	
	-.002	1
M	17.04	4.04
SD	4.4	1.63
α	.76	.54

10.3.1.6 Coping Scale

The Kaiser-Meyer-Olkin Measure of Sampling Adequacy (.632) and Bartlett's Test of Sphericity (χ^2_{153} 475.20/78, $p < .001$) both indicated the factorability of the correlation matrix. There were four eigenvalues greater than one but Cattel's scree plot, Tabachnick and Fidell's (2013) criterion of choice, indicated the presence of two factors which explained 36.12% of the variance. These factors were labelled Pro-Active Engagement and Management. Cronbach's alpha for these scales was .68 and .62, respectively. Factor loadings and descriptive statistics are shown in Table 10.6.

Table10.6: Factor Loadings and Descriptive Statistics for the Coping Scale

Item	Loading Pro-active	Loading Management
I actively search for solutions to each of the issues faced by my child	.676	
I try to introduce new food tastes to my child's diet	.654	
I try to make meal time fun	.571	
I can identify triggers that make my child upset	.564	
I try to remain calm when my child gets upset	.510	
I am willing to implement new treatments and therapies that promises improvement in my child	.491	
I believe with therapies and hard work my child will improve significantly	.466	
I have changed my life to suit my child with ASD	.421	
I find medications help with my child's behavioural issues		.734
I find medication help with my child's medical issues		.725
I can handle my child's aggressive behaviour(R)		.557
I find it hard to make changes in my life to manage my child's ASD		.518
I find it hard to keep up with my child		.518
Eigenvalue	2.556	2.14
% Variance Explained	19.66%	16.46%
Correlation	1	
	.001	1
M	28.15	14.14
SD	4.55	3.92
α	.68	.62

10.4 Discussion Study 3A

The aim in this study was to develop scales specific to the impact on families living with a child with ASD. The preference for domain-specific scales follows the example of several past researchers. For instance, the Multidimensional Health Locus of Control Scale (Wallston, K, Wallston, B., & DeVellis, 1978), the Domain-Specific Resilient Systems Scales (Maltby, Day, Hall, & Chivers, 2017) and the Domain-Specific Risk-Attitude Scale (Weber, Blais, & Betz, 2002) and a subsequent recommendation by Bandura (1986) although, at the time, he was referring to self-efficacy. The items used in the design of the current scales were derived from information provided directly by families during interviews (see Chapter 9, Study 2).

Of the six scales developed in this study, four were unifactorial: social support; impact on relationship; social impact, and the feelings scales. Each of these scales demonstrates good internal reliability ($\alpha \geq .76$). Two scales written for this study yielded independent structure with two factors each: the financial impact scale yielded factors named financial costs and financial support; and the coping scale yielded factors labelled pro active engagement and management. Three of these factors demonstrated acceptable internal reliability ($\alpha \geq .62$) and although alpha for financial support was poor ($\alpha = .54$) it was decided to retain it for its relevance to families.

The new Social Support Scale reflected both emotional and practical support (e.g., I get emotional support from others; my family looks after my child when required). These elements reflect past findings from McStay et al. (2014) and Paynter et al. (2013) who found that support was linked to variations in families' outcomes. The availability of social support has also been found in past research to moderate the impact of high levels of stress on psychological wellbeing (Cohen, 2004; Payne et al., 2012; Steptoe, 2000) and as such is important for families living with a child with ASD.

As shown in past research, the Impact on Relationships Scale identified the risk to the partners' relationship as a result of their child's ASD (e.g., My child's diagnosis has placed my relationship with my partner at risk; My child's diagnosis led to my failed relationship with my partner). In fact, six of the participants (40%) in Study 2 reported that their marriage broke down due to their child's ASD, and others, including Hock, Timm, and Ramisch (2012); Ramisch et al. (2013); Saini et al. (2015) and Sim, Cordier, Vaz, and Falkmer (2016) have also shown that relationship strain or breakdown is an issue for these families. In fact, research has shown that families living with a child with ASD experience more detrimental effects than those families of children with other disorders (Altiere & von Kluge 2009; Macks & Reeve, 2007; Paynter et al., 2018; Vasilopoulou & Nisbet, 2016; Yamada et al., 2012).

The impact on family's socialisation was shown clearly in the Social Impact Scale (e.g., I restrict our outings as a family because of my child's behaviour; I have to leave social gatherings because of my child's behaviour). Past studies have also reported this effect (e.g., Dyke et al., 2009; Ooi et al., 2016; Divian et al., 2012). Others too, have shown that the behaviour of the child with ASD disrupts all areas of family life (Khanna, et al., 2011; Kuhlthau et al., 2014; Tung et al., 2014) creating multiple challenges and leading to a range of negative outcomes including scarcity of time for parents as a couple (Hock et al., 2012; Saini et al., 2015; Sim et al., 2016). Past research has indicated that parents of children with ASD are vulnerable to relationship stress, lower marital satisfaction, and potential relationship dissolution (Ramisch, Timm, Hock, & Topor, 2013).

The Feelings Scale captured states of fatigue, sleep disturbance as well as feelings of being ostracized. Dyke et al. (2009) are among those who have identified feelings of embarrassment and ostracism among families of children with ASD while Giallo, Wood, Jellett, and Porter (2013) found that the parents of children with ASD reported significantly greater fatigue than parents of normally developing children. A part of the fatigue parents experience has been attributed to the child's sleeplessness or even a sleep disorder (Klukowski et al., 2015; Rossignol & Frye, 2011) which Miano, Giannotti, and Cortesi (2016) reported to be the case in up to 50% of children with ASD. As a result, parents often experience unrelenting stress as a result of what is often a chronic lack of sleep (Gorlin et al., 2016).

In terms of the Financial Scale, two factors emerged: financial costs (e.g., My child's therapies are a financial burden; My child's ASD affected my ability to hold a job) and financial support (e.g., I rely on financial support from others; My friends help me financially). The greater investment in health care for the child with ASD has also been shown previously to exacerbate the family's financial burden (Fletcher et al., 2012; Järbrink, 2007; Karst & van Hecke, 2012). The time demands of therapies also place a restriction on parents' ability to engage in paid employment (Fletcher, Markoulakis, & Bryden, 2012; Matthews et al., 2011; Sitimin, Fikry, Ismail, & Hussein, 2017) which, in

conjunction with greater investment in health care (Fletcher et al., 2012; Järbrink, 2007; Karst & van Hecke, 2012), exacerbates the family's financial burden.

In terms of the Coping Scale, two factors were extracted: Pro-active engagement (e.g., I actively search for solutions to each of the issues faced by my child) and Management (e.g., I find medications help with my child's behavioural issues). Certainly, pro-active coping or seeing things as a challenge has been reported by several authors (e.g., Greenglass, 2009; Moore, 2003) to include positive strategies to address and challenge difficulties. Furthermore, use of these strategies has been associated with better health outcomes (e.g., McStay et al., 2014; Paynter et al., 2013). These proactive strategies by families of children with ASD are largely in contrast to some past research. For example, Benson et al. (2010) and Hastings et al. (2005) described these parents as engaging in active avoidance while Pisula and Kossakowska (2010) found that such parents engage in escape avoidance strategies, that is, parents attempt to avoid dealing with situations around the child's ASD.

In sum, the six scales developed from parents and caregivers' interview data have yielded good construct validity and overall, reasonable internal reliability. It is proposed that these scales be used to test a modified version of the Double ABCX Model (see Chapter 5.6) of the Impact of Living with a Child with ASD.

10.5 Chapter Summary

In this Chapter, the development and reliability of six new domain-specific scales to assess the impact of living with a child with ASD were presented. These scales: Social Support Scale, Impact on Relationships Scale, Social Impact Scale, Feelings Scale, Financial Scale and Coping Scale all yielded good construct validity and reasonable internal reliability. The findings generally reflect several factors reported in previous studies but, it is suggested, these scales are more relevant as they are domain-specific. These scales will be used in a test of a model of the impact of living with a child with ASD to be reported next.

Chapter 11. Study 3B: Exploratory Model of the Impact of Living with a Child with ASD

Several studies have looked at the impact of ASD on those experiencing the disorder (Criado et al., 2017; Frye & Rossignol, 2016; Klukowski et al., 2015; Tang et al., 2011) but fewer studies (e.g., Kamio & Inada, 2014; Lavelle et al., 2014) have investigated the impact on the family. Of the published studies that have considered the effects on parents or families of a child with ASD, no researcher has used ASD domain-specific scales when modelling the effects on families.

11.1 Aim

The aim in the following analysis was to test the hypothesised model of *The Impact of Living with a Child with ASD* using a modified version of the Double ABCX model (McCubbin & Patterson, 1983) (Figure 5.6, Extended Figure 11.1) which itself is an extension of the ABCX Model (Hill, 1949). The scales derived from the qualitative data and completed by participants in Study 3A, will be utilised to test the model. In this instance, the fact that participants are living with a child with a chronic disability is considered the à priori and ongoing stressor described by McCubbin and Patterson (1983) in the Double ABCX Model.

Participants' levels of perceived support, in this instance both social and financial support, together with the coping strategies they use to manage and proactively challenge their circumstances will constitute their inferred levels of resilience. The impact on family will be determined by their assessments of the financial costs associated with living with their child with ASD, their feelings or mood, and the impact on their social life and on their intimate relationship.

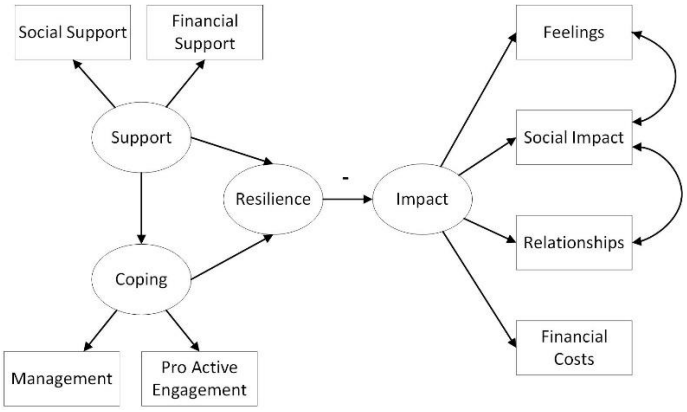

Figure 11.1 Proposed model of the impact on Families living with a child with ASD.

11.2 Analysis

Structural Equational Modelling (SEM) (Amos, Version 26) was used to test the hypothesised model using data from the 162 participants who responded to Study 3A and who completed all scales (see Chapter 10.3).

11.3 Results

Overall, the data provided an adequate fit to the model (Table 11.1; Figure 11.2). As expected, resilience was inferred by participants' levels of support, notably social support which loaded highly on the latent variable (β = .91, *p* <.001) but less so financial support (β = .13, *p* <.05), and by coping, that is, participants' strategies to manage difficulties (β = .77, *p* <.001), but not their levels of proactive engagement (β = .09, *p* >.05). The variance explained in resilience by support and coping was 85%.

Table 11.1: Goodness of Fit Statistics for the Model of Impact of Living with a child with ASD

	χ^2	*Df*	*p*	χ^2/df	GFI	AGFI	RMSEA	p/close	SRMR
Hypothesised Model	74.39	20	<.001	3.72	.905	.829	.13	<.001	.10

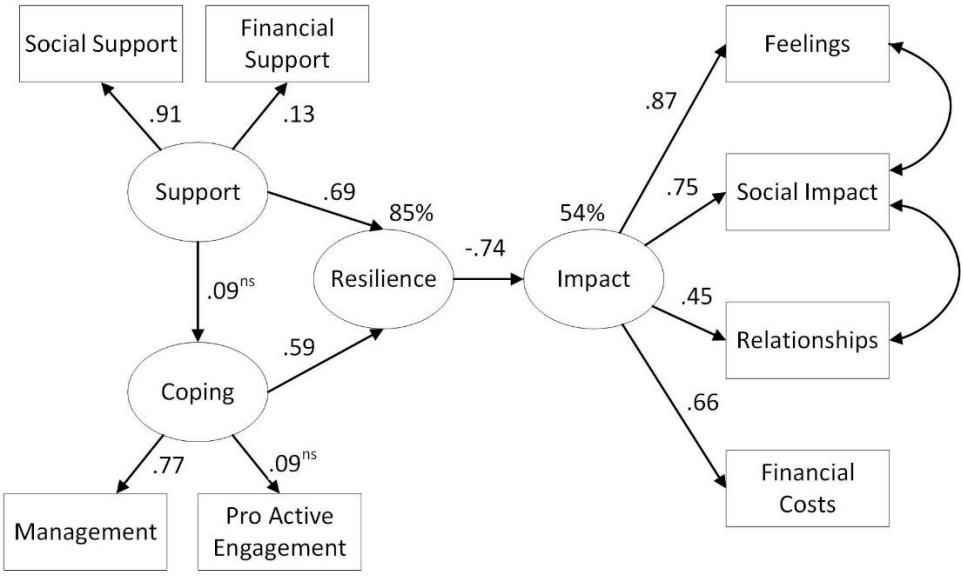

Figure 11.2 Proposed Model of Impact on Families Living with a Child with ASD

As hypothesised, participants' feelings (β = .87, *p* <.001), followed by the social impact on the family (β = .75, *p* <.001), the financial costs associated with living with a child with ASD (β = .66, *p* <.001), and the effect on the parental relationship (β = .45, *p* <.001) each contributed to the latent construct of the Impact on Family of living with a child with ASD.

Overall, 54% of the variance in the Impact on Family was significantly negatively explained by the latent construct of Resilience (β = -.74), as operationalised by the measures of support and coping. Statistical modifications suggested by AMOS (e.g., financial costs to pro-active engagement) would have improved the overall fit of the data to the model, but these were not applied, partly for reasons of parsimony.

11.4 Discussion

The proposed model of the Impact of Living with a Child with ASD was largely supported by the data. The inferred construct of Impact was, as hypothesised, explained by participants' Feelings (e.g., I feel depressed; I feel fatigued; I feel ostracized); the Social Impact on the family (e.g., I restrict our outings as a family because of my child's behaviour; I have little time for social activities); the Financial Costs associated with living with a child with ASD (e.g., My child's therapies are a financial burden; My child's ASD affected my ability to hold a job); and the effect on the Parental Relationship (e.g., My child's diagnosis has placed my relationship with my partner at risk; My child's diagnosis led to the failed relationship with my partner).

Support was inferred by participants' scores on social support (e.g., I get emotional support from others; I am satisfied with the help I get from others) and financial support (e.g., I rely on financial support from others; My friends help me financially). Coping was a combination of proactive engagement (e.g., I actively search for solutions to each of the issues faced by my child; I try to make meal time fun) and management strategies (e.g., I find medications help with my child's behavioural issues) however, proactive engagement (β = 09) was only borderline (p <.10). Support and coping as hypothesised contributed to the latent construct of Resilience and explained 85% of the variance in that latent construct. High levels of Resilience acted to reduce the impact of living with a child with ASD (54% variance explained).

The results of this model provide support for much previous research. The factors which contributed to impact in this model: social impact, financial costs, feelings and impact on the parental relationship have each been reported in previous research (Dyke et al., 2009; Fletcher et al., 2012; Matthews et al., 2011; Rogge & Janssen, 2019).

These impact factors are also, by default, closely aligned and individually offer support to past research. For instance, financial difficulties limit the family's socialising (Meny et al., 2018) and the ability of siblings to attend extra curricular activities (Rao & Beidel, 2009; Wingston et al., 2017) as

well as having an impact on participants' negative feelings (Chan et al., 2018; Divan et al., 2012; Meny et al., 2018) and the quality of their relationship (Papp & Hartley, 2019).

Following past research, for example Moore et al. (2017), we hypothesised that coping and support from others, would contribute to participants' sense of resilience. This hypothesis was supported for both components of support: social and financial and for management strategies as a coping strategy. A sense of resilience has been associated with positive outcomes in parents of children with ASD in past research (e.g., Byrne et al., 2018) and, clearly, resilience is an important protective and adaptive factor in one's ability to deal with stressors, not less so family stressors. The combination of resources, that is support, and coping were referred to as capabilities by Patterson (1988) in the FAAR Model and by McCubbin and Patterson (1982) in their DABCX Model. In each of these models, the authors hypothesised that capabilities would reduce impact or enhance adaptation.

Overall, the current data provided an acceptable fit of the data to the model. While the program AMOS suggested some statistical modifications to improve the fit, these were not implemented for several reasons. The first was for parsimony, but more importantly following Cohen it is argued that "exploratory studies {such as this} do not result in conclusions, but in hypotheses, which then need to be tested (or, depending on the research context, cross-validated)" (Cohen, 1968, *p*. 442). Future research is required therefore, to validate this proposed model in a larger longitudinal study.

One limitation of the current findings relates to the relatively small sample size (N =162) which, although providing sufficient power for the SEM analysis, did not permit the use of a hold-out sample for cross-validation purposes. A further consideration is that while all references have been to families, only one member of the family, typically the mother, completed the assessments. Future studies would be strengthened by comparing the responses of dyads in the parental relationship as well as the stability of the model across the dyads. It may also be important for future research to

ascertain if the impact of living with a child with ASD varies across the three diagnostic levels of ASD severity.

Despite the limitations of the current analysis and its' cross-sectional nature, the data do provide insight into the impact reported by families who live with a child with ASD. The data also indicate that social support is imperative to the wellbeing of these families as are strategies to help them proactively engage with and manage their circumstances. By inference, the data also suggest that further education is required to destigmatize ASD and the families with a child with ASD, so that further support can be provided not only by family and friends, but the wider community.

11.5 Chapter Summary

The aim in this chapter was to test a model of the impact on families living with a child with ASD based on a modified version of the Double ABCX model. Results indicate overall support for the model. The factors which contributed to the impact were: social impact, financial costs, feelings and impact on the parental relationship. Resilience, comprising participants' social and financial support and coping strategies, acted to reduce the impact of living with a child with ASD. Although the SEM program (AMOS) suggested modifications to the model to improve the fit of the data, these were not implemented. Rather Cohen's (1988) recommendation that such models are exploratory and require subsequent validation was heeded.

Chapter 12. Conclusion

Autism Spectrum Disorder (ASD) is a debilitating lifelong condition with an unknown aetiology. The prevalence rate of ASD in Australia is estimated to be 1.10% among 4-year olds (May & Williams, 2018). Studies consistently show that boys have a higher prevalence rate for ASD than girls (between 3 to 5:1) (Burstyn et al., 2010; Loomes et al., 2017) although why this is so, is not known. There are no specific treatments for ASD, rather both pharmacological and non-pharmacological, treatments are aimed at the symptoms of ASD (e.g, repetitive speech and routines, tantrums and self-harm) (Kim et al., 2016; Soke et al., 2016), any co-morbidities (e.g., anxiety) (Kreslins et al., 2015; Sukhodolsky et al., 2013; Ung et al., 2015), and sleep disturbances (Sharma et al., 2018). This lack of specific treatments may be because the aetiology of ASD is unknown despite much research which has been directed towards ascertaining the cause/s of ASD.

One relatively recent hypothesis put forward by Cannell (2008; 2017) was that Vitamin D may be implicated in the onset and/or the maintenance of ASD. Some research has provided tentative support for this hypothesis by demonstrating Vitamin D deficiencies in those with ASD (Jia, Wang, Shan, Xu, Staal, & Du, 2015; Stubbs, Henley, & Green, 2016) although whether this deficiency is cause or effect is not clear. Vitamin D is synthesised through the body's absorption of UVB through sunlight and therefore it was hypothesised that the prevalence of ASD would vary by latitude, that is, as distance from the equator increases where the hours and intensity of sunlight are greatest, the rates of ASD would also increase.

In Study 1, a review of the international literature, as well as one Australian study across wide latitudes, provided some tentative support for the hypothesis that the prevalence rates of ASD would increase as distance from the equator increases and hours of sunlight correspondingly decrease. This result provides some support for Cannell's proposition that Vitamin D is implicated in ASD. If this proposition holds, other factors may also be involved in determining prevalence rates based on whether people are exposed to sunlight or not, such as the amount of time spent

outdoors, the type of clothing worn when in sunlight, and one's skin pigmentation. Whatever the aetiology of ASD and the role of Vitamin D, it remains clear that ASD has an impact on the person experiencing it and also on the family. It is recommended that children, especially those with ASD, spend more time in outdoor activities where exposure to sunlight may have some beneficial effects. Results of the extant research show the therapeutic benefits of outdoor activity for children with ASD, such as motor-sensory, emotional and social benefits (Li et al., 2019) as discussed earlier. In conjunction with the findings from Study 1, based on Cannell's (2008; 2017) hypothesis that Vitamin D is implicated in ASD, it is advisable for parents and caregivers to ensure that children engage in appropriate outdoor activities as part of their lifestyle. More research is warranted to further elucidate the benefits of outdoor activity on children with ASD. If this is the case, it may lead to a reduction in the impact on families living with a child with ASD.

Several researchers have investigated the impact of living with a child with ASD (Altiere & von Kluge 2009; Kamio & Inada, 2014; Lavelle et al., 2014; Paynter et al., 2018; Tsai et al., 2018) but in Study 2 of this thesis, the interest was on ascertaining the impact on families living in a regional area of Victoria, Australia. Interviews with 16 independent participants caring for a child with ASD revealed seven psychosocial themes: regional living; financial; participant's personal and family lives; child's health and behaviour; child's school and school-time; concerns about child's future; and limited support. In addition the data revealed five coping themes which were a mixture of proactive and avoidant strategies: time management, social withdrawl, emotional coping, medication and problem solving. Clearly, participants' families were affected by their child's disability. Living in a regional area added to parents' burden due to the lack of nearby facilities and inadequate access to healthcare providers. The additional time required for parents to travel to therapy and medical appointments led many mothers to quit their jobs. This reduction in or lack of income exacerbated the monetary burden on families and reduced their ability to engage in social activities and other entertainments. Encouraging suitably qualified specialist practitioners and therapists to relocate to these regional areas and/or having practitioners travel into regional areas on a regular, consultancy

basis is a possible solution. This approach might be more successful if Government increased financial incentives (fee/rebates) to the practitioners traveling to these areas or, as is the case for practitioners coming from overseas, practitioners were required to spend some time in rural and regional areas post registration.

Past empirical research into the impact on families who live with a child with ASD has used a range of scales, none of which were domain-specific. Following Bandura's (1986) recommendation and the work of others (e.g., Maltby et al., 2017; Wallston et al., 1978 Weber et al., 2002) the data from the interviews conducted for this thesis were used to develop domain-specific measures in Study 3A. These measures were tested in a sample of 162 participants caring for a child with ASD. All six scales written for this study (i.e., social support, impact on relationship, social impact, feelings, financial impact and coping) were submitted to Principal Components Analysis and reliability analysis and yielded satisfactory psychometric properties. These scales were then used to test a modified version of the Double ABCX Model (McCubbin & Patterson, 1982; 1983) (Study 3B) where living with a child with ASD was considered the à priori and ongoing stressor. In this modified model, it was proposed that coping and support would contribute to participants' sense of resilience which in turn, would reduce the impact experienced by families.

The data provided tentative support for the model. The proposal that coping and support would contribute to the latent construct of Resilience was partially supported. Social support, financial support and management coping strategies but not proactive engagement which was in the doubtful range (p >.05 <.10), loaded onto resilience. In terms of the impact of living with a child with ASD, this latent construct reflected participants' views of the financial costs associated with their child's ASD, the impact on their social life and on their relationships, as well as their feelings. As hypothesised, high levels of resilience, as operationalised by support and coping – two factors which are amenable to change – negatively predicted the impact of living with a child with ASD, explaining 54% of the variance in Impact.

Clearly, families living with a child with ASD face many difficulties. Some of these can be ameliorated by public awareness of the social impact families might experience as a result of their child's ASD (e.g., ostracism by others in social and public places), financial assistance from government and greater provision of medical and other therapeutic resources[4], as well as interventions to assist families deal with their own feelings regarding their child's disability and the impact it has on their partner relationship. Families also need to be made aware of their rights and those of their child, notably when the child is subject to bullying or ostracism at school or more broadly. Future research could explore parents' current understanding of these rights. Another area for future investigation is reframing. Manning et al.'s (2011) showed that reframing allowed parents to view the situation of having a child with autism in a more positive light and related to lower parenting stress, a future study might investigate reframing specifically in relation to regional living. The question to investigate would be the relation between reframing as a predictor of both family functioning and parental distress and how reframing may allow parents to view the situation of having a child with autism in a more positive light. Another area of research within the reframing scope would be the utility of reframing across a range of behaviour problems and how this might help in developing effective coping strategies for the families of children with ASD.

It is also imperative that families be supported both financially but more importantly emotionally to manage the impact of Living with a child with ASD. In the current model, resilience mclearly acts to ameliorate the impact reported by parents and, it is suggested that resilience, as operationalised here, can be developed in families with appropriate assistance. It might also be beneficial for clinicians to work with families to reframe their perceptions of ASD and its impact on their child and family. Such an approach is a central component of cognitive-behavioural therapy.

[4] This thesis was conceived and data collected before the National Disability Insurance Scheme (NDIS) was implemented. The NDIS has removed much of the financial burden for many families. A future study could investigate how effective this program has been in assisting families, not just financially but emotionally as well.

In conclusion, the studies conducted for this thesis have strengthened the hypothesis that Vitamin D synthesised from Ultraviolet radiation (UVB) in sunlight, is implicated in ASD. It is not clear whether this deficiency relates to the aetiology or the maintainence of ASD and its symptoms. Future research by those more qualified to conduct biological studies might address and answer this question.

The impact of living with a child with ASD was clearly confirmed in the current studies as well as determining additional impacts on families living in regional areas among which are reduced access to specialist medical services, time and cost of travel to therapy sessions, and lack of specialist integration aids at local schools. The model of the impact of living with a child with ASD using domain-specific measures developed from the Study 2 data, confirmed the factors which affected families but also, and perhaps more importantly, indicated that family's resilience, that is, their supports and coping strategies, can reduce this impact or at least the perceptions of it. As these studies were exploratory that is, the role of Vitamin D and the effect of sunlight, specifically UVB, on ASD and the cross-sectional evaluation of the model, further work is required to validate each of these conclusions. Despite these limitations, the studies in the current thesis have resulted in some support for the Vitamin D theory of Cannell (2009, 2017), produced domain-specific scales to assess the impact on and coping strategies of, families with a child with ASD, and an innovative model of the impact of living with a child with ASD.

References

Adams, J. B., Johansen, L. J., Powell, L. D., Quig, D., & Rubin, R. A. (2011). Gastrointestinal flora and gastrointestinal status in children with autism–comparisons to typical children and correlation with autism severity. *BMC gastroenterology, 11*(1), 22.

Addo, R. N., Wiens, S., Nord, M., & Larsson, M. (2017). Olfactory functions in adults with autism spectrum disorders. *Perception, 46*(3-4), 530-537.

Agrawal, S., Rao, S. C., Bulsara, M. K., & Patole, S. K. (2018). Prevalence of autism spectrum disorder in preterm infants: a meta-analysis. *Pediatrics, 142*(3), e20180134.

Ahearn, W. H., Castine, T., Nault, K., & Green, G. (2001). An assessment of food acceptance in children with autism or pervasive developmental disorder-not otherwise specified. *Journal of Autism and Developmental Disorders, 31*(5), 505-511.

Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. N. (2015). *Patterns of attachment: A psychological study of the strange situation*. New York: Psychology Press.

Al-Farsi, Y. M., Al-Sharbati, M. M., Al-Farsi, O. A., Al-Shafaee, M. S., Brooks, D. R., & Waly, M. I. (2011). Brief report: Prevalence of autistic spectrum disorders in the Sultanate of Oman. *Journal of Autism and Developmental Disorders, 41*(6), 821-825.

Al-Farsi, Y. M., Waly, M. I., Al-Sharbati, M. M., Al-Shafaee, M. A., Al-Farsi, O. A., Al-Khaduri, M. M., Gupta, I., Ouhtit, A., Al-Adawi, S., Al-Said, M. F. & Deth, R. C. (2013). Levels of heavy metals and essential minerals in hair samples of children with autism in Oman: a case–control study. *Biological Trace Element Research, 151*(2), 181-186.

Al-Yagon, M., & Margalit, M. (2009). Positive and negative affect among mothers of children with intellectual disabilities. *The British Journal of Developmental Disabilities, 55*, 109–127.

Al-Zahrani, A. (2013). Prevalence and clinical characteristics of autism spectrum disorders in school-age children in Taif-KSA. *International Journal of Medical Science and Public Health, 2*(3), 578-582.

Alkozei, A., Smith, R., Dailey, N. S., Bajaj, S., & Killgore, W. D. (2017). Acute exposure to blue wavelength light during memory consolidation improves verbal memory performance. *PloS one, 12*(9), e0184884.

Alon, R. (2019). Social support and post-crisis growth among mothers of children with autism spectrum disorder and mothers of children with Down syndrome. *Research in Developmental Disabilities, 90*, 22-30.

Alonso-Gonzalez, A., Rodriguez-Fontenla, C., & Carracedo, A. (2018). De novo mutations (DNMs) in Autism Spectrum Disorder (ASD): pathway and network analysis. *Frontiers in genetics*, *9*, 406.

American Psychiatric Association (APA) 2013. *Diagnostic and Statistical Manual of Mental Disorders: DSM-5* (5th ed.). Washington, DC: American Psychiatric Association.

Anderson, L. T., Campbell, M., Grega, D. M., Perry, R., Small, A. M., & Green, W. H. (1984). Haloperidol in the treatment of infantile autism: effects on learning and behavioral symptoms. *The American Journal of Psychiatry, 141*(10), 195-202.

Anagnostou, E., Soorya, L., Chaplin, W., Bartz, J., Halpern, D., Wasserman, S., Wang, A. T., Pepa, L., Tanel, N., Kushki, A. & Hollander, E. (2012). Intranasal oxytocin versus placebo in the treatment of adults with autism spectrum disorders: a randomized controlled trial. *Molecular Autism, 3*(1), 16.

Anagnostou, E., Aman, M. G., Handen, B. L., Sanders, K. B., Shui, A., Hollway, J. A., Brian, J., Arnold, L. E., Capano, L., Hellings, J. A. & Butter, E., (2016). Metformin for treatment of overweight induced by atypical antipsychotic medication in young people with autism spectrum disorder: a randomized clinical trial. *JAMA Psychiatry, 73*(9), 928-937.

Anastopoulos, A. D., Guevremont, D. C., Shelton, T. L., & DuPaul, G. J. (1992). Parenting stress among families of children with attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology, 20*(5), 503-520.

Angell, R. O. (1936). *The family encounters the Depression*. New York: Charles Scribner.

Antonovsky, A. (1979*) Health, stress and coping. The Jossey-Bass social and behavioral science series*. Jossey-Bass Inc, San Francisco.

Antonovsky, A. (1993). The structure and properties of the sense of coherence scale. *Social Science & Medicine, 36*(6), 725-733.

Antonovsky, A., & Sourani, T. (1988). Family sense of coherence and family adaptation. *Journal of Marriage and the Family*, 79-92.

Argyropoulos, A., Gilby, K., & Hill-Yardin, E. L. (2013). Studying autism in rodent models: reconciling endophenotypes with comorbidities. *Frontiers in Human Neuroscience, 7*, 417.

Atun-Einy, O., Lotan, M., Harel, Y., Shavit, E., Burstein, S., & Kempner, G. (2013). Physical therapy for young children diagnosed with Autism Spectrum Disorders–clinical frameworks model in an Israeli setting. *Frontiers in Pediatrics*, *1*, 19.

Ausderau, K., & Juarez, M. (2013). The impact of autism spectrum disorders and eating challenges on family mealtimes. *ICAN: Infant, Child, & Adolescent Nutrition,* 1941406413502808.

Bakian, A. V., Bilder, D. A., Coon, H., & McMahon, W. M. (2015). Spatial relative risk patterns of autism spectrum disorders in Utah. *Journal of Autism and Developmental Disorders, 45*(4), 988-1000.

Bailey, A., Le Couteur, A., Gottesman, I., Bolton, P., Simonoff, E., Yuzda E., Rutter, M. (1995). Autism as a strongly genetic disorder: evidence from a British twin study. *Psychological Medicine, 25*, 63-77.

Baio, J. (2012). *Prevalence of Autism Spectrum Disorders: Autism and Developmental Disabilities Monitoring Network, 14 Sites, United States, 2008*. Morbidity and Mortality Weekly Report. Surveillance Summaries. Volume 61, Number 3. Centers for Disease Control and Prevention.

Baker, J. K., Seltzer, M. M., & Greenberg, J. S. (2012). Behaviour problems, maternal internalising symptoms and family relations in families of adolescents and adults with fragile X syndrome. *Journal of Intellectual Disability Research, 56*(10), 984-995.

Bandura, A. (1986). *Social foundations of thought and action*. Englewood Cliffs, NJ, 1986.

Branford, D., Bhaumik, S., & Naik, B. (1998). Selective serotonin re-uptake inhibitors for the treatment of perseverative and maladaptive behaviours of people with intellectual disability. *Journal of Intellectual Disability Research, 42*(4), 301-306.

Barber, B. L., Eccles, J. S. & Stone, M. R. (2001). Whatever happened to the jock, the brain, and the princess? Young adult pathways linked to adolescent activity involvement and social identity. *Journal of Adolescent Research, 16*(5), 429-455.

Baribeau, D. A., & Anagnostou, E. (2014). An update on medication management of behavioral disorders in autism. *Current Psychiatry Reports, 16*(3), 437.

Barnett, R. C., & Baruch, G. K. (1985). Women's involvement in multiple roles and psychological distress. *Journal of Personality and Social Psychology, 49*(1), 135.

Baron-Cohen, S., Scott, F. J., Allison, C., Williams, J., Bolton, P., Matthews, F. E., & Brayne, C. (2009). Prevalence of autism-spectrum conditions: UK school-based population study. *The British Journal of Psychiatry, 194*(6), 500-509.

Bass, M. M., Duchowny, C. A., & Llabre, M. M. (2009). The effect of therapeutic horseback riding on social functioning in children with autism. *Journal of Autism and Developmental Disorders, 39*(9), 1261-1267.

Bartram, L. A., Lozano, J., & Coury, D. L. (2019). Aripiprazole for treating irritability associated with autism spectrum disorders. *Expert Opinion on Pharmacotherapy, 20*(12), 1421-1427.

Bauman, M. L. (2010). Medical comorbidities in autism: challenges to diagnosis and treatment. *Neurotherapeutics, 7*(3), 320-327.

Bearss, K., Johnson, C., Smith, T., Lecavalier, L., Swiezy, N., Aman, M., McAdam, D.B., Butter, E., Stillitano, C., Minshawi, N. and Sukhodolsky, D.G., (2015). Effect of parent training vs parent education on behavioral problems in children with autism spectrum disorder: a randomized clinical trial. *JAMA, 313*(15), 1524-1533.

Becerra, T. A., Wilhelm, M., Olsen, J., Cockburn, M., & Ritz, B. (2013). Ambient air pollution and autism in Los Angeles county, California. *Environmental Health Perspectives (Online), 121*(3), 380.

Becerra, T. A., von Ehrenstein, O. S., Heck, J. E., Olsen, J., Arah, O. A., Jeste, S. S., Rodriguez, M. & Ritz, B. (2014). Autism spectrum disorders and race, ethnicity, and nativity: a population-based study. *Pediatrics, 134*(1), e63-e71.

Becker, J. L., Rogers, E. C., & Burrows, B. (2017). Animal-assisted social skills training for children with autism spectrum disorders. *Anthrozoös, 30*(2), 307-326.

Benson, P. R. (2010). Coping, distress, and well-being in mothers of children with autism. *Research in Autism Spectrum Disorders, 4*(2), 217-228.

Benson, P. R. (2012). Network characteristics, perceived social support, and psychological adjustment in mothers of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 42*(12), 2597-2610.

Benson, P. R., & Karlof, K. L. (2009). Anger, stress proliferation, and depressed mood among parents of children with ASD: A longitudinal replication. *Journal of Autism and Developmental Disorders, 39*(2), 350-362.

Bent, C. A., Dissanayake, C., & Barbaro, J. (2015). Mapping the diagnosis of autism spectrum disorders in children aged under 7 years in Australia, 2010–2012. *Medical Journal of Australia, 202*(6), 317-320.

Bernaerts, S., Boets, B., Bosmans, G., Steyaert, J., & Alaerts, K. (2020). Behavioral effects of multiple-dose oxytocin treatment in autism: a randomized, placebo-controlled trial with long-term follow-up. *Molecular Autism, 11*(1), 1-14.

Berson, D. M., Dunn, F. A., & Takao, M. (2002). Phototransduction by retinal ganglion cells that set the circadian clock. *Science, 295*(5557), 1070-1073.

Berry, J. O., & Jones, W. H. (1995). The parental stress scale: Initial psychometric evidence. *Journal of Social and Personal Relationships, 12*(3), 463-472.

Bhat, A. N., Landa, R. J., & Galloway, J. C. C. (2011). Current perspectives on motor functioning in infants, children, and adults with autism spectrum disorders. *Physical Therapy, 91*(7), 1116.

Bienenstock, J., & Collins, S. (2010). 99th Dahlem Conference on Infection, Inflammation and Chronic Inflammatory Disorders: Psycho-neuroimmunology and the intestinal microbiota: clinical observations and basic mechanisms. *Clinical & Experimental Immunology, 160*(1), 85-91.

Bilaver, L. A., Cushing, L. S., & Cutler, A. T. (2016). Prevalence and correlates of educational intervention utilization among children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 46*(2), 561-571.

Blagrave, A. J., & Colombo-Dougovito, A. M. (2019). Experiences Participating in Community Physical Activity by Families with a Child on the Autism Spectrum: A Phenomenological Inquiry. *Advances in Neurodevelopmental Disorders, 3*(1), 72-84.

Blaurock-Busch, E., Amin, O. R., Dessoki, H. H., & Rabah, T. (2012). Toxic metals and essential elements in hair and severity of symptoms among children with autism. *Maedica–a Journal of Clinical Medicine, 7*(1), 38.

Bodfish, J. W., Symons, F. J., Parker, D. E., & Lewis, M. H. (2000). Varieties of repetitive behavior in autism: Comparisons to mental retardation. *Journal of Autism and Developmental Disorders*, *30*(3), 237-243.

Boilson, A. M., Staines, A., Ramirez, A., Posada, M., & Sweeney, M. R. (2016). Operationalisation of the European Protocol for Autism Prevalence (EPAP) for autism spectrum disorder prevalence measurement in Ireland. *Journal of Autism and Developmental Disorders, 46*(9), 3054-3067.

Bolton, S., McDonald, D., Curtis, E., Kelly, S., & Gallagher, L. (2014). Autism in a recently arrived immigrant population. *European Journal of Pediatrics, 173*(3), 337-343.

Bondy, A. S., & Frost, L. A. (1994). The picture exchange communication system. *Focus on Autistic Behavior, 9*(3), 1-19.

Boss, P. (1987). Family stress. In M. B. Sussman & S. K. Steinmetz (Eds.), *Handbook of marriage and the family* (pp. 695-723). New York, NY: Plenum Press.

Boss, P. (2002). *Family Stress Management* (2nd ed.). Newbury Park, CA: Sage.

Boyd, B. A. (2002). Examining the relationship between stress and lack of social support in mothers of children with autism. *Focus on Autism and Other Developmental Disabilities, 17*(4), 208-215.

Brede, J., Remington, A., Kenny, L., Warren, K., & Pellicano, E. (2017). Excluded from school: Autistic students' experiences of school exclusion and subsequent re-integration into school. *Autism & Developmental Language Impairments, 2*, 1-20.

Bremer, E., Crozier, M., & Lloyd, M. (2016). A systematic review of the behavioural outcomes following exercise interventions for children and youth with autism spectrum disorder. *Autism, 20*(8), 899-915.

Brignell, A., Chenausky, K. V., Song, H., Zhu, J., Suo, C., & Morgan, A. T. (2018). Communication interventions for autism spectrum disorder in minimally verbal children. *Cochrane Database of Systematic Reviews*, (11).

Brimacombe, M., Ming, X., & Lamendola, M. (2007). Prenatal and birth complications in autism. *Maternal and Child Health Journal*, *11*(1), 73-79.

Bristol, M. M. (1987). Mothers of children with autism or communication disorders: Successful adaptation and the double ABCX model. *Journal of Autism and Developmental Disorders, 17*(4), 469-486.

Bromley, J., Hare, D. J., Davison, K., & Emerson, E. (2004). Mothers supporting children with autistic spectrum disorders: Social support, mental health status and satisfaction with services. *Autism, 8*(4), 409-423.

Buchmayer, S., Johansson, S., Johansson, A., Hultman, C. M., Sparén, P., & Cnattingius, S. (2009). Can association between preterm birth and autism be explained by maternal or neonatal morbidity? *Pediatrics, 124*(5), e817-e825.

Buchsbaum, M. S., Hollander, E., Mehmet Haznedar, M., Tang, C., Spiegel-Cohen, J., Wei, T. C., Solimando, A., Buchsbaum, B. R., Robins, D., Bienstock, C., & Cartwright, C. (2001). Effect of fluoxetine on regional cerebral metabolism in autistic spectrum disorders: a pilot study. *International Journal of Neuropsychopharmacology, 4*(2), 119-125.

Buie, T. (2015). Potential etiologic factors of microbiome disruption in autism. *Clinical Therapeutics, 37*(5), 976-983.

Bundy, A. C. (1993). Assessment of play and leisure: Delineation of the problem. *American Journal of Occupational Therapy, 47*(3), 217-222.

Burgess, E. W. (1926). The family as a unity of interacting personalities. *The family, 7*(1), 3-9.

Burr, W. R. (1973). *Theory Construction and the Sociology of the Family*. New York: John Wiley & Sons.

Burstyn, I., Sithole, F., & Zwaigenbaum, L. (2010). Autism spectrum disorders, maternal characteristics and obstetric complications among singletons born in Alberta, Canada. *Chronic Diseases in Canada, 30*(4), 125-134.

Byrne, G., Sarma, K. M., Hendler, J., & O'Connell, A. (2018). On the spectrum, off the beaten path. A qualitative study of Irish parents' experiences of raising a child with autism spectrum conditions. *British Journal of Learning Disabilities, 46*(3), 182-192.

Calderoni, S., Bellani, M., Hardan, A. Y., Muratori, F., & Brambilla, P. (2014). Basal ganglia and restricted and repetitive behaviours in Autism Spectrum Disorders: current status and future perspectives. *Epidemiology and Psychiatric Sciences, 23*(03), 235-238.

Campbell, M., Anderson, L. T., Meier, M., Cohen, I. L., Small, A. M., Samit, C., & Sachar, E. J. (1978). A comparison of haloperidol and behavior therapy and their interaction in autistic children. *Journal of the American Academy of Child Psychiatry, 17*(4), 640-655.

Campbell, M., Anderson, L. T., Small, A. M., Perry, R., Green, W. H., & Caplan, R. (1982). The effects of haloperidol on learning and behavior in autistic children. *Journal of Autism and Developmental Disorders, 12*(2), 167-175.

Campbell, M., Armenteros, J. L., Malone, R. P., Adams, P. B., Eisenberg, Z. W., & Overall, J. E. (1997). Neuroleptic-related dyskinesias in autistic children: a prospective, longitudinal study. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(6), 835-843.

Campbell, S. S., & Murphy, P. J. (1998). Extraocular circadian phototransduction in humans. *Science, 279*(5349), 396-399.

Cannell, J. J. (2008). Autism and vitamin D. *Medical Hypotheses, 70*(4), 750-759.

Cannell, J. J. (2017). Vitamin D and autism, what's new? *Reviews in Endocrine and Metabolic Disorders, 18*(2), 183-193.

Cantwell, J., Muldoon, O. T., & Gallagher, S. (2014). Social support and mastery influence the association between stress and poor physical health in parents caring for children with developmental disabilities. *Research in Developmental Disabilities, 35*(9), 2215-2223.

Canty-Mitchell, J., & Zimet, G. D. (2000). Psychometric properties of the Multidimensional Scale of Perceived Social Support in urban adolescents. *American Journal of Community Psychology, 28*(3), 391-400.

Cappe, É., Poirier, N., Sankey, C., Belzil, A., & Dionne, C. (2018). Quality of life of French Canadian parents raising a child with autism spectrum disorder and effects of psychosocial factors. *Quality of Life Research, 27*(4), 955-967.

Carlisle, G. K. (2015). The social skills and attachment to dogs of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 45*(5), 1137-1145.

Carter, C. S. (2007). Sex differences in oxytocin and vasopressin: implications for autism spectrum disorders? *Behavioural Brain Research, 176*(1), 170-186.

Carter, A. S., Messinger, D. S., Stone, W. L., Celimli, S., Nahmias, A. S., & Yoder, P. (2011). A randomized controlled trial of Hanen's 'More Than Words' in toddlers with early autism symptoms. *Journal of Child Psychology and Psychiatry, 52*(7), 741-752.

Caspersen, C. J., Powell, K. E., & Christenson, G. M. (1985). Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. *Public Health Reports, 100*(2), 126-31.

Cavan, R. S., & Ranck, K. H. (1938). *The family and the Depression*. Chicago: University of Chicago Press.

Celiberti, D. A., Bobo, H. E., Kelly, K. S., Harris, S. L., & Handleman, J. S. (1997). The differential and temporal effects of antecedent exercise on the self-stimulatory behavior of a child with autism. *Research in Developmental Disabilities, 18*(2), 139-150.

Chaidez, V., Hansen, R. L., & Hertz-Picciotto, I. (2014). Gastrointestinal problems in children with autism, developmental delays or typical development. *Journal of Autism and Developmental Disorders, 44*(5), 1117-1127.

Chan, K. K. S., Lam, C. B., Law, N. C. W., & Cheung, R. Y. M. (2018). From child autistic symptoms to parental affective symptoms: A family process model. *Research in Developmental Disabilities, 75*, 22-31.

Charlop, M. H., & Gumaer, C. B. (2019). Use of a speech generating device and various prompting procedures might facilitate vocalizations in minimally verbal children with autism spectrum disorder. *Evidence-Based Communication Assessment and Intervention*, 1-6.

Chen, J., Xin, K., Wei, J., Zhang, K., & Xiao, H. (2016). Lower maternal serum 25 (OH) D in first trimester associated with higher autism risk in Chinese offspring. *Journal of Psychosomatic Research, 89*, 98-101.

Chaddock, L., Erickson, K. I., Prakash, R. S., Kim, J., Voss, M. W., VanPatter, M., & Kramer, A. F. (2010a). A neuroimaging investigation of the association between aerobic fitness, hippocampal volume, and memory performance in preadolescent children. *Brain Research, 1358*, 172–183.

Chaddock, L., Erickson, K.I., Prakash, R.S., VanPatter, M., Voss, M.W., Pontifex, M.B., Raine, L.B., Hillman, C.H. and Kramer, A.F., (2010b). Basal ganglia volume is associated with aerobic fitness in preadolescent children. *Developmental Neuroscience, 32*(3), 249-256.

Chaaya, M., Saab, D., Maalouf, F. T., & Boustany, R. M. (2016). Prevalence of autism spectrum disorder in nurseries in Lebanon: a cross sectional study. *Journal of Autism and Developmental Disorders, 46*(2), 514-522.

Chien, I. C., Lin, C. H., Chou, Y. J., & Chou, P. (2011). Prevalence and incidence of autism spectrum disorders among national health insurance enrollees in Taiwan from 1996 to 2005. *Journal of Child Neurology, 26*(7), 830-834.

Christie, H., Talmon, A., Schäfer, S.K., De Haan, A., Vang, M.L., Haag, K., Gilbar, O., Alisic, E. and Brown, E., (2017). The transition to parenthood following a history of childhood maltreatment: A review of the literature on prospective and new parents' experiences. *European Journal of Psychotraumatology, 8*(sup7), p.1492834.

Chen, A. Y., Kim, S. E., Houtrow, A. J., & Newacheck, P. W. (2010). Prevalence of obesity among children with chronic conditions. *Obesity, 18*(1), 210-213.

Chonchaiya, W., Nuntnarumit, P., & Pruksananonda, C. (2011). Comparison of television viewing between children with autism spectrum disorder and controls. *Acta Paediatrica, 100*(7), 1033-1037.

Cohen, J. (1968). Multiple regression as a general data-analytic system. *Psychological Bulletin, 70*, 426-443.

Cobb, S. (1976). Social support as a moderator of life stress. *Psychosomatic Medicine, 38*, 300-314

Cohen, S. (1988). Psychosocial models of the role of social support in the etiology of physical disease. *Health Psychology, 7*(3), 269.

Cohen, S. (2004). Social relationships and health. *American Psychologist, 59*(8), 676.

Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. Psychological bulletin, 98(2), 310.

Cohrs, A. C., & Leslie, D. L. (2017). Depression in parents of children diagnosed with autism spectrum disorder: A claims-based analysis. *Journal of Autism and Developmental Disorders, 47*(5), 1416-1422.

Colombo-Dougovito, A. M., & Block, M. E. (2019). Fundamental motor skill interventions for children and adolescents on the autism spectrum: A literature review. *Review Journal of Autism and Developmental Disorders, 6*(2), 159–171.

Condy, E. E., Scarpa, A., & Friedman, B. H. (2019). Restricted Repetitive Behaviors in Autism Spectrum Disorder: A Systematic Review from the Neurovisceral Integration Perspective. *Biological Psychology, 148*, 107739.

Couper, L., Sutherland, D., & van Bysterveldt, A. (2013). Children with autism spectrum disorder in the mainstream playground. *Kairaranga, 14*(1), 25-31.

Criado, K. K., Sharp, W. G., McCracken, C. E., De Vinck-Baroody, O., Dong, L., Aman, M. G., McDougle, C. J., McCracken, J. T., Eugene Arnold, L., Weitzman, C. & Leventhal, J. M. (2017). Overweight and obese status in children with autism spectrum disorder and disruptive behavior. *Autism*, 1362361316683888.

Cridland, E. K., Caputi, P., Jones, S. C., & Magee, C. A. (2014). Understanding high-functioning autism during adolescence: A personal construct theory approach. *Journal of Intellectual and Developmental Disability, 39*(1), 108-118.

Critchfield, J. W., Van Hemert, S., Ash, M., Mulder, L., & Ashwood, P. (2011). The potential role of probiotics in the management of childhood autism spectrum disorders. *Gastroenterology research and practice*, 2011:1-8

Cohen, S. (1988). Psychosocial models of the role of social support in the etiology of physical disease. *Health Psychology, 7*(3), 269.

Cohen, S. (2004). Social relationships and health. *American Psychologist, 59*(8), 676.

Cryan, J. F., & Dinan, T. G. (2012). Mind-altering microorganisms: the impact of the gut microbiota on brain and behaviour. *Nature Reviews Neuroscience, 13*(10), 701-712

Curtin, C., Anderson, S. E., Must, A., & Bandini, L. (2010). The prevalence of obesity in children with autism: a secondary data analysis using nationally representative data from the National Survey of Children's Health. *BMC Pediatrics*, *10*(1), 11.

da Silva, C. A., Fernandes, A. R., & Grohmann, A. P. (2014). *STAR: speech therapy with augmented reality for children with autism spectrum disorders*. In International Conference on Enterprise Information Systems (pp. 379-396). Springer, Cham.

Dahlem, N. W., Zimet, G. D., & Walker, R. R. (1991). The multidimensional scale of perceived social support: a confirmation study. *Journal of Clinical Psychology, 47*(6), 756-761.

Danial, J. T., & Wood, J. J. (2013). Cognitive behavioral therapy for children with autism: review and considerations for future research. *Journal of Developmental & Behavioral Pediatrics, 34*(9), 702-715.

Davis, N. O., & Kollins, S. H. (2012). Treatment for co-occurring attention deficit/hyperactivity disorder and autism spectrum disorder. *Neurotherapeutics, 9*(3), 518-530.

Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., Donaldson, A. and Varley, J., (2010). Randomized, controlled trial of an intervention for toddlers with autism: The Early Start Denver Model. *Pediatrics, 125*(1), e17-e23.

Dawson, G., & Burner, K. (2011). Behavioral interventions in children and adolescents with autism spectrum disorder: a review of recent findings. *Current Opinion in Pediatrics, 23*(6), 616-620.

De Rubeis, S., He, X., Goldberg, A.P., Poultney, C.S., Samocha, K., Cicek, A.E., Kou, Y., Liu, L., Fromer, M., Walker, S. and Singh, T., (2014). Synaptic, transcriptional and chromatin genes disrupted in autism. *Nature, 515*(7526), p.209-215

Dealberto, M. J. (2011). Prevalence of autism according to maternal immigrant status and ethnic origin. *Acta Psychiatrica Scandinavica, 123*(5), 339-348.

DeGrace, B. W. (2004). The everyday occupation of families with children with autism. *American Journal of Occupational Therapy, 58*(5), 543-550.

Dekkers, L. M., Groot, N. A., Mosquera, E. N. D., Zúniga, I. P. A., & Delfos, M. F. (2015). Prevalence of autism spectrum disorders in ecuador: a pilot study in quito. *Journal of Autism and Developmental Disorders, 45*(12), 4165-4173.

Denda, M. (2015). Epidermis as the "Third Brain"? *Dermatologica Sinica, 33*(2), 70-73.

Derogatis, L. R., Lipman, R. S., & Covi, L. (1973). SCL-90: an outpatient psychiatric rating scale–preliminary report. *Psychopharmacol Bull, 9*(1), 13-28.

Díaz-Román, A., Zhang, J., Delorme, R., Beggiato, A., & Cortese, S. (2018). Sleep in youth with autism spectrum disorders: systematic review and meta-analysis of subjective and objective studies. *Evidence-based Mental Health, 21*(4), 146-154.

Dickie, V. A., Baranek, G. T., Schultz, B., Watson, L. R., & McComish, C. S. (2009). Parent reports of sensory experiences of preschool children with and without autism: A qualitative study. *American Journal of Occupational Therapy, 63*(2), 172-181.

Dijk, D. J., & Lockley, S. W. (2002). Invited Review: Integration of human sleep-wake regulation and circadian rhythmicity. *Journal of Applied Physiology, 92*(2), 852-862.

Dijk, D. J., & Archer, S. N. (2009). Light, sleep, and circadian rhythms: together again. *PLoS Biology, 7*(6), e1000145.

Dillon, S. R., Adams, D., Goudy, L., Bittner, M., & McNamara, S. (2017). Evaluating Exercise as evidence-based practice for individuals with autism spectrum disorder. *Frontiers in Public Health,* 4, 290.

DiStefano, C., & Kasari, C. (2016). The window to language is still open: Distinguishing between preverbal and minimally verbal children with ASD. *Perspectives of the ASHA Special Interest Groups, 1*(1), 4–11.

Ditzen, B., & Heinrichs, M. (2014). Psychobiology of social support: the social dimension of stress buffering. *Restorative Neurology and Neuroscience, 32*(1), 149-162.

Divan, G., Vajaratkar, V., Desai, M. U., Strik-Lievers, L., & Patel, V. (2012). Challenges, Coping Strategies, and Unmet Needs of Families with a Child with Autism Spectrum Disorder in Goa, India. *Autism Research, 5*(3), 190-200.

Doenyas, C. (2018). Dietary interventions for autism spectrum disorder: New perspectives from the gut-brain axis. *Physiology & Behavior, 194*, 577-582.

DuBois, D., Lymer, E., Gibson, B., Desarkar, P., & Nalder, E. (2017). Assessing sensory processing dysfunction in adults and adolescents with autism spectrum disorder: a scoping review. *Brain Sciences, 7*(8), 108.

Duerden, E. G., Oatley, H. K., Mak-Fan, K. M., McGrath, P. A., Taylor, M. J., Szatmari, P., & Roberts, S. W. (2012). Risk factors associated with self-injurious behaviors in children and adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 42*(11), 2460-2470.

Duerden, E. G., Oatley, H. K., Mak-Fan, K. M., McGrath, P. A., Taylor, M. J., Szatmari, P., & Roberts, S. W. (2012). Risk factors associated with self-injurious behaviors in children and adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 42*(11), 2460-2470.

Durkin, M. S., Maenner, M. J., Newschaffer, C. J., Lee, L. C., Cunniff, C. M., Daniels, J. L., Kirby, R. S., Leavitt, L., Miller, L., Zahorodny, W. and Schieve, L. A. (2008). Advanced parental age and the risk of autism spectrum disorder. *American Journal of Epidemiology, 168*(11), 1268-1276.

Dunst, C. J., Jenkin, V., & Trivette, C. M. (1984). Family Support Scale: Reliability and validity. Journal of Individual. *Family and Community Wellness, 1*, 45–52.

Dunst, C. J. (1985). Rethinking early intervention. *Analysis and Intervention in Developmental Disabilities, 5*(1-2), 165-201.

Dyke, P., Mulroy, S., & Leonard, H. (2009). Siblings of children with disabilities: Challenges and opportunities. *Acta Paediatrica, 98*(1), 23.

Eccles, J. S., Barber, B. L., Stone, M., & Hunt, J. (2003). Extracurricular activities and adolescent development. *Journal of Social Issues, 59*(4), 865-889.

Egan, A. M., Dreyer, M. L., Odar, C. C., Beckwith, M., & Garrison, C. B. (2013). Obesity in young children with autism spectrum disorders: prevalence and associated factors. *Childhood Obesity, 9*(2), 125-131.

Ekas, N. V., Whitman, T. L., & Shivers, C. (2009). Religiosity, spirituality, and socioemotional functioning in mothers of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 39*(5), 706-719.

Emberti Gialloreti, L., Mazzone, L., Benvenuto, A., Fasano, A., Garcia Alcon, A., Kraneveld, A., Moavero, R., Raz, R., Riccio, M.P., Siracusano, M. and Zachor, D.A., (2019). Risk and protective environmental factors associated with autism spectrum disorder: Evidence-based principles and recommendations. *Journal of Clinical Medicine, 8*(2), 217.

Evans, E. W., Must, A., Anderson, S. E., Curtin, C., Scampini, R., Maslin, M., & Bandini, L. (2012). Dietary patterns and body mass index in children with autism and typically developing children. *Research in Autism Spectrum Disorders, 6*(1), 399-405.

Farrugia, D. (2009). Exploring stigma: Medical knowledge and the stigmatization of parents of children diagnosed with autism spectrum disorder. *Sociology of Health & Illness, 31*(7), 1011–1027.

Fedewa, A., & Ahn, S. (2011). The effects of physical activity and physical fitness on children's achievement and cognitive outcomes: A meta-analysis. *Research Quarterly for Exercise and Sport, 82,* 521-535.

Fein, D., Barton, M., Eigsti, I. M., Kelley, E., Naigles, L., Schultz, R. T., Stevens, M., Helt, M., Orinstein, A., Rosenthal, M., & Troyb, E., (2013). Optimal outcome in individuals with a history of autism. Journal of Child Psychology and Psychiatry, 54(2), 195-205.

Fein, D., Barton, M., & Dumont-Mathieu, T. (2017). Optimizing outcome in autism spectrum disorders. *Policy Insights from the Behavioral and Brain Sciences, 4*(1), 71-78.

Feldman, A. F., & Matjasko, J. L. (2005). The role of school-based extracurricular activities in adolescent development: A comprehensive review and future directions. *Review of Educational Research, 75*(2), 159-210.

Feng, J., Shan, L., Du, L., Wang, B., Li, H., Wang, W., Wang, T., Dong, H., Yue, X., Xu, Z. and Staal, W.G., (2017). Clinical improvement following vitamin D3 supplementation in autism spectrum disorder. *Nutritional neuroscience*, *20*(5), pp.284-290.

Fernell, E., Bejerot, S., Westerlund, J., Miniscalco, C., Simila, H., Eyles, D., Gillberg, C. and Humble, M.B., (2015). Autism spectrum disorder and low vitamin D at birth: a sibling control study. *Molecular Autism, 6*(1), p.3.

Fisman, S., Wolf, L., Ellison, D., & Freeman, T. (2000). A longitudinal study of siblings of children with chronic disabilities. *The Canadian Journal of Psychiatry, 45*(4), 369-375.

Flanagan, J. E., Landa, R., Bhat, A., & Bauman, M. (2012). Head lag in infants at risk for autism: a preliminary study. *American Journal of Occupational Therapy, 66*(5), 577-585.

Fletcher, P. C., Markoulakis, R., & Bryden, P. J. (2012). The costs of caring for a child with an autism spectrum disorder. *Issues in Comprehensive Pediatric Nursing, 35*(1), 45-69.

Fombonne, E., Marcin, C., Manero, A.C., Bruno, R., Diaz, C., Villalobos, M., Ramsay, K. & Nealy, B., (2016). Prevalence of autism spectrum disorders in Guanajuato, Mexico: The Leon survey. *Journal of Autism and Developmental Disorders, 46*(5), 1669-1685.

Fortea, M. S., Escandell, M. B., & Castro, J. S. (2013). Estimated prevalence of autism spectrum disorders in the Canary Islands. *Anales de Pediatria (Barcelona, Spain: 2003), 79*(6), 352-359.

Frazier, T. W., Thompson, L., Youngstrom, E. A., Law, P., Hardan, A. Y., Eng, C., & Morris, N. (2014). A twin study of heritable and shared environmental contributions to autism. *Journal of Autism and Developmental Disorders, 44*(8), 2013-2025.

Frye, R. E., & Rossignol, D. A. (2016). Identification and treatment of pathophysiological comorbidities of autism spectrum disorder to achieve optimal outcomes. *Clinical Medicine Insights: Pediatrics, 10*, 43-56.

Funahashi, A., Gruebler, A., Aoki, T., Kadone, H., & Suzuki, K. (2014). Brief report: the smiles of a child with autism spectrum disorder during an animal-assisted activity may facilitate social positive behaviors quantitative analysis with smile-detecting interface. *Journal of Autism and Developmental Disorders, 44*(3), 685–693.

Fung, S. C. (2015). Increasing the social communication of a boy with autism using animal-assisted play therapy: a case report. *Advances in Mind Body Medicine, 29*(3), 27–31.

Fung, L. K., Mahajan, R., Nozzolillo, A., Bernal, P., Krasner, A., Jo, B., Coury, D., Whitaker, A., Veenstra-Vanderweele, J. and Hardan, A. Y. (2016). Pharmacologic treatment of severe irritability and problem behaviors in autism: a systematic review and meta-analysis. *Pediatrics, 137*(Supplement 2), S124-S135.

Furnham, A. (2010). A parental locus of control scale. *Individual Differences Research, 8*(3), 151–163.

Gabriels, R.L., Agnew, J.A., Holt, K.D., Shoffner, A., Zhaoxing, P., Ruzzano, S., Clayton, G.H. and Mesibov, G., (2012). Pilot study measuring the effects of therapeutic horseback riding on

school-age children and adolescents with autism spectrum disorders. *Research in Autism Spectrum Disorders, 6*(2), pp.578-588.

Gabriels, R. L., Pan, Z., Dechant, B., Agnew, J. A., Brim, N., & Mesibov, G. (2015). Randomized controlled trial of therapeutic horseback riding in children and adolescents with autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 54*(7), 541-549.

Gadow, K. D., Roohi, J., DeVincent, C. J., Kirsch, S., & Hatchwell, E. (2010). Brief report: Glutamate transporter gene (SLC1A1) single nucleotide polymorphism (rs301430) and repetitive behaviors and anxiety in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 40*(9), 1139-1145.

Garon, N., Bryson, S. E., Zwaigenbaum, L., Smith, I. M., Brian, J., Roberts, W., & Szatmari, P. (2009). Temperament and its relationship to autistic symptoms in a high-risk infant sib cohort. *Journal of Abnormal Child Psychology, 37*(1), 59-78.

Gray, D. E., & Holden, W. J. (1992). Psycho-social well-being among the parents of children with autism. *Australia and New Zealand Journal of Developmental Disabilities, 18*(2), 83-93.

Gates, J. A., Kang, E., & Lerner, M. D. (2017). Efficacy of group social skills interventions for youth with autism spectrum disorder: A systematic review and meta-analysis. *Clinical Psychology Review, 52*, 164-181.

Geier, D. A., Kern, J. K., King, P. G., Sykes, L. K., & Geier, M. R. (2012). Hair toxic metal concentrations and autism spectrum disorder severity in young children. *International Journal of Environmental Research and Public Health, 9*(12), 4486-4497.

Genesoni, L., & Tallandini, M. A. (2009). Men's psychological transition to fatherhood: an analysis of the literature, 1989–2008. *Birth, 36*(4), 305-318.

Geretsegger, M., Elefant, C., Mössler, K., & Gold, C. (2014). Music therapy for people with autism spectrum disorder. *Cochrane Database of Systematic Reviews*, (6).

Ghayan, A. (2017). *Neurophology*. Islamabad: Subagh.

Ghayan, A., Ghayan, S. (2014). *Reducing Autism Poisoning Impacts*. Islamabad: Subagh.

Ghayan A. & Ghayan, S. (2017). Case report on long-term autism therapy involving fresh food diet, outdoor activity and sunlight. In A. Ghayan (Ed.), Autism and Sunlight (pp. 23-30). Islamabad: Subagh.

Giallo, R., & Gavidia-Payne, S. (2006). Child, parent and family factors as predictors of adjustment for siblings of children with a disability. *Journal of Intellectual Disability Research, 50*(12), 937-948.

Giallo, R., Wood, C. E., Jellett, R., & Porter, R. (2013). Fatigue, wellbeing and parental self-efficacy in mothers of children with an autism spectrum disorder. *Autism, 17*(4), 465-480.

Gordon-Lipkin, E., Marvin, A. R., Law, J. K., & Lipkin, P. H. (2018). Anxiety and mood disorder in children with autism spectrum disorder and ADHD. *Pediatrics, 141*(4), e20171377.

Gordon, K., Pasco, G., McElduff, F., Wade, A., Howlin, P., & Charman, T. (2011). A communication-based intervention for nonverbal children with autism: What changes? Who benefits? *Journal of Consulting and Clinical Psychology, 79*(4), 447–457.

Gorlin, J. B., McAlpine, C. P., Garwick, A., & Wieling, E. (2016). Severe childhood autism: The family lived experience. *Journal of Pediatric Nursing, 31*(6), 580-597.

Grant, W. B., & Cannell, J. J. (2013). Autism prevalence in the United States with respect to solar UV-B doses: an ecological study. *Dermato-endocrinology, 5*(1), 159-164.

Grant, W. B., & Soles, C. M. (2009). Epidemiologic evidence for supporting the role of maternal vitamin D deficiency as a risk factor for the development of infantile autism. *Dermato-Endocrinology, 1*(4), 223-228.

Gray, D. E. (2006). Coping over time: The parents of children with autism. *Journal of Intellectual Disability Research, 50*(12), 970-976.

Gray, D. E., & Holden, W. J. (1992). Psycho-social well-being among the parents of children with autism. *Australia and New Zealand Journal of Developmental Disabilities, 18*(2), 83-93.

Green, J., Charman, T., McConachie, H., Aldred, C., Slonims, V., Howlin, P., Le Couteur, A., Leadbitter, K., Hudry, K., Byford, S., & Barrett, B. (2010). Parent-mediated communication-focused treatment in children with autism (PACT): a randomised controlled trial. *The Lancet, 375*(9732), 2152-2160.

Greenspan, S. I., & Wieder, S. (1997). Developmental patterns and outcomes in infants and children with disorders in relating and communicating: A chart review of 200 cases of children with autistic spectrum diagnoses. *Journal of Developmental and Learning Disorders, 1*, 87-142.

Green, S. A., & Ben-Sasson, A. (2010). Anxiety disorders and sensory over-responsivity in children with autism spectrum disorders: is there a causal relationship? *Journal of Autism and Developmental Disorders, 40*(12), 1495-1504.

Green, S. A., Ben-Sasson, A., Soto, T. W., & Carter, A. S. (2012). Anxiety and sensory over-responsivity in toddlers with autism spectrum disorders: Bidirectional effects across time. *Journal of Autism and Developmental Disorders, 42*(6), 1112-1119.

Green, S. A., Rudie, J. D., Colich, N. L., Wood, J. J., Shirinyan, D., Hernandez, L., Tottenham, N., Dapretto, M. and Bookheimer, S. Y. (2013). Overreactive brain responses to sensory stimuli in youth with autism spectrum disorders. *Journal of the American Academy of Child & Adolescent Psychiatry, 52*(11), 1158-1172.

Green, J., Charman, T., Pickles, A., Wan, M.W., Elsabbagh, M., Slonims, V., Taylor, C., McNally, J., Booth, R., Gliga, T. and Jones, E. J. (2015). Parent-mediated intervention versus no

intervention for infants at high risk of autism: a parallel, single-blind, randomised trial. *The Lancet Psychiatry*, *2*(2), 133-140.

Guastella, A. J., & Hickie, I. B. (2016). Oxytocin treatment, circuitry, and autism: a critical review of the literature placing oxytocin into the autism context. *Biological Psychiatry, 79*(3), 234-242.

Guinchat, V., Thorsen, P., Laurent, C., Cans, C., Bodeau, N., & Cohen, D. (2012). Pre-, peri-and neonatal risk factors for autism. *Acta Obstetricia et Gynecologica Scandinavica, 91*(3), 287-300.

Hallmayer, J., Cleveland, S., Torres, A., Phillips, J., Cohen, B., Torigoe, T., Miller, J., Fedele, A., Collins, J., Smith, K., Lotspeich, L., Croen, L. A., Ozonoff, S., Lajonchere, C., Grether, J. K., & Risch, N. (2011). Genetic heritability and shared environmental factors among twin pairs with autism. *Archives of General Psychiatry, 68*(11), 1095-1102.

Hampton, L. H., & Kaiser, A. P. (2016). Intervention effects on spoken-language outcomes for children with autism: a systematic review and meta-analysis. *Journal of Intellectual Disability Research, 60*(5), 444-463.

Handen, B. L., Sahl, R., & Hardan, A. Y. (2008). Guanfacine in children with autism and/or intellectual disabilities. *Journal of Developmental & Behavioral Pediatrics, 29*(4), 303-308.

Hanley, B., Tassé, M. J., Aman, M. G., & Pace, P. (1998). Psychometric properties of the family support scale with head start families. *Journal of Child and Family Studies, 7*(1), 69-77.

Hartshorn, K., Olds, L., Field, T., Delage, J., Cullen, C., & Escalona, A. (2001). Creative movement therapy benefits children with autism. *Early Child Development and Care, 166*(1), 1–5.

Hartley, S. L., Barker, E. T., Seltzer, M. M., Floyd, F., Greenberg, J., Orsmond, G., & Bolt, D. (2010). The relative risk and timing of divorce in families of children with an autism spectrum disorder. *Journal of Family Psychology, 24*(4), 449.

Hastings, R. P., & Brown, T. (2002). Behavior problems of children with autism, parental self-efficacy, and mental health. *American Journal on Mental Retardation, 107*(3), 222-232.

Hastings, R. P., & Johnson, E. (2001). Stress in UK families conducting intensive home-based behavioral intervention for their young child with autism. *Journal of Autism and Developmental Disorders, 31*(3), 327-336.

Hastings R., & Beck A. (2004). Practitioner review: Stress interventions for parents of children with intellectual disabilities. *Journal of Child Psychology and Psychiatry, 45*(8), 1338–1349.

Hastings, R. P., Kovshoff, H., Brown, T., Ward, N. J., Espinosa, F. D., & Remington, B. (2005). Coping strategies in mothers and fathers of preschool and school-age children with autism. *Autism, 9*(4), 377-391.

Healy, S., Nacario, A., Braithwaite, R. E., & Hopper, C. (2018). The effect of physical activity interventions on youth with autism spectrum disorder: A meta-analysis. *Autism Research, 11*(6), 818-833.

Heaton, P. (2009). Assessing musical skills in autistic children who are not savants. *Philosophical Transactions of the Royal Society B: Biological Sciences, 364*(1522), 1443-1447.

Heijtz, R. D., Wang, S., Anuar, F., Qian, Y., Bjorkholm, B., Samuelsson, A., Hibberd, M. L., Forssberg, H., Pettersson, S. (2011). Normal gut microbiota modulates brain development and behavior. *Proceedings of the National Academy of Sciences USA, 108*:3047–3052.

Heil, K. M., & Schaaf, C. P. (2013). The genetics of autism spectrum disorders–a guide for clinicians. *Current Psychiatry Reports, 15*(1), 334.

Hesselmark, E., Plenty, S., & Bejerot, S. (2014). Group cognitive behavioural therapy and group recreational activity for adults with autism spectrum disorders: A preliminary randomized controlled trial. *Autism, 18*(6), 672-683.

Higashida, H., Munesue, T., Kosaka, H., Yamasue, H., Yokoyama, S., & Kikuchi, M. (2019). Social interaction improved by oxytocin in the subclass of autism with comorbid intellectual disabilities. *Diseases, 7*(1), 24.

Hill, R. (1949). *Families under stress*. New York: Harper & Brothers.

Hill, R. (1958). Generic features of families under stress. *Social Casework, 49*, 139-150.

Hill, A.P., Zuckerman, K.E., Hagen, A.D., Kriz, D.J., Duvall, S.W., Van Santen, J., Nigg, J., Fair, D. and Fombonne, E., (2014). Aggressive behavior problems in children with autism spectrum disorders: prevalence and correlates in a large clinical sample. *Research in Autism Spectrum Disorders, 8*(9), 1121-1133.

Hill, A. P., Zuckerman, K. E., & Fombonne, E. (2015). Obesity and autism. *Pediatrics, 136*(6), 1051.

Hoagwood, K. E., Acri, M., Morrissey, M., & Peth-Pierce, R. (2017). Animal-assisted therapies for youth with or at risk for mental health problems: A systematic review. *Applied Developmental Science, 21*(1), 1-13.

Hock, R. M., Timm, T. M., & Ramisch, J. L. (2012). Parenting children with autism spectrum disorders: A crucible for couple relationships. *Child & Family Social Work, 17*(4), 406-415.

Hodapp, R. M. (1997). Direct and indirect behavioral effects of different genetic disorders of mental retardation. *American Journal on Mental Retardation, 102*(1), 67-79.

Holahan, C. J., & Moos, R. H. (1983). The quality of social support: Measures of family and work relationships. *British Journal of Clinical Psychology, 22*(3), 157-162.

Holick, M. F. (2004). Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers, and cardiovascular disease. *The American Journal of Clinical Nutrition, 80*(6), 1678S-1688S.

Hollander, E., Phillips, A., Chaplin, W., Zagursky, K., Novotny, S., Wasserman, S., & Iyengar, R. (2005). A placebo controlled crossover trial of liquid fluoxetine on repetitive behaviors in childhood and adolescent autism. *Neuropsychopharmacology, 30*(3), 582.

Hollander, E., Soorya, L., Chaplin, W., Anagnostou, E., Taylor, B. P., Ferretti, C. J., Wasserman, S., Swanson, E., & Settipani, C. (2012). A double-blind placebo-controlled trial of fluoxetine for repetitive behaviors and global severity in adult autism spectrum disorders. *American Journal of Psychiatry, 169*(3), 292-299.

Holloway, J. M., Long, T. M., & Biasini, F. (2018). Relationships between gross motor skills and social function in young boys with autism spectrum disorder. *Pediatric Physical Therapy, 30*(3), 184-190.

Hayward, D., Eikeseth, S., Gale, C., & Morgan, S. (2009). Assessing progress during treatment for young children with autism receiving intensive behavioural interventions. *Autism, 13*(6), 613-633.

Hsiao, E. Y., McBride, S. W., Hsien, S., Sharon, G., Hyde, E. R., McCue, T., Codelli, J. A., Chow, J., Reisman, S. E., Petrosino, J. F. and Patterson, P. H. (2013). Microbiota modulate behavioral and physiological abnormalities associated with neurodevelopmental disorders. *Cell, 155*(7), 1451-1463.

Hughes, H. K., Rose, D., & Ashwood, P. (2018). The gut microbiota and dysbiosis in autism spectrum disorders. *Current Neurology and Neuroscience Reports, 18*(11), 81.

Hutton, A. M., & Caron, S. L. (2005). Experiences of families with children with autism in rural New England. *Focus on Autism and Other Developmental Disabilities, 20*(3), 180-189.

Hyman, S. L., Levy, S. E., & Myers, S. M. (2020). Identification, evaluation, and management of children with autism spectrum disorder. *Pediatrics, 145*(1), e20193447.

Ikejiri, K., Hosozawa, M., Mitomo, S., Tanaka, K., & Shimizu, T. (2016). Reduced growth during early infancy in very low birth weight children with autism spectrum disorder. *Early Human Development, 98*, 23.

Idring, S., Lundberg, M., Sturm, H., Dalman, C., Gumpert, C., Rai, D., Lee, B. K. & Magnusson, C. (2015). Changes in prevalence of autism spectrum disorders in 2001–2011: findings from the Stockholm youth cohort. *Journal of Autism and Developmental Disorders, 45*(6), 1766-1773.

Ingram, K. M., Jones, D. A., Fass, R. J., Neidig, J. L., & Song, Y. S. (1999). Social support and unsupportive social interactions: Their association with depression among people living with HIV. *AIDS Care, 11*(3), 313-329.

Ingram, K. M., Betz, N. E., Mindes, E. J., Schmitt, M. M., & Smith, N. G. (2001). Unsupportive responses from others concerning a stressful life event: Development of the Unsupportive Social Interactions Inventory. *Journal of Social and Clinical Psychology, 20*(2), 173-207.

Ingersoll, B., & Schreibman, L. (2006). Teaching reciprocal imitation skills to young children with autism using a naturalistic behavioral approach: Effects on language, pretend play, and joint attention. *Journal of Autism and Developmental Disorders, 36*(4), 487.

Iossifov, I., Ronemus, M., Levy, D., Wang, Z., Hakker, I., Rosenbaum, J., Yamrom, B., Lee, Y.H., Narzisi, G., Leotta, A., & Kendall, J. (2012). De novo gene disruptions in children on the autistic spectrum. *Neuron, 74*(2), 285-299.

Järbrink, K. (2007). The economic consequences of autistic spectrum disorder among children in a Swedish municipality. *Autism, 11*(5), 453-463.

Jarvis, P. A., & Creasey, G. L. (1991). Parental stress, coping, and attachment in families with an 18-month-old infant. *Infant Behavior and Development, 14*(4), 383-395.

Jaselskis, C. A., Cook, E. H., & Fletcher, K. E. (1992). Clonidine treatment of hyperactive and impulsive children with autistic disorder. *Journal of Clinical Psychopharmacology, 12*(5), 322-327.

Jia, F., Wang, B., Shan, L., Xu, Z., Staal, W. G., & Du, L. (2015). Core symptoms of autism improved after vitamin D supplementation. *Pediatrics, 135*(1), e196-e198.

Jesner, O. S., Aref-Adib, M., & Coren, E. (2007). Risperidone for autism spectrum disorder. *Cochrane Database of Systematic Reviews*, (1), CD005040.

Joachim B., Lyon D., & Farrell S. (2003). Augmenting treatment of Obsessive-Compulsive Disorder with Antonovsky's sense of coherence theory. *Perspectives in Psychiatric Care, 39*(4), 163–168

Jobsis, F. F. (1977). Noninvasive, infrared monitoring of cerebral and myocardial oxygen sufficiency and circulatory parameters. *Science, 198*(4323), 1264-1267.

Jones, J. B. (2018). *Family quality of life for parents of children with autism: The role of social support and unsupportive social interactions* (Doctoral thesis, University of Windsor). Electronic Theses and Dissertations. 7528.

Jones, R.A., Downing, K., Rinehart, N.J., Barnett, L.M., May, T., McGillivray, J.A., Papadopoulos, N.V., Skouteris, H., Timperio, A. & Hinkley, T., (2017). Physical activity, sedentary behavior and their correlates in children with Autism Spectrum Disorder: A systematic review. *PloS one, 12*(2), e0172482.

Jung, C. R., Lin, Y. T., & Hwang, B. F. (2013). Air pollution and newly diagnostic autism spectrum disorders: a population-based cohort study in Taiwan. *PloS One, 8*(9), e75510.

Kaat, A. J., & Lecavalier, L. (2013). Disruptive behavior disorders in children and adolescents with autism spectrum disorders: A review of the prevalence, presentation, and treatment. *Research in Autism Spectrum Disorders, 7*(12), 1579-1594.

Kalkbrenner, A. E., Daniels, J. L., Chen, J. C., Poole, C., Emch, M., & Morrissey, J. (2010). Perinatal exposure to hazardous air pollutants and autism spectrum disorders at age 8. *Epidemiology (Cambridge, Mass.), 21*(5), 631.

Kamio, Y., & Inada, N. (2014). *Early Diagnosis of ASD in Toddlers and School Children: Community Studies and National Surveys in Japan*. In Comprehensive Guide to Autism, (pp. 2561-2577).

Kamioka, H., Okada, S., Tsutani, K., Park, H., Okuizumi, H., Handa, S., Oshio, T., Park, S.J., Kitayuguchi, J., Abe, T. and Honda, T., (2014). Effectiveness of animal-assisted therapy: A systematic review of randomized controlled trials. *Complementary Therapies in Medicine, 22*(2), 371-390.

Kamio, Y., Haraguchi, H., Stickley, A., Ogino, K., Ishitobi, M., & Takahashi, H. (2015). Brief report: Best discriminators for identifying children with autism spectrum disorder at an 18-month health check-up in Japan. *Journal of Autism and Developmental Disorders, 45*(12), 4147-4153.

Kaniasty, K. Z., Norms, F. H., & Murrell, S. A. (1990). Received and Perceived Social Support Following Natural Disaster. *Journal of Applied Social Psychology, 20*(2), 85-114.

Karst, J. S., & van Hecke, A. V. (2012). Parent and family impact of autism spectrum disorders: A review and proposed model for intervention evaluation. *Clinical Child and Family Psychology Review, 15*(3), 247-277.

Kasari, C., Freeman, S., Paparella, T., Wong, C., Kwon, S., & Gulsrud, A. C. (2005). Early intervention on core deficits in autism. *Clinical Neuropsychiatry, 2*(6), 380–388.

Kawa, R., Saemundsen, E., Lóa Jónsdóttir, S., Hellendoorn, A., Lemcke, S., Canal-Bedia, R., Garcia-Primo, P., & Moilanen, I. (2016). European studies on prevalence and risk of autism spectrum disorders according to immigrant status—a review. *The European Journal of Public Health, 27*(1), 101-110.

Keen, D. V. (2008). Childhood autism, feeding problems and failure to thrive in early infancy. *European Child & Adolescent Psychiatry, 17*(4), 209-216.

Kelly, A. B., Garnett, M. S., Attwood, T., & Peterson, C. (2008). Autism spectrum symptomatology in children: The impact of family and peer relationships. *Journal of Abnormal Child Psychology*, 36(7), 1069.

Kent, J.M., Kushner, S., Ning, X., Karcher, K., Ness, S., Aman, M., Singh, J. & Hough, D. (2013). Risperidone dosing in children and adolescents with autistic disorder: a double-blind, placebo-controlled study. *Journal of Autism and Developmental Disorders, 43*(8), 1773-1783.

Kerley, C. P., Power, C., Gallagher, L., & Coghlan, D. (2017). Lack of effect of vitamin D3 supplementation in autism: a 20-week, placebo-controlled RCT. *Archives of disease in childhood*, *102*(11), 1030-1036.

Khanna, R., Madhavan, S. S., Smith, M. J., Patrick, J. H., Tworek, C., & Becker-Cottrill, B. (2011). Assessment of health-related quality of life among primary caregivers of children with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 41*(9), 1214-1227.

Kim, S. J., Young, L. J., Gonen, D., Veenstra-VanderWeele, J., Courchesne, R., Courchesne, E., Lord, C., Leventhal, B.L., Cook Jr, E.H. & Insel, T. R., (2002). Transmission disequilibrium testing of arginine vasopressin receptor 1A (AVPR1A) polymorphisms in autism. *Molecular psychiatry, 7*(5), 503.

Kim, Y. S., Leventhal, B. L., Koh, Y. J., Fombonne, E., Laska, E., Lim, E. C., Cheon, K. A., Kim, S. J., Kim, Y. K., Lee, H. & Song, D. H. (2011). Prevalence of autism spectrum disorders in a total population sample. *American Journal of Psychiatry, 168*(9), 904-912.

Kim, H., Lim, C. S., & Kaang, B. K. (2016). Neuronal mechanisms and circuits underlying repetitive behaviors in mouse models of autism spectrum disorder. *Behavioral and Brain Functions, 12*(1), 3.

Kim, I., & Dababnah, S. (2019). Appreciation of the 'Subtle Changes': Perspectives of Korean American Parents Raising Children with Developmental Disabilities. *Journal of Developmental and Physical Disabilities*, 1-16.

Kimlin, M. G. (2008). Geographic location and vitamin D synthesis. *Molecular Aspects of Medicine, 29*(6), 453-461.

Kingsbury, N., & Scanzoni, J. (1993). Structural-Functionalism. In P. G. Boss, W. J. Doherty, R. LaRossa, W. R. Schumm, & S. K. Steinmetz (Eds.), *Sourcebook of family theories and methods: A contextual approach* (pp. 195-217). New York: Plenum.

Kinney, D. K., Barch, D. H., Chayka, B., Napoleon, S., & Munir, K. M. (2010). Environmental risk factors for autism: do they help cause de novo genetic mutations that contribute to the disorder? *Medical Hypotheses, 74*(1), 102-106.

Kirby, A. V., Bagatell, N., & Baranek, G. T. (2019). The Formation of Postsecondary Expectations Among Parents of Youth With Autism Spectrum Disorder. *Focus on Autism and Other Developmental Disabilities*, 1088357619881221.

Klukowski, M., Wasilewska, J., & Lebensztejn, D. (2015). Sleep and gastrointestinal disturbances in autism spectrum disorder in children. *Developmental Period Medicine, 19*(2), 157-61.

Kočovská, E., Biskupstø, R., Gillberg, I.C., Ellefsen, A., Kampmann, H., Stórá, T., Billstedt, E. & Gillberg, C. (2012). The rising prevalence of autism: a prospective longitudinal study in the Faroe Islands. *Journal of Autism and Developmental Disorders, 42*(9), 1959-1966.

Kočovská, E., Andorsdóttir, G., Weihe, P., Halling, J., Fernell, E., Stóra, T., Biskupstø, R., Gillberg, I.C., Shea, R., Billstedt, E. and Bourgeron, T., (2014). Vitamin D in the general population of young adults with autism in the Faroe Islands. *Journal of Autism and Developmental Disorders, 44*(12), 2996-3005.

Koegel, L. K., Ashbaugh, K., & Koegel, R. L. (2016). *Pivotal response treatment*. In R. Lang, T. B. Hancock, & N. N. Singh (Eds.) *Early intervention for young children with autism spectrum disorder* (pp. 85-112). Springer, Cham.

Koegel, L. K., Park, M. N., & Koegel, R. L. (2014). Using self-management to improve the reciprocal social conversation of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, *44*(5), 1055-1063.

Koos, E. L. (1946). *Families in trouble*. New York: King's Crown Press.

Kozlowski, A. M., Matson, J. L., & Rieske, R. D. (2012). Gender effects on challenging behaviors in children with autism spectrum disorders. *Research in Autism Spectrum Disorders, 6*(2), 958-964.

Kreslins, A., Robertson, A. E., & Melville, C. (2015). The effectiveness of psychosocial interventions for anxiety in children and adolescents with autism spectrum disorder: A systematic review and meta-analysis. *Child and Adolescent Psychiatry and Mental Health, 9*(1), 22.

Kretzmann, M., Shih, W., & Kasari, C. (2015). Improving peer engagement of children with autism on the school playground: A randomized controlled trial. *Behavior Therapy, 46*(1), 20-28.

Kuhlthau, K., Payakachat, N., Delahaye, J., Hurson, J., Pyne, J. M., Kovacs, E., & Tilford, J. M. (2014). Quality of life for parents of children with autism spectrum disorders. *Research in Autism Spectrum Disorders, 8*(10), 1339-1350.

Kulms, D., & Schwarz, T. (2002). 20 years after–milestones in molecular photobiology. *Journal of Investigative Dermatology Symposium Proceedings, 7*(1), 46-50.

Kumazaki, H., Okamoto, M., Yoshimura, Y., Ikeda, T., Hasegawa, C., Saito, D.N., Iwanaga, R., Tomiyama, S., An, K.M., Minabe, Y. and Kikuchi, M., (2018). Brief Report: Odour Awareness in Young Children with Autism Spectrum Disorders. *Journal of Autism and Developmental Disorders*, 1-7.

Kuo, F. E., & Faber Taylor, A. (2004). A potential natural treatment for attention-deficit/hyperactivity disorder: evidence from a national study. *American Journal of Public Health, 94*(9), 1580-1586.

Lai, M. C., Lombardo, M. V., & Baron-Cohen, S. (2014). Autism. *Lancet, 383*(9920), 896-910.

Landa, R. J. (2018). Efficacy of early interventions for infants and young children with, and at risk for, autism spectrum disorders. *International Review of Psychiatry, 30*(1), 25-39.

Lang, R., Kuriakose, S., Lyons, G., Mulloy, A., Boutot, A., Britt, C., Caruthers, S., Ortega, L., O'Reilly, M. and Lancioni, G., 2011. Use of school recess time in the education and treatment of children with autism spectrum disorders: A systematic review. *Research in Autism Spectrum Disorders, 5*(4), 1296-1305.

Lanning, B. A., Baier, M. E. M., Ivey-Hatz, J., Krenek, N., & Tubbs, J. D. (2014). Effects of equine assisted activities on autism spectrum disorder. *Journal of Autism and Developmental Disorders, 44*(8), 1897-1907.

Lanz, M., & Maino, E. (2014). Family Environment Scale. *Encyclopedia of Quality of Life and Well-Being Research*, 2170-2173.

Larson, L.R., Barger, B., Ogletree, S., Torquati, J., Rosenberg, S., Gaither, C.J., Bartz, J.M., Gardner, A., Moody, E. & Schutte, A., 2018. Gray space and green space proximity associated with higher anxiety in youth with autism. *Health & Place, 53*, 94-102.

Larsson, M., Tirado, C., & Wiens, S. (2017). A Meta-Analysis of Odor Thresholds and Odor Identification in Autism Spectrum Disorders. *Frontiers in Psychology, 8*, 679.

Lavee, Y. (1997). Beyond ABCX: Understanding changes in marital relationships under stress. In the *Theory Construction and Research Methodology Workshop, National Council on Family Relations*, Crystal City, VA.

Lavee, Y. (2013). Stress Processes in Families and Couples. In G. Peterson & K. Bush (Eds.), *Handbook of Marriage and the Family* (pp. 159-176). New York, NY: Springer.

Lavee, Y., McCubbin, H. I., & Patterson, J. M. (1985). The double ABCX model of family stress and adaptation: An empirical test by analysis of structural equations with latent variables. *Journal of Marriage and the Family, 42*(2) 811-825.

Lavelle, T. A., Weinstein, M. C., Newhouse, J. P., Munir, K., Kuhlthau, K. A., & Prosser, L. A. (2014). Economic burden of childhood autism spectrum disorders. *Pediatrics, 133*(3), e520-e529.

Law, M. (2002). Participation in the occupations of everyday life. *American Journal of Occupational Therapy, 56*(6), 640-649.

Lazarus, R. S., & Folkman, S. (1984). *Stress, Appraisal and Coping*. New York, NY: Springer.

Leavey, A., Zwaigenbaum, L., Heavner, K., & Burstyn, I. (2013). Gestational age at birth and risk of autism spectrum disorders in Alberta, Canada. *The Journal of Pediatrics, 162*(2), 361-368.

LeBlanc, A. G., Spence, J.C., Carson, V., Connor Gorber, S., Dillman, C., Janssen, I., Kho, M. E., Stearns, J. A., Timmons, B. W. and Tremblay, M. S. (2012). Systematic review of sedentary behaviour and health indicators in the early years (aged 0–4 years). *Applied Physiology, Nutrition, and Metabolism, 37*(4), 753-772.

Ledford, J. R., Whiteside, E., & Severini, K. E. (2018). A systematic review of interventions for feeding-related behaviors for individuals with autism spectrum disorders. *Research in Autism Spectrum Disorders, 52*, 69-80.

Lee, G. K. (2009). Parents of children with high functioning autism: How well do they cope and adjust? *Journal of Developmental and Physical Disabilities, 21*(2), 93-114.

Lee, G.K., Lopata, C., Volker, M.A., Thomeer, M.L., Nida, R.E., Toomey, J.A., Chow, S.Y. and Smerbeck, A.M., (2009). Health-related quality of life of parents of children with high-functioning autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities, 24*(4), 227-239.

Lee, E. J., Choi, S. Y., & Kim, E. (2015). NMDA receptor dysfunction in autism spectrum disorders. *Current Opinion in Pharmacology, 20*, 8-13.

Leekam, S. R., Prior, M. R., & Uljarevic, M. (2011). Restricted and repetitive behaviors in autism spectrum disorders: a review of research in the last decade. *Psychological bulletin*, *137*(4), 562.

Lehti, V., Hinkka-Yli-Salomäki, S., Cheslack-Postava, K., Gissler, M., Brown, A. S., & Sourander, A. (2013). The risk of childhood autism among second-generation migrants in Finland: a case–control study. *BMC Pediatrics, 13*(1), 171.

Levy, S. E., & Hyman, S. L. (2008). Complementary and alternative medicine treatments for children with autism spectrum disorders. *Child and Adolescent Psychiatric Clinics of North America, 17*(4), 803-820.

Levy, S. E., Pinto-Martin, J. A., Bradley, C. B., Chittams, J., Johnson, S. L., Pandey, J., Pomykacz, A., Ramirez, A., Reynolds, A., Rubenstein, E. & Schieve, L. A. (2019). Relationship of weight outcomes, co-occurring conditions, and severity of autism spectrum disorder in the study to explore early development. *The Journal of Pediatrics, 205*, 202-209.

Lewis, M. H., & Bodfish, J. W. (1998). Repetitive behavior disorders in autism. *Mental Retardation and Developmental Disabilities Research Reviews, 4*(2), 80-89.

Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., Tager-Flusberg, H., & Lainhart, J. E. (2006). Comorbid psychiatric disorders in children with autism: interview development and rates of disorders. *Journal of Autism and Developmental Disorders, 36*(7), 849-861.

Li, D., Larsen, L., Yang, Y., Wang, L., Zhai, Y., & Sullivan, W. C. (2019). Exposure to nature for children with autism spectrum disorder: Benefits, caveats, and barriers. *Health & Place, 55*, 71-79.

Liao, X., Lei, X., & Li, Y. (2019). Stigma among parents of children with autism: A literature review. *Asian Journal of Psychiatry, 45*, 88-94.

Liddle, K. (2001). Implementing the picture exchange communication system (PECS). *International Journal of Language & Communication Disorders, 36*(S1), 391-395.

Linderman, T. M., & Stewart, K. B. (1999). Sensory integrative–based occupational therapy and functional outcomes in young children with pervasive developmental disorders: A single-subject study. *American Journal of Occupational Therapy, 53*(2), 207-213.

Linke, A. C., Keehn, R. J. J., Pueschel, E. B., Fishman, I., & Müller, R. A. (2018). Children with ASD show links between aberrant sound processing, social symptoms, and atypical auditory interhemispheric and thalamocortical functional connectivity. *Developmental Cognitive Neuroscience, 29*, 117-126.

Liu, X., Hubbard, J. A., Fabes, R. A., & Adam, J. B. (2006). Sleep disturbances and correlates of children with autism spectrum disorders. *Child psychiatry and Human Development, 37*(2), 179-191.

Liu, X., Malenfant, P., Reesor, C., Lee, A., Hudson, M. L., Harvard, C., Qiao, Y., Persico, A. M., Cohen, I. L., Chudley, A. E. & Forster-Gibson, C. (2011). 2p15–p16. 1 microdeletion syndrome: molecular characterization and association of the OTX1 and XPO1 genes with autism spectrum disorders. *European Journal of Human Genetics, 19*(12), 1264-1270.

Locke, J., Shih, W., Kretzmann, M., & Kasari, C. (2016). Examining playground engagement between elementary school children with and without autism spectrum disorder. *Autism, 20*(6), 653-662.

Lockley, S. W., Evans, E. E., Scheer, F. A., Brainard, G. C., Czeisler, C. A., & Aeschbach, D. (2006). Short-wavelength sensitivity for the direct effects of light on alertness, vigilance, and the waking electroencephalogram in humans. *Sleep, 29*(2), 161-168.

Loomes, R., Hull, L., & Mandy, W. P. L. (2017). What is the male-to-female ratio in autism spectrum disorder? A systematic review and meta-analysis. *Journal of the American Academy of Child & Adolescent Psychiatry, 56*(6), 466-474.

Lord, C., Elsabbagh, M., Baird, G., & Veenstra-Vanderweele, J. (2018). Autism spectrum disorder. *The Lancet, 392*(10146), 508-520.

Lotter, V. (1966). Epidemiology of autistic conditions in young children. *Social psychiatry, 1*(3), 124-135.

Louis, P. (2012). Does the human gut microbiota contribute to the etiology of autism spectrum disorders? *Digestive Diseases and Sciences, 57*(8), 1987–1989.

Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology, 55*(1), 3-9.

Lovell, B., Moss, M., & Wetherell, M. A. (2015). The psychophysiological and health corollaries of child problem behaviours in caregivers of children with autism and ADHD. *Journal of Intellectual Disability Research, 59*(2), 150-157.

Lovell, B., & Wetherell, M. A. (2016). The psychophysiological impact of childhood autism spectrum disorder on siblings. *Research in Developmental Disabilities, 49*, 226-234.

Ma, D. Q., Rabionet, R., Konidari, I., Jaworski, J., Cukier, H. N., Wright, H. H., Abramson, R. K., Gilbert, J. R., Cuccaro, M. L., PericakVance, M. A., & Martin, E. R. (2010). Association and gene-gene interaction of SLC6A4 and ITGB3 in autism. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 153*(2), 477-483.

Magnusson, C., Lundberg, M., Lee, B.K., Rai, D., Karlsson, H., Gardner, R., Kosidou, K., Arver, S. & Dalman, C. (2016). Maternal vitamin D deficiency and the risk of autism spectrum disorders: population-based study. *British Journal of Psychiatry Open, 2*(2), 170-172.

Mahan, S., & Matson, J. L. (2011). Convergent and discriminant validity of the autism spectrum disorder-problem behavior for children (ASD-PBC) against the behavioral assessment system for children, (BASC-2). *Research in Autism Spectrum Disorders, 5*(1), 222-229.

Mahoney, J. L., Cairns, B. D., & Farmer, T. W. (2003). Promoting interpersonal competence and educational success through extracurricular activity participation. *Journal of Educational Psychology, 95*(2), 409.

Mahoney, G., & Perales, F. (2005). Relationship-focused early intervention with children with pervasive developmental disorders and other disabilities: A comparative study. *Journal of Developmental & Behavioral Pediatrics, 26*(2), 77-85.

Maglione, M. A., Gans, D., Das, L., Timbie, J., & Kasari, C. (2012). Nonmedical interventions for children with ASD: Recommended guidelines and further research needs. *Pediatrics, 130*(Supplement 2), S169-S178.

Makrygianni, M. K., Gena, A., Katoudi, S., & Galanis, P. (2018). The effectiveness of applied behavior analytic interventions for children with Autism Spectrum Disorder: A meta-analytic study. *Research in Autism Spectrum Disorders, 51*, 18-31.

Malia, J. A. (2006). Basic concepts and models of family stress. *Stress, Trauma, and Crisis, 9*(3-4), 141-160.

Maltby, J., Day, L., Hall, S. S., & Chivers, S. (2017). The Measurement and Role of Ecological Resilience Systems Theory Across Domain-Specific Outcomes: The Domain-Specific Resilient Systems Scales. https://doi.org/10.1177/1073191117738045

Mangiola, F., Ianiro, G., Franceschi, F., Fagiuoli, S., Gasbarrini, G., & Gasbarrini, A. (2016). Gut microbiota in autism and mood disorders. *World Journal of Gastroenterology, 22*(1), 361.

Manning, M. M., Wainwright, L., & Bennett, J. (2011). The double ABCX model of adaptation in racially diverse families with a school-age child with autism. *Journal of Autism and Developmental Disorders, 41*(3), 320-331.

Maneeton, N., Maneeton, B., Putthisri, S., Suttajit, S., Likhitsathian, S., & Srisurapanont, M. (2018). Aripiprazole in acute treatment of children and adolescents with autism spectrum disorder: a systematic review and meta-analysis. *Neuropsychiatric Disease and Treatment, 14*, 3063.

Mannion, A., & Leader, G. (2013). Comorbidity in autism spectrum disorder: A literature review. *Research in Autism Spectrum Disorders, 7*(12), 1595-1616.

Manwaring, S. S., & Barber, A. B. (2019). *Speech-Language Pathology*. In Handbook of Interdisciplinary Treatments for Autism Spectrum Disorder. Springer, Cham. 225-257.

Marco, E. J., Hinkley, L. B., Hill, S. S., & Nagarajan, S. S. (2011). Sensory processing in autism: a review of neurophysiologic findings. *Pediatric Research, 69*, 48R-54R.

Margalit M., Leyser Y., Avraham Y., & Lewy-Osin M. (1998). Social-environmental characteristics (family climate) and sense of coherence in kibbutz families with disabled and non-disabled children. *European Journal of Special Needs Education, 3*(2), 87–98.

Margalit M., Raviv A., & Ankonina D. (1992). Coping and coherence among parents with disabled children. *Journal of Clinical Child Psychology, 21*(3), 202–209.

Marquenie, K., Rodger, S., Mangohig, K., & Cronin, A. (2011). Dinnertime and bedtime routines and rituals in families with a young child with an autism spectrum disorder. *Australian Occupational Therapy Journal, 58*(3), 145-154.

Martin, F., & Farnum, J. (2002). Animal-assisted therapy for children with pervasive developmental disorders. *Western Journal of Nursing Research, 24*(6), 657-670.

Martins, Y., Young, R. L., & Robson, D. C. (2008). Feeding and eating behaviors in children with autism and typically developing children. *Journal of Autism and Developmental Disorders, 38*(10), 1878-1887.

Masten, A. S. (2009). Ordinary Magic: Lessons from Research on Resilience in Human Development. *Education Canada, 49*(3), 28-32.

Matson, J. L., Baglio, C. S., Smiroldo, B. B., Hamilton, M., Packlowskyj, T., Williams, D., & Kirkpatrick-Sanchez, S. (1996). Characteristics of autism as assessed by the diagnostic assessment for the severely handicapped—II (DASH-II). *Research in Developmental Disabilities, 17*(2), 135-143.

Matson, J. L., Gonzalez, M. L., & Rivet, T. T. (2008). Reliability of the autism spectrum disorder-behavior problems for children (ASD-BPC). *Research in Autism Spectrum Disorders, 2*(4), 696-706.

Matson, M. L., Matson, J. L., & Beighley, J. S. (2011). Comorbidity of physical and motor problems in children with autism. *Research in Developmental Disabilities, 32*(6), 2304-2308.

Matthews, R. A., Booth, S. M., Taylor, C. F., & Martin, T. (2011). A qualitative examination of the work–family interface: Parents of children with autism spectrum disorder. *Journal of Vocational Behavior, 79*(3), 625-639.

May, T., & Williams, K. (2018). Brief report: gender and age of diagnosis time trends in children with autism using Australian medicare data. *Journal of Autism and Developmental Disorders, 48*(12), 4056-4062.

Mayer, E. A., Padua, D., & Tillisch, K. (2014). Altered brain-gut axis in autism: Comorbidity or causative mechanisms? *Bioessays, 36*(10), 933-939.

Mayes, S. D., & Zickgraf, H. (2019). Atypical eating behaviors in children and adolescents with autism, ADHD, other disorders, and typical development. *Research in Autism Spectrum Disorders, 64*, 76-83.

Mazahery, H., Camargo, C., Conlon, C., Beck, K., Kruger, M., & von Hurst, P. (2016). Vitamin D and autism spectrum disorder: a literature review. *Nutrients, 8*(4), 236.

Mazurek, M. O., & Engelhardt, C. R. (2013). Video game use in boys with autism spectrum disorder, ADHD, or typical development. *Pediatrics, 132*(2), 260-266.

Mazurek, M.O., Vasa, R.A., Kalb, L.G., Kanne, S.M., Rosenberg, D., Keefer, A., Murray, D.S., Freedman, B. & Lowery, L.A., (2013). Anxiety, sensory over-responsivity, and gastrointestinal problems in children with autism spectrum disorders. *Journal of Abnormal Child Psychology, 41*(1), 165-176.

Mazurek, M. O., Kanne, S. M., & Wodka, E. L. (2013). Physical aggression in children and adolescents with autism spectrum disorders. *Research in Autism Spectrum Disorders, 7*(3), 455-465.

Mazurek, M. O., Lu, F., Symecko, H., Butter, E., Bing, N. M., Hundley, R. J., Poulsen, M., Kanne, S. M., Macklin, E. A. & Handen, B. L. (2017). A prospective study of the concordance of DSM-IV and DSM-5 diagnostic criteria for autism spectrum disorder. *Journal of Autism and Developmental Disorders, 47*(9), 2783-2794.

Mazurek, M. O., Carlson, C., Baker-Ericzén, M., Butter, E., Norris, M., & Kanne, S. (2018). Construct validity of the autism impact measure (AIM). *Journal of Autism and Developmental Disorders, 17*, 1-13.

McAuliffe, T., Vaz, S., Falkmer, T., & Cordier, R. (2017). A comparison of families of children with autism spectrum disorders in family daily routines, service usage, and stress levels by regionality. *Developmental Neurorehabilitation, 20*(8), 483-490.

McCann, D., Bull, R., & Winzenberg, T. (2012). The daily patterns of time use for parents of children with complex needs: A systematic review. *Journal of Child Health Care, 16*(1), 26-52.

McConachie, H., McLaughlin, E., Grahame, V., Taylor, H., Honey, E., Tavernor, L., Rodgers, J., Freeston, M., Hemm, C., Steen, N. & Le Couteur, A. (2014). Group therapy for anxiety in children with autism spectrum disorder. *Autism, 18*(6), 723-732.

McCoy, S. M., Jakicic, J. M., & Gibbs, B. B. (2016). Comparison of obesity, physical activity, and sedentary behaviors between adolescents with autism spectrum disorders and without. *Journal of Autism and Developmental Disorders, 46*(7), 2317-2326.

McCracken, J. T., Badashova, K. K., Posey, D. J., Aman, M. G., Scahill, L., Tierney, E., Arnold, L. E., Vitiello, B., Whelan, F., Chuang, S. Z., & Davies, M. (2014). Positive effects of methylphenidate on hyperactivity are moderated by monoaminergic gene variants in children with autism spectrum disorders. *The Pharmacogenomics Journal, 14*(3), 295-302.

McCubbin, H., Olson, D., & Larsen, A. (1981). Family Crisis Oriented Personal Evaluation Scales (F-COPES). In H. I. McCubbin, A. I. Thompson, & M. A. McCubbin (Eds.), *Family Assessment: Resiliency, coping and adaptation inventories for research and practice* (pp. 455–507). Madison: University of Wisconsin.

McCubbin, H. L., & Patterson, J. M. (1982). Family adaptation to crises. In H. L. McCubbin, E. Cauble, & J. M. Patterson (Eds.), *Family stress, coping, and social support* (pp. 26-47). Springfield, IL: Charles C. Thomas.

McCubbin, H. I., & Sussman, M. B. (1983). *Social stress and the family: Advances and developments in family stress theory and research*. New York: Haworth Press.

McCubbin H. I. & Patterson J. M. (1983). The family stress process: The Double ABCX Model of adjustment and adaptation. In H. I. McCubbin, M. B. Sussman & J. M. Patterson (Ed.), *Social Stress and the Family: Advances and Developments in Family Stress Theory and Research* (pp. 7-37). New York, NY: Haworth Press.

McInnis, O. A., McQuaid, R. J., Matheson, K., & Anisman, H. (2015). The moderating role of an oxytocin receptor gene polymorphism in the relation between unsupportive social interactions and coping profiles: implications for depression. *Frontiers in Psychology, 6*, 1133.

McInnis, O. A., McQuaid, R. J., Matheson, K., & Anisman, H. (2017). Unsupportive social interactions and affective states: examining associations of two oxytocin-related polymorphisms. *Stress, 20*(1), 122-129.

McKinney, C., & Renk, K. (2011). Atypical antipsychotic medications in the management of disruptive behaviors in children: safety guidelines and recommendations. *Clinical Psychology Review, 31*(3), 465-471.

McPheeters, M. L., Warren, Z., Sathe, N., Bruzek, J. L., Krishnaswami, S., Jerome, R. N., & Veenstra-VanderWeele, J. (2011). A systematic review of medical treatments for children with autism spectrum disorders. *Pediatrics, 127*(5), e1312-e1321.

McStay, R. L., Trembath, D., & Dissanayake, C. (2014). Stress and family quality of life in parents of children with autism spectrum disorder: parent gender and the double ABCX model. *Journal of Autism and Developmental Disorders, 44*(12), 3101-3118.

Menaghan, E. G. (1983). Individual coping efforts and family studies: Conceptual and methodological issues. *Marriage & Family Review, 6*(1-2), 113-135.

Menear, K. S., Smith, S. C., & Lanier, S. (2006). A multipurpose fitness playground for individuals with autism: Ideas for design and use. *Journal of Physical Education, Recreation & Dance, 77*(9), 20-25.

Meny, H. A., Hayat, A. A., & Wright, J. J. (2018). Mothers of autistic children: Is the burden of care, extended time spend and money constraints, effecting their social participation? *International Annals of Medicine, 2*(12), 1-6.

Mercer, R. T. (2004). Becoming a mother versus maternal role attainment. *Journal of Nursing Scholarship, 36*(3), 226-232.

Miano S., Giannotti F., & Cortesi F. (2016). Sleep Disorders and Autism Spectrum Disorder. In L. Mazzone, B. Vitiello B. (Ed.), *Psychiatric Symptoms and Comorbidities in Autism Spectrum Disorder*. (pp. 111–128.) Springer, Cham

Minshawi, N. F., Hurwitz, S., Morriss, D., & McDougle, C. J. (2015). Multidisciplinary assessment and treatment of self-injurious behavior in autism spectrum disorder and intellectual disability: integration of psychological and biological theory and approach. *Journal of Autism and Developmental Disorders, 45*(6), 1541-1568.

Moore, K.A., Russell, K., & Bouchoucha, S.L. (2017). Coping and resilience: cut from the same cloth? In K. A. Moore & P. Buchwald (eds.) *Stress, Coping and Resilience*. Berlin: Logos Publishers.

Moos, R. H., Insel, P. M., & Humphrey, B. (1974). *Preliminary manual for Family Environment Scale, Work Environment Scale, Group Environment Scale*. Palo Alto, CA: Consulting Psychologists Press.

Moos, R. H. (1990). Conceptual and empirical approaches to developing family-based assessment procedures: Resolving the case of the Family Environment Scale. *Family Process, 29*(2), 199-208.

Moos, R. H., & Moos, B. S. (1981). *Family Environment Scale manual*. Palo Alto, CA: Consulting Psychologists Press.

Mössler, K., Gold, C., Aßmus, J., Schumacher, K., Calvet, C., Reimer, S., Iversen, G., & Schmid, W. (2019). The therapeutic relationship as predictor of change in music therapy with young children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 49(*7), 2795-2809.

Morgan, S. B. (1988). The autistic child and family functioning: A developmental-family systems perspective. *Journal of Autism and Developmental Disorders, 18*(2), 263-280.

Mouridsen, S. E., Rich, B., & Isager, T. (2008). Body mass index in male and female children with pervasive developmental disorders. *Pediatrics International, 50*(4), 569-571.

Must, A., Phillips, S. M., Curtin, C., Anderson, S. E., Maslin, M., Lividini, K., & Bandini, L. G. (2014). Comparison of sedentary behaviors between children with autism spectrum disorders and typically developing children. *Autism, 18*(4), 376-384.

Myers, B. J., Mackintosh, V. H., & Goin-Kochel, R. P. (2009). "My greatest joy and my greatest heart ache:" Parents' own words on how having a child in the autism spectrum has affected their lives and their families' lives. *Research in Autism Spectrum Disorders, 3*(3), 670-684.

Nabors, L., Cunningham, J. F., Lang, M., Wood, K., Southwick, S., & Stough, C. O. (2018). Family Coping During Hospitalization of Children with Chronic Illnesses. *Journal of Child and Family Studies, 27*(5), 1482-1491.

National Research Council. (2001). *Educating children with autism*. In Committee on Educational Interventions for Children with Autism, C. Lord, & J. P. McGee (Eds.), Division of behavioral and social sciences and education. Washington, DC: National Academy Press.

National Statistics (2016). Summary Statistics for Schools in Scotland, No. 7: 2016 Edition, Available:http://www.gov.scot/Publications/2016/12/9271/downloads#res511474.

Nguyen, C. T., Krakowiak, P., Hansen, R., Hertz-Picciotto, I., & Angkustsiri, K. (2016). Sociodemographic disparities in intervention service utilization in families of children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 46*(12), 3729-3738.

NHS (2017). Web Resource on Autism Spectrum Disorders (ASDs) For GeneralPractitioners and Primary Care Practitioners. Available:http://asd.nes.scot.nhs.uk/1144.

Nicholson, H., Kehle, T. J., Bray, M. A., & Heest, J. V. (2011). The effects of antecedent physical activity on the academic engagement of children with autism spectrum disorder. *Psychology in the Schools, 48*(2), 198-213.

Nickels, K. C., Katusic, S. K., Colligan, R. C., Weaver, A. L., Voigt, R. G., & Barbaresi, W. J. (2008). Stimulant medication treatment of target behaviors in children with autism: a population-based study. *Journal of Developmental and Behavioral Pediatrics, 29*(2), 75-81.

Norris, F. H., & Kaniasty, K. (1996). Received and perceived social support in times of stress: a test of the social support deterioration deterrence model. *Journal of Personality and Social Psychology, 71*(3), 498.

O'leary, L., Henderson, A., Jacobs, M., & Cooper, S. A. (2016). Children with intellectual disabilities and children with autism in Scottish schools. *Journal of Intellectual Disability Research, 60*(7-8), 717.

O'Neill, C., Kazantzidis, A., Ryan, M., Barber, N., Sempos, C., Durazo-Arvizu, R., Jorde, R., Grimnes, G., Eiriksdottir, G., Gudnason, V. & Cotch, M. (2016). Seasonal changes in vitamin D-effective UVB availability in Europe and associations with population serum 25-hydroxyvitamin D. *Nutrients, 8*(9), 533.

Obrusnikova, I., & Cavalier, A. (2011). Perceived barriers and facilitators of participation in after school physical activity by children with autism spectrum disorders. *Journal of Developmental and Physical Disabilities, 23*, 195–211.

Oelofsen, N., & Richardson, P. (2006). Sense of coherence and parenting stress in mothers and fathers of preschool children with developmental disability. *Journal of Intellectual and Developmental Disability, 31*(1), 1-12.

Office of the United Nations High Commissioner for Human Rights. (1990). *Convention on the Rights of the Child.* Convention on the rights of the child: General Assembly Resolution 44/25 of 20 November 1989.

Øien, R., & Eisemann, M. R. (2016). Brief report: Parent-reported problems related to communication, behavior and interests in children with autistic disorder and their impact on quality of life. *Journal of Autism and Developmental Disorders, 46*(1), 328-331.

Olson, D. H., Sprenkle, D. H., & Russell, C. S. (1979). Circumplex model of marital and family systems: I. Cohesion and adaptability dimensions, family types, and clinical applications. *Family Process, 18*(1), 3-28.

Olson, D. H. (2000). Circumplex model of marital and family systems. *Journal of Family Therapy, 22*(2), 144-167.

Olsson, M. B., & Hawang, C. P. (2002). Sense of coherence in parents of children with different developmental disabilities. *Journal of Intellectual Disability Research, 46*, 548–559.

Ooi, K. L., Ong, Y. S., Jacob, S. A., & Khan, T. M. (2016). A meta-synthesis on parenting a child with autism. *Neuropsychiatric Disease and Treatment, 12*, 745.

Oono, I. P., Honey, E. J., & McConachie, H. (2013). Parent-mediated early intervention for young children with autism spectrum disorders (ASD). *Evidence-Based Child Health: A Cochrane Review Journal, 8*(6), 2380-2479.

Oriel, K. N., George, C. L., Peckus, R., & Semon, A. (2011). The effects of aerobic exercise on academic engagement in young children with autism spectrum disorder. *Pediatric Physical Therapy, 23*(2), 187-193.

Owen, R., Sikich, L., Marcus, R. N., Corey-Lisle, P., Manos, G., McQuade, R. D., Carson, W. H. & Findling, R. L., (2009). Aripiprazole in the treatment of irritability in children and adolescents with autistic disorder. *Pediatrics, 124*(6), 1533-1540.

Ozonoff, S., Young, G.S., Carter, A., Messinger, D., Yirmiya, N., Zwaigenbaum, L., Bryson, S., Carver, L.J., Constantino, J.N., Dobkins, K. & Hutman, T., (2011). Recurrence risk for autism spectrum disorders: A Baby Siblings Research Consortium study. *Pediatrics, 128*(3), e488-e495.

Padden, C., & James, J. E. (2017). Stress among parents of children with and without autism spectrum disorder: a comparison involving physiological indicators and parent self-reports. *Journal of Developmental and Physical disabilities, 29*(4), 567-586.

Pakenham, K. I., Samios, C., & Sofronoff, K. (2005). Adjustment in mothers of children with asperger syndrome: An application of the double ABCX model of family adjustment. *Autism, 9*(2), 191–212.

Papp, L. M., & Hartley, S. L. (2019). Child-present and child-themed marital conflict in daily life of parents of children with and without autism spectrum disorder. *Developmental Psychology, 55*(1), 148.

Patten, S. B., Waheed, W., & Bresee, L. (2012). A review of pharmacoepidemiologic studies of antipsychotic use in children and adolescents. *The Canadian Journal of Psychiatry, 57*(12), 717-721.

Patterson, J. M. (1988). Families experiencing stress: I. The Family Adjustment and Adaptation Response Model: II. Applying the FAAR Model to health-related issues for intervention and research. *Family Systems Medicine, 6*(2), 202.

Patterson, J. M. (2002). Integrating family resilience and family stress theory. *Journal of Marriage and Family, 64*(2), 349-360.

Patterson, J. M., & Mccubbin, H. I. (1983). The impact of family life events and changes on the health of a chronically ill child. *Family Relations, 32*, 255-264.

Parisi, A. V., Turnbull, D. J., & Turner, J. (2007). Influence of clouds on pre-vitamin D3 effective solar UV exposures. *Environmental Health, 7*(3), 75.

Paul, R., Campbell, D., Gilbert, K., & Tsiouri, I. (2013). Comparing spoken language treatments for minimally verbal preschoolers with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 43*(2), 418–431.

Paula, C. S., Ribeiro, S. H., Fombonne, E., & Mercadante, M. T. (2011). Brief report: prevalence of pervasive developmental disorder in Brazil: a pilot study. *Journal of Autism and Developmental Disorders, 41*(12), 1738-1742.

Payne, T. J., Andrew, M., Butler, K. R., Wyatt, S. B., Dubbert, P. M., & Mosley, T. H. (2012). Psychometric evaluation of the Interpersonal Support Evaluation List–Short Form in the ARIC study cohort. *Sage Open, 2*(3), 2158244012461923.

Paynter, J., Riley, E., Beamish, W., Davies, M., & Milford, T. (2013). The double ABCX model of family adaptation in families of a child with an autism spectrum disorder attending an Australian early intervention service. *Research in Autism Spectrum Disorders, 7*(10), 1183-1195.

Paynter, J., Davies, M., & Beamish, W. (2018). Recognising the "forgotten man": Fathers' experiences in caring for a young child with autism spectrum disorder. Journal of Intellectual & Developmental Disability, 43(1), 112-124.

Pearlin, L. I., & Schooler, C. (1978). The structure of coping. *Journal of Health and Social Behavior, 19*, 2-21.

Penagarikano, O., & Geschwind, D. (2013). *CNTNAP2 and autism spectrum disorders*. In: The autisms (Powel CM, ed), pp 274-285. Oxford University Press: New York, NY.

Pepperell, T. A., Paynter, J., & Gilmore, L. (2016). Social support and coping strategies of parents raising a child with autism spectrum disorder. *Early Child Development and Care, 188*(10), 1392-1404.

Peters-Scheffer, N., Didden, R., Korzilius, H., & Sturmey, P. (2011). A meta-analytic study on the effectiveness of comprehensive ABA-based early intervention programs for children with autism spectrum disorders. *Research in Autism Spectrum Disorders, 5*(1), 60-69.

Petrus, C., Adamson, S. R., Block, L., Einarson, S. J., Sharifnejad, M., & Harris, S. R. (2008). Effects of exercise interventions on stereotypic behaviours in children with autism spectrum disorder. *Physiotherapy Canada, 60*(2), 134-145.

Pfeiffer, B. A., Koenig, K., Kinnealey, M., Sheppard, M., & Henderson, L. (2011). Effectiveness of sensory integration interventions in children with autism spectrum disorders: A pilot study. *American Journal of Occupational Therapy, 65*(1), 76-85.

Pickard, K. E., & Ingersoll, B. R. (2017). Using the double ABCX model to integrate services for families of children with ASD. *Journal of Child and Family Studies, 26*(3), 810-823.

Pickles, A., Le Couteur, A., Leadbitter, K., Salomone, E., Cole-Fletcher, R., Tobin, H., Gammer, I., Lowry, J., Vamvakas, G., Byford, S. & Aldred, C., (2016). Parent-mediated social communication therapy for young children with autism (PACT): long-term follow-up of a randomised controlled trial. *The Lancet, 388*(10059), 2501-2509.

Pilisuk, M., & Parks, S. H. (1981). The place of network analysis in the study of supportive social associations. *Basic and Applied Social Psychology, 2*(2), 121-135.

Pinto, D., Pagnamenta, A.T., Klei, L., Anney, R., Merico, D., Regan, R., Conroy, J., Magalhaes, T.R., Correia, C., Abrahams, B.S. & Almeida, J. (2010). Functional impact of global rare copy number variation in autism spectrum disorders. *Nature, 466*(7304), 368.

Pisula, E., & Kossakowska, Z. (2010). Sense of coherence and coping with stress among mothers and fathers of children with autism. *Journal of Autism and Developmental Disorders, 40*(12), 1485-1494.

Politte, L. C., & McDougle, C. J. (2014). Atypical antipsychotics in the treatment of children and adolescents with pervasive developmental disorders. *Psychopharmacology, 231*(6), 1023-1036.

Polo-Kantola, P., Lampi, K. M., Hinkka-Yli-Salomäki, S., Gissler, M., Brown, A. S., & Sourander, A. (2014). Obstetric risk factors and autism spectrum disorders in Finland. *The Journal of Pediatrics, 164*(2), 358-365.

Poovathinal, S. A., Anitha, A., Thomas, R., Kaniamattam, M., Melempatt, N., Anilkumar, A., & Meena, M. (2016). Prevalence of autism spectrum disorders in a semiurban community in south India. *Annals of Epidemiology, 26*(9), 663-665.

Posey, D. J., Stigler, K. A., Erickson, C. A., & McDougle, C. J. (2008). Antipsychotics in the treatment of autism. *The Journal of Clinical Investigation, 118*(1), 6-14.

Pottie, C. G., Cohen, J., & Ingram, K. M. (2009). Parenting a child with autism: Contextual factors associated with enhanced daily parental mood. *Journal of Pediatric Psychology, 34*(4), 419-429.

Pozo, P., Sarriá, E., & Brioso, A. (2014). Family quality of life and psychological wellbeing in parents of children with autism spectrum disorders: A double ABCX model. *Journal of Intellectual Disability Research, 58*(5), 442–458.

Quittner, A. L., Glueckauf, R. L., & Jackson, D. N. (1990). Chronic parenting stress: Moderating versus mediating effects of social support. *Journal of Personality and Social Psychology, 59*(6), 1266.

Raina, S. K., Kashyap, V., Bhardwaj, A. K., Kumar, D., & Chander, V. (2015). Prevalence of autism spectrum disorders among children (1-10 years of age)–Findings of a mid-term report from Northwest India. *Journal of Postgraduate Medicine, 61*(4), 243.

Ramisch, J. L., Timm, T. M., Hock, R. M., & Topor, J. A. (2013). Experiences delivering a marital intervention for couples with children with autism spectrum disorder. *The American Journal of Family Therapy, 41*(5), 376-388.

Rao, P. A., & Beidel, D. C. (2009). The impact of children with high-functioning autism on parental stress, sibling adjustment, and family functioning. *Behavior Modification, 33*(4), 437-451.

Rattaz, C., Michelon, C., Munir, K., & Baghdadli, A. (2018). Challenging behaviours at early adulthood in autism spectrum disorders: topography, risk factors and evolution. *Journal of Intellectual Disability Research, 62*(7), 637-649.

Raz, R., Weisskopf, M. G., Davidovitch, M., Pinto, O., & Levine, H. (2015). Differences in autism spectrum disorders incidence by sub-populations in Israel 1992–2009: a total population study. *Journal of Autism and Developmental Disorders, 45*(4), 1062-1069.

Reichow, B., Barton, E. E., Boyd, B. A., & Hume, K. (2012). Early intensive behavioral intervention (EIBI) for young children with autism spectrum disorders (ASD). *Cochrane Database of Systematic Reviews*, (10). Rev 4:CD009774.25.

Reynolds, K. C., Patriquin, M., Alfano, C. A., Loveland, K. A., & Pearson, D. A. (2017). Parent-reported problematic sleep behaviors in children with comorbid autism spectrum disorder and attention-deficit/hyperactivity disorder. *Research in Autism Spectrum Disorders, 39*, 20-32.

Richdale, A. L., & Schreck, K. A. (2009). Sleep problems in autism spectrum disorders: prevalence, nature, & possible biopsychosocial aetiologies. *Sleep Medicine Reviews, 13*(6), 403-411.

Richdale, A. L., & Schreck, K. A. (2019). Examining sleep hygiene factors and sleep in young children with and without autism spectrum disorder. *Research in Autism Spectrum Disorders, 57*, 154-162.

Rijlaarsdam, J., van IJzendoorn, M. H., Verhulst, F. C., Jaddoe, V. W., Felix, J. F., Tiemeier, H., & Bakermans-Kranenburg, M. J. (2017). Prenatal stress exposure, oxytocin receptor gene (OXTR) methylation, and child autistic traits: the moderating role of OXTR rs53576 genotype. *Autism Research, 10*(3), 430-438.

Roach, M. A., Orsmond, G. I., & Barratt, M. S. (1999). Mothers and fathers of children with Down syndrome: Parental stress and involvement in childcare. *American Journal on Mental Retardation, 104*(5), 422-436.

Roberts, M. Y., & Kaiser, A. P. (2011). The effectiveness of parent-implemented language interventions: A meta-analysis. *American Journal of Speech-Language Pathology, 20*(3), 180-199.

Robinson, S., Weiss, J. A., Lunsky, Y., & Ouellette-Kuntz, H. (2016). Informal support and burden among parents of adults with intellectual and/or developmental disabilities. *Journal of Applied Research in Intellectual Disabilities, 29*(4), 356-365.

Roche, L., Sigafoos, J., Lancioni, G.E., O'Reilly, M.F., Schlosser, R.W., Stevens, M., van der Meer, L., Achmadi, D., Kagohara, D., James, R. & Carnett, A., (2014). An evaluation of speech production in two boys with neurodevelopmental disorders who received communication intervention with a speech-generating device. *International Journal of Developmental Neuroscience, 38*, 10-16.

Rogers, S. J., & Dawson, G. (2010). *Early Start Denver Model for young children with autism: Promoting language, learning, and engagement*. Guilford Press: New York.

Rogers, S. J., & Ozonoff, S. (2005). Annotation: What do we know about sensory dysfunction in autism? A critical review of the empirical evidence. *Journal of Child Psychology and Psychiatry, 46*(12), 1255-1268.

Rogers, S. J., & Vismara, L. A. (2008). Evidence-based comprehensive treatments for early autism. *Journal of Clinical Child & Adolescent Psychology, 37*(1), 8-38.

Rogge, N., & Janssen, J. (2019). The economic costs of autism spectrum disorder: a literature review. *Journal of Autism and Developmental Disorders, 49*(7), 2873-2900.

Roggman, L. A., Moe, S. T., Hart, A. D., & Forthun, L. F. (1994). Family leisure and social support: Relations with parenting stress and psychological well-being in Head Start parents. *Early Childhood Research Quarterly, 9*(3-4), 463-480.

Rosenfeld, C. S. (2015). Microbiome disturbances and autism spectrum disorders. *Drug Metabolism and Disposition, 43*(10), 1557-1571.

Rosero-Bixby, L. (2004). Spatial access to health care in Costa Rica and its equity: a GIS-based study. *Social Science & Medicine, 58*(7), 1271-1284.

Rossignol, D. A. (2009). Novel and emerging treatments for autism spectrum disorders: A systematic review. *Annals of Clinical Psychiatry, 21*(4), 213–236.

Rossignol, D. A., & Frye, R. E. (2011). Melatonin in autism spectrum disorders: a systematic review and meta-analysis. *Developmental Medicine & Child Neurology, 53*(9), 783-792.

Rybak, Y. E., McNeely, H. E., Mackenzie, B. E., Jain, U. R., & Levitan, R. D. (2006). An open trial of light therapy in adult attention-deficit/hyperactivity disorder. *Journal of Clinical Psychiatry, 67*(10), 1527-1535.

Rubin, R. (1967). Attainment of the maternal role: Part I. Processes. *Nursing Research, 16*(3), 237-245.

Rudie, J. D., Hernandez, L. M., Brown, J. A., Beck-Pancer, D., Colich, N. L., Gorrindo, P., Thompson, P. M., Geschwind, D. H., Bookheimer, S. Y., Levitt P., & Dapretto, M. (2012). Autism associated promoter variant in MET impacts functional and structural brain networks. *Neuron, 75*, 904-915.

Saemundsen, E., Magnússon, P., Georgsdóttir, I., Egilsson, E., & Rafnsson, V. (2013). Prevalence of autism spectrum disorders in an Icelandic birth cohort. *BMJ Open, 3*(6), e002748.

Saini, M., Stoddart, K.P., Gibson, M., Morris, R., Barrett, D., Muskat, B., Nicholas, D., Rampton, G. & Zwaigenbaum, L. (2015). Couple relationships among parents of children and adolescents with autism spectrum disorder: Findings from a scoping review of the literature. *Research in Autism Spectrum Disorders, 17*, 142-157.

Salazar, F., Baird, G., Chandler, S., Tseng, E., O'sullivan, T., Howlin, P., Pickles, A. & Simonoff, E., (2015). Co-occurring psychiatric disorders in preschool and elementary school-aged children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 45*(8), 2283-2294.

Salomone, E., Leadbitter, K., Aldred, C., Barrett, B., Byford, S., Charman, T., Howlin, P., Green, J., Le Couteur, A., McConachie, H. & Parr, J. R. (2018). The association between child and family characteristics and the mental health and wellbeing of caregivers of children with autism in mid-childhood. *Journal of Autism and Developmental Disorders, 48*(4), 1189-1198.

Saloviita, T., Itälinna, M., & Leinonen, E. (2003). Explaining the parental stress of fathers and mothers caring for a child with intellectual disability: A double ABCX model. *Journal of Intellectual Disability Research, 47*, 300–312.

Sampson, T. R., & Mazmanian, S. K. (2015). Control of brain development, function, and behavior by the microbiome. *Cell Host & Microbe, 17*(5), 565-576.

Sanders, S.J., He, X., Willsey, A.J., Ercan-Sencicek, A.G., Samocha, K.E., Cicek, A.E., Murtha, M.T., Bal, V.H., Bishop, S.L., Dong, S., & Goldberg, A.P. (2015). Insights into autism spectrum disorder genomic architecture and biology from 71 risk loci. *Neuron, 87*(6), 1215-1233.

Sandin, S., Lichtenstein, P., Kuja-Halkola, R., Larsson, H., Hultman, C. M., & Reichenberg, A. (2014). The familial risk of autism. *JAMA, 311*(17), 1770-1777.

Savedoff, W. D. (2009). A moving target: universal access to healthcare services in Latin America and the Caribbean (No. 667). Working paper//Inter-American Development Bank, Research Department.

Scahill, L., & Boorin, S. G. (2011). Psychopharmacology in children with PDD: review of current evidence. In *Evidence-based practices and treatments for children with autism* (pp. 231-243). Springer, Boston, MA.

Schauder, K. B., & Bennetto, L. (2016). Toward an interdisciplinary understanding of sensory dysfunction in autism spectrum disorder: an integration of the neural and symptom literatures. *Frontiers in Neuroscience, 10*, 268.

Schelly, D., González, P. J., & Solís, P. J. (2015). The diffusion of autism spectrum disorder in Costa Rica: Evidence of information spread or environmental effects? *Health & Place, 35*, 119-127.

Schendel, D., & Bhasin, T. K. (2008). Birth weight and gestational age characteristics of children with autism, including a comparison with other developmental disabilities. *Pediatrics, 121*(6), 1155-1164.

Schlosser, R. W., & Koul, R. K. (2015). Speech output technologies in interventions for individuals with autism spectrum disorders: A scoping review. *Augmentative and Alternative Communication, 31*(4), 285-309.

Schreck, K. A., Williams, K., & Smith, A. F. (2004). A comparison of eating behaviors between children with and without autism. *Journal of Autism and Developmental Disorders, 34*(4), 433-438.

Schreibman, L., Dawson, G., Stahmer, A.C., Landa, R., Rogers, S.J., McGee, G.G., Kasari, C., Ingersoll, B., Kaiser, A.P., Bruinsma, Y. & McNerney, E., (2015). Naturalistic developmental behavioral interventions: Empirically validated treatments for autism spectrum disorder. *Journal of Autism and Developmental Disorders, 45*(8), 2411-2428.

Sealy, J., & Glovinsky, I. P. (2016). Strengthening the reflective functioning capacities of parents who have a child with a neurodevelopmental disability through a brief, relationship-focused intervention. *Infant Mental Health Journal, 37*(2), 115-124.

Searing, B. M. J., Graham, F., & Grainger, R. (2015). Support needs of families living with children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 45*(11), 3693-3702.

Seckmeyer, G., Schrempf, M., Wieczorek, A., Riechelmann, S., Graw, K., Seckmeyer, S., & Zankl, M. (2013). A novel method to calculate solar UV exposure relevant to vitamin D production in humans. *Photochemistry and Photobiology, 89*(4), 974-983.

Sedgewick, F., Leppanen, J., & Tchanturia, K. (2019). Autistic adult outcomes on weight and body mass index: A large-scale online study. *Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity*, 1-7.

Sedgwick, A., & Stothard, J. (2018). A systematic review of school-based, mainstream, oral language interventions for key stage 1 children. *Support for Learning, 33*(4), 360-387.

Seymour, M., Wood, C., Giallo, R., & Jellett, R. (2013). Fatigue, stress and coping in mothers of children with an autism spectrum disorder. *Journal of Autism and Developmental Disorders, 43*(7), 1547-1554.

Shafiq, S., & Pringsheim, T. (2018). Using antipsychotics for behavioral problems in children. *Expert Opinion on Pharmacotherapy, 19*(13), 1475-1488.

Shane, H. C., & Albert, P. D. (2008). Electronic screen media for persons with autism spectrum disorders: Results of a survey. *Journal of Autism and Developmental Disorders, 38*(8), 1499-1508.

Sharma, S. R., Gonda, X., & Tarazi, F. I. (2018). Autism spectrum disorder: classification, diagnosis and therapy. *Pharmacology & Therapeutics, 190*, 91-104.

Sharp, W.G., Berry, R.C., McCracken, C., Nuhu, N.N., Marvel, E., Saulnier, C.A., Klin, A., Jones, W. & Jaquess, D.L., (2013). Feeding problems and nutrient intake in children with autism spectrum disorders: a meta-analysis and comprehensive review of the literature. *Journal of Autism and Developmental Disorders, 43*(9), 2159-2173.

Shepherd, D., Landon, J., & Goedeke, S. (2018). Symptom severity, caregiver stress and intervention helpfulness assessed using ratings from parents caring for a child with autism. *Autism, 22*(5), 585-596.

Shepherd, D., Landon, J., Taylor, S., & Goedeke, S. (2018). Coping and care-related stress in parents of a child with autism spectrum disorder. *Anxiety, Stress, & Coping, 31*(3), 277-290.

Sherer, M. R., & Schreibman, L. (2005). Individual behavioral profiles and predictors of treatment effectiveness for children with autism. *Journal of Consulting and Clinical Psychology, 73*(3), 525–538.

Siddiqui, A., & Hägglöf, B. (2000). Does maternal prenatal attachment predict postnatal mother–infant interaction? *Early Human Development, 59*(1), 13-25.

Silbaugh, B. C., Penrod, B., Whelan, C. M., Hernandez, D. A., Wingate, H. V., Falcomata, T. S., & Lang, R. (2016). A systematic synthesis of behavioral interventions for food selectivity of children with autism spectrum disorders. *Review Journal of Autism and Developmental Disorders, 3*(4), 345-357.

Silva, K., Correia, R., Lima, M., Magalhães, A., & de Sousa, L. (2011). Can dogs prime autistic children for therapy? Evidence from a single case study. *The Journal of Alternative and Complementary Medicine, 17*(7), 655-659.

Silva, L. M., Schalock, M., Gabrielsen, K. R., Budden, S. S., Buenrostro, M., & Horton, G. (2015). Early intervention with a parent-delivered massage protocol directed at tactile abnormalities decreases severity of autism and improves child-to-parent interactions: a replication study. *Autism Research and Treatment,* 2015.

Sim, A., Cordier, R., Vaz, S., & Falkmer, T. (2016). Relationship satisfaction in couples raising a child with autism spectrum disorder: A systematic review of the literature. *Research in Autism Spectrum Disorders, 31*, 30-52.

Siman-Tov, A., & Kaniel, S. (2011). Stress and personal resource as predictors of the adjustment of parents to autistic children: A multivariate model. *Journal of Autism and Developmental Disorders, 41*(7), 879-890.

Singer, A., & Ravi, R. (2015). Complementary and alternative treatments for autism part 2: identifying and avoiding non-evidence-based treatments. *AMA journal of ethics, 17*(4), 375-380.

Sitimin, S. A., Fikry, A., Ismail, Z., & Hussein, N. (2017). Work-family Conflict among Working Parents of Children with Autism in Malaysia. *Procedia Computer Science, 105*, 345-352.

Skaar, D. A., Shao, Y., Haines, J. L., Stenger, J. E., Jaworski, J., Martin, E. R., DeLong, G. R., Moore, J. H., McCauley, J. L., Sutcliffe, J. S., AshleyKoch, A. E., Cuccaro, M. L., Folstein, S. E., Gilbert, J. R., & Pericak-Vance, M. A. (2005). Analysis of the RELN gene as a genetic risk factor for autism. *Molecular Psychiatry, 10*(6), 563.

Soke, G. N., Rosenberg, S. A., Hamman, R. F., Fingerlin, T., Robinson, C., Carpenter, L., Giarelli, E., Lee, L. C., Wiggins, L. D., Durkin, M. S. & DiGuiseppi, C. (2016). Brief report: Prevalence of self-injurious behaviors among children with Autism Spectrum Disorder—A population-based study. *Journal of Autism and Developmental Disorders, 46*(11), 3607-3614.

Sommer, F., & Bäckhed, F. (2013). The gut microbiota—masters of host development and physiology. *Nature Reviews Microbiology, 11*(4), 227.

Srinivasan, S. M., Pescatello, L. S., & Bhat, A. N. (2014). Current perspectives on physical activity and exercise recommendations for children and adolescents with autism spectrum disorders. *Physical Therapy, 94*(6), 875-889.

Stalgis-Bilinski, K. L., Boyages, J., Salisbury, E. L., Dunstan, C. R., Henderson, S. I., & Talbot, P. L. (2011). Burning daylight: balancing vitamin D requirements with sensible sun exposure. *Medical Journal of Australia, 194*(7), 345-348.

Stanish, H. I., Curtin, C., Must, A., Phillips, S., Maslin, M., & Bandini, L. G. (2017). Physical activity levels, frequency, and type among adolescents with and without autism spectrum disorder. *Journal of Autism and Developmental Disorders, 47*(3), 785-794.

Steege, M. W., Mace, F. C., Perry, L., & Longenecker, H. (2007). Applied behavior analysis: Beyond discrete trial teaching. *Psychology in the Schools, 44*(1), 91-99.

Stein, L. I., Polido, J. C., Mailloux, Z., Coleman, G. G., & Cermak, S. A. (2011). Oral care and sensory sensitivities in children with autism spectrum disorders. *Special Care in Dentistry, 31*(3), 102-110.

Steptoe, A. (2000). Psychosocial factors in the development of hypertension. *Annals of Medicine, 32*(5), 371-375.

Stern, D. N., & Bruschweiler-Stern, N. (1998). *The birth of a mother: How the motherhood experience changes you forever*. New York: Basic Books.

Stevenson, K., Jarred, S., Hinchcliffe, V., & Roberts, K. (2015). Can a dog be used as a motivator to develop social interaction and engagement with teachers for students with autism? *Support for Learning, 30*(4), 341–363.

Stuart, M., & McGrew, J. H. (2009). Caregiver burden after receiving a diagnosis of an autism spectrum disorder. *Research in Autism Spectrum Disorders, 3*(1), 86-97.

Stubbs, G., Henley, K., & Green, J. (2016). Autism: will vitamin D supplementation during pregnancy and early childhood reduce the recurrence rate of autism in newborn siblings? *Medical Hypotheses, 88*, 74-78.

Stuckelman, Z. D., Mulqueen, J. M., Ferracioli-Oda, E., Cohen, S. C., Coughlin, C. G., Leckman, J. F., & Bloch, M. H. (2017). Risk of irritability with psychostimulant treatment in children with ADHD: A meta-analysis. *Journal of Clinical Psychiatry, 78*(6), e648-e655.

Sukhodolsky, D. G., Bloch, M. H., Panza, K. E., & Reichow, B. (2013). Cognitive-behavioral therapy for anxiety in children with high-functioning autism: a meta-analysis. *Pediatrics, 132*(5), e1341-e1350.

Sullivan, K. (2015). An application of family stress theory to clinical work with military families and other vulnerable populations. *Clinical Social Work Journal, 43*(1), 89-97.

Sun, X., Allison, C., Matthews, F. E., Zhang, Z., Auyeung, B., Baron-Cohen, S., & Brayne, C. (2015). Exploring the underdiagnosis and prevalence of autism spectrum conditions in Beijing. *Autism Research, 8*(3), 250-260.

Super, S., Wagemakers, M. A. E., Picavet, H. S. J., Verkooijen, K. T., & Koelen, M. A. (2015). Strengthening sense of coherence: opportunities for theory building in health promotion. *Health Promotion International, 31*(4), 869-878.

Sutcliffe, J. S., Delahanty, R. J., Prasad, H. C., McCauley, J. L., Han, Q., Jiang, L., Li, C., Folstein, S. E., & Blakely, R. D. (2005). Allelic heterogeneity at the serotonin transporter locus (SLC6A4) confers susceptibility to autism and rigid-compulsive behaviors. *The American Journal of Human Genetics, 77*(2), 265-279.

Swaab, L., McCormack, L., & Campbell, L. E. (2017). Distress and psychological growth in parenting an adult child with autism spectrum disorder and aggression. *Advances in Neurodevelopmental Disorders, 1*(4), 260-270.

Tabak, B. A., Vrshek-Schallhorn, S., Zinbarg, R. E., Prenoveau, J. M., Mineka, S., Redei, E. E., Adam, E. K. & Craske, M. G. (2016). Interaction of CD38 variant and chronic interpersonal stress prospectively predicts social anxiety and depression symptoms over 6 years. *Clinical Psychological Science, 4*(1), 17-27.

Tager-Flusberg, H., & Kasari, C. (2013). Minimally verbal school-aged children with autism spectrum disorder: the neglected end of the spectrum. *Autism Research, 6*(6), 468-478.

Talebi, M., Matheson, K., & Anisman, H. (2016). The stigma of seeking help for mental health issues: Mediating roles of support and coping and the moderating role of symptom profile. *Journal of Applied Social Psychology, 46*(8), 470-482.

Tang, B., Piazza, C. C., Dolezal, D., & Stein, M. T. (2011). Severe feeding disorder and malnutrition in 2 children with autism. *Journal of Developmental & Behavioral Pediatrics, 32*(3), 264-267.

Tarakeshwar, N., & Pargament, K. I. (2001). Religious coping in families of children with autism. *Focus on Autism and Other Developmental Disabilities, 16*(4), 247-260.

Taylor, A. F., Kuo, F. E., & Sullivan, W. C. (2001). Coping with ADD: The surprising connection to green play settings. *Environment and Behavior, 33*(1), 54-77.

Taylor, A. F., & Kuo, F. E. (2011). Could exposure to everyday green spaces help treat ADHD? Evidence from children's play settings. *Applied Psychology: Health and Well-Being, 3*(3), 281-303.

Thoits, P. A. (1982). Conceptual, methodological, and theoretical problems in studying social support as a buffer against life stress. *Journal of Health and Social behavior, 23*, 145-159.

Thompson, G. A. (2018). Long-term perspectives of family quality of life following music therapy with young children on the autism spectrum: A phenomenological study. *Journal of Music Therapy, 54*(4), 432–459.

Thompson, G. A., & McFerran, K. S. (2015). “We’ve got a special connection”: Qualitative analysis of descriptions of change in the parent–child relationship by mothers of young children with autism spectrum disorder. *Nordic Journal of Music Therapy, 24*(1), 3–26.

Tick, B., Bolton, P., Happé, F., Rutter, M., & Rijsdijk, F. (2016). Heritability of autism spectrum disorders: a meta-analysis of twin studies. *Journal of Child Psychology and Psychiatry, 57*(5), 585-595.

Timmons, B. W., LeBlanc, A. G., Carson, V., Connor Gorber, S., Dillman, C., Janssen, I., Kho, M. E., Spence, J. C., Stearns, J. A. & Tremblay, M. S. (2012). Systematic review of physical activity and health in the early years (aged 0–4 years). *Applied Physiology, Nutrition, and Metabolism, 37*(4), 773-792.

Tobing, L. E., & Glenwick, D. S. (2007). Predictors and moderators of psychological distress in mothers of children with pervasive developmental disorders. *Journal of Family Social Work, 10*(4), 1-22.

Tomeny, T. S., Ellis, B. M., Rankin, J. A., & Barry, T. D. (2017). Sibling relationship quality and psychosocial outcomes among adult siblings of individuals with autism spectrum disorder and individuals with intellectual disability without autism. *Research in developmental disabilities, 62*, 104-114.

Toscano, C. V., Carvalho, H. M., & Ferreira, J. P. (2018). Exercise effects for children with autism spectrum disorder: metabolic health, autistic traits, and quality of life. *Perceptual and Motor Skills, 125*(1), 126-146.

Trice, A. D. (1985). An academic locus of control scale for college students. *Perceptual and Motor Skills, 61*, 1043-1046.

Tsai, H. W. J., Cebula, K., Liang, S. H., & Fletcher-Watson, S. (2018). Siblings' experiences of growing up with children with autism in Taiwan and the United Kingdom. *Research in Developmental Disabilities, 83*, 206-216.

Tung, L.-C., Huang, C.-Y., Tseng, M.-H., Yen, H.-C., Tsai, Y.-P., Lin, Y.-C., et al. (2014). Correlates of health-related quality of life and the perception of its importance in caregivers of children with autism. *Research in Autism Spectrum Disorders, 8*(9), 1235–1242.

Twoy, R., Connolly, P. M., & Novak, J. M. (2007). Coping strategies used by parents of children with autism. *Journal of the American Academy of Nurse Practitioners, 19*(5), 251-260.

Tyagi, V., Juneja, M., & Jain, R. (2018). Sleep Problems and Their Correlates in Children with Autism Spectrum Disorder: An Indian Study. *Journal of Autism and Developmental Disorders, 49*(3), 1169-1181.

Tyler, C. V., Schramm, S. C., Karafa, M., Tang, A. S., & Jain, A. K. (2011). Chronic disease risks in young adults with autism spectrum disorder: forewarned is forearmed. *American journal on intellectual and developmental disabilities*, *116*(5), 371-380.

Ung, D., Selles, R., Small, B. J., & Storch, E. A. (2015). A systematic review and meta-analysis of cognitive-behavioral therapy for anxiety in youth with high-functioning autism spectrum disorders. *Child Psychiatry & Human Development, 46*(4), 533-547.

Van Bakel, M. M. E., Delobel-Ayoub, M., Cans, C., Assouline, B., Jouk, P. S., Raynaud, J. P., & Arnaud, C. (2015). Low but increasing prevalence of autism spectrum disorders in a French area from register-based data. *Journal of Autism and Developmental Disorders, 45*(10), 3255-3261.

Van der Plas, E., Dupuis, A., Arnold, P., Crosbie, J., & Schachar, R. (2016). Association of autism spectrum disorder with obsessive-compulsive and attention-deficit/hyperactivity traits and response inhibition in a community sample. *Journal of Autism and Developmental Disorders, 46*(9), 3115-3125.

Van Steensel, F. J., Bögels, S. M., & Perrin, S., (2011). Anxiety disorders in children and adolescents with autistic spectrum disorders: a meta-analysis. *Clinical Child and Family Psychology Review, 14*(3), 302.

Vandewalle, G., Balteau, E., Phillips, C., Degueldre, C., Moreau, V., Sterpenich, V., Albouy, G., Darsaud, A., Desseilles, M., Dang-Vu, T. T., & Peigneux, P. (2006). Daytime light exposure dynamically enhances brain responses. *Current Biology, 16*(16), 1616-1621.

Vandewalle, G., Schmidt, C., Albouy, G., Sterpenich, V., Darsaud, A., Rauchs, G., Berken, P.Y., Balteau, E., Degueldre, C., Luxen, A. & Maquet, P. (2007). Brain responses to violet, blue, and green

monochromatic light exposures in humans: prominent role of blue light and the brainstem. *PloS one, 2*(11), e1247.

Vasa, R.A., Kalb, L., Mazurek, M., Kanne, S., Freedman, B., Keefer, A., Clemons, T. & Murray, D., (2013). Age-related differences in the prevalence and correlates of anxiety in youth with autism spectrum disorders. *Research in Autism Spectrum Disorders, 7*(11), 1358-1369.

Vasilopoulou, E., & Nisbet, J. (2016). The quality of life of parents of children with autism spectrum disorder: A systematic review. *Research in Autism Spectrum Disorders, 23*, 36-49.

Vehling, L., Brownell, M., Kaufert, J., Nickel, N. C., & Alessi-Severini, S. (2016). Children with autism spectrum disorder in Manitoba: prevalence, population characteristics and psychotropic medication use. *Health promotion and chronic disease prevention in Canada: research, policy and practice, 36*(11), 270.

Veltman, M. W., Craig, E. E., & Bolton, P. F. (2005). Autism spectrum disorders in Prader–Willi and Angelman syndromes: a systematic review. *Psychiatric Genetics, 15*(4), 243-254.

Ventura, J. N. (1987). The stresses of parenthood reexamined. *Family Relations, 36*, 26-29.

Vernhet, C., Dellapiazza, F., Blanc, N., Cousson-Gélie, F., Miot, S., Roeyers, H., & Baghdadli, A. (2018). Coping strategies of parents of children with autism spectrum disorder: a systematic review. *European Child & Adolescent Psychiatry, 28*, 747-758.

Vinkhuyzen, A. A., Eyles, D. W., Burne, T. H., Blanken, L. M., Kruithof, C. J., Verhulst, F., Jaddoe, V. W., Tiemeier, H. & McGrath, J. J. (2018). Gestational vitamin D deficiency and autism-related traits: The Generation R Study. *Molecular Psychiatry, 23*(2), 240.

Volk, H. E., Hertz-Picciotto, I., Delwiche, L., Lurmann, F., & McConnell, R. (2011). Residential proximity to freeways and autism in the CHARGE study. *Environmental Health Perspectives, 119*(6), 873.

Volk, H. E., Lurmann, F., Penfold, B., Hertz-Picciotto, I., & McConnell, R. (2013). Traffic-related air pollution, particulate matter, and autism. *JAMA Psychiatry, 70*(1), 71-77.

Vuong, H. E., & Hsiao, E. Y. (2017). Emerging roles for the gut microbiome in autism spectrum disorder. *Biological Psychiatry, 81*(5), 411-423.

Wacker, M., & Holick, M. F. (2013). Sunlight and Vitamin D: A global perspective for health. *Dermato-Endocrinology, 5*(1), 51-108.

Wallston, K. A., Strudler Wallston, B., & DeVellis, R. (1978). Development of the multidimensional health locus of control (MHLC) scales. *Health Education Monographs, 6*(1), 160-170.

Walters, S., Loades, M., & Russell, A. (2016). A systematic review of effective modifications to cognitive behavioural therapy for young people with autism spectrum disorders. *Review Journal of Autism and Developmental Disorders, 3*(2), 137-153.

Wang, L., Jia, M., Yue, W., Tang, F., Qu, M., Ruan, Y., Lu, T., Zhang, H., Yan, H., Liu, J., Guo, Y., Zhang, J., Yang, X., & Zhang, D. (2008). Association of the ENGRAILED 2 (EN2) gene with autism in Chinese Han population. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 147*(4), 434-438.

Wang, T., Shan, L., Du, L., Feng, J., Xu, Z., Staal, W. G., & Jia, F. (2016). Serum concentration of 25-hydroxyvitamin D in autism spectrum disorder: A systematic review and meta-analysis. *European Child & Adolescent Psychiatry, 25*(4), 341-350.

Wang, C., Geng, H., Liu, W., & Zhang, G. (2017). Prenatal, perinatal, and postnatal factors associated with autism: A meta-analysis. *Medicine, 96*(18), e6696.

Wang, Y., Wang, M. J., Rong, Y., He, H. Z., & Yang, C. J. (2019). Oxytocin therapy for core symptoms in autism spectrum disorder: An updated meta-analysis of randomized controlled trials. *Research in Autism Spectrum Disorders, 64*, 63-75.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality and Social Psychology, 54*(6), 1063.

Weber, E. U., Blais, A.-R., & Betz, N. E. (2002). A domain-specific risk-attitude scale: Measuring risk perceptions and risk behaviors. *Journal of Behavioral Decision Making, 15*(4), 263–290.

Weintraub, K. (2011). The prevalence puzzle: autism counts. *Nature News, 479*, 22–4.

Weiss, M. J. (2002). Hardiness and social support as predictors of stress in mothers of typical children, children with autism, and children with mental retardation. *Autism, 6*(1), 115-130.

Weiss, J. A., MacMullin, J. A., & Lunsky, Y. (2015). Empowerment and parent gain as mediators and moderators of distress in mothers of children with autism spectrum disorders. *Journal of Child and Family Studies, 24*(7), 2038-2045.

Weiss, J. A., Tajik-Parvinchi, D., Maughan, A. L., & Lake, J. (2018). Family Wellness in Intellectual and Developmental Disabilities: A Balanced Approach. *Current Developmental Disorders Reports, 5*(3), 157-164.

Weitlauf, A.S., McPheeters, M.L., Peters, B., Sathe, N., Travis, R., Aiello, R., Williamson, E., Veenstra-VanderWeele, J., Krishnaswami, S., Jerome, R. & Warren, Z. (2014). *Therapies for children with autism spectrum disorder: Behavioral Interventions Update.* Rockville, Md: Agency for Healthcare Research and Quality; 2014.Comparative Effectiveness Review No. 137. Prepared by the Vander-bilt Evidence-based Practice Center under contract 290-2012-00009-I.

Weitlauf AS, Sathe NA, McPheeters ML, & Warren Z. (2017). Interventions targeting sensory challenges in children with autism spectrum disorder—an update. Comparative Effectiveness Review No. 186. Rockville, MD: Agency for Healthcare Research and Quality, 2017.

Welch, M. G., & Ruggiero, D. A. (2005). Predicted role of secretin and oxytocin in the treatment of behavioral and developmental disorders: Implications for autism. *International Review of Neurobiology, 71*, 273-315.

Weston, L., Hodgekins, J., & Langdon, P. E. (2016). Effectiveness of cognitive behavioural therapy with people who have autistic spectrum disorders: A systematic review and meta-analysis. *Clinical Psychology Review, 49*, 41-54.

Wheeler, D., Williams, K., Seida, J., & Ospina, M. (2008). The Cochrane library and autism spectrum disorder: An overview of reviews. *Evidence-Based Child Health: A Cochrane Review Journal, 3*(1), 3–15.

White, S. W., Ollendick, T., Scahill, L., Oswald, D., & Albano, A. M. (2009). Preliminary efficacy of a cognitive-behavioral treatment program for anxious youth with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 39*(12), 1652.

White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review, 29*(3), 216-229.

Whiteley, P., Dodou, K., Todd, L., & Shattock, P. (2004). Body mass index of children from the United Kingdom diagnosed with pervasive developmental disorders. *Pediatrics International, 46*(5), 531-533.

Wigston, C., Falkmer, M., Vaz, S., Parsons, R., & Falkmer, T. (2017). Participation in extracurricular activities for children with and without siblings with autism spectrum disorder. *Developmental Neurorehabilitation, 20*(1), 25-39.

Williams, K., MacDermott, S., Ridley, G., Glasson, E. J., & Wray, J. A. (2008). The prevalence of autism in Australia. Can it be established from existing data? *Journal of Paediatrics and Child Health, 44*(9), 504-510.

Williams, K., Helmer, M., Duncan, G. W., Peat, J. K., & Mellis, C. M. (2008). Perinatal and maternal risk factors for autism spectrum disorders in New South Wales, Australia. *Child: Care, Health and Development, 34*(2), 249-256.

Williams, K., Brignell, A., Randall, M., Silove, N., & Hazell, P. (2013). Selective serotonin reuptake inhibitors (SSRIs) for autism spectrum disorders (ASD). *Cochrane Database of Systematic Reviews, 8* (2013), Cd004677.

Williams, S., Leader, G., Mannion, A., & Chen, J. (2015). An investigation of anxiety in children and adolescents with autism spectrum disorder. *Research in Autism Spectrum Disorders, 10*, 30-40.

Windham, G. C., Zhang, L., Gunier, R., Croen, L. A., & Grether, J. K. (2006). Autism spectrum disorders in relation to distribution of hazardous air pollutants in the San Francisco Bay area. *Environmental health perspectives, 114*(9), 1438-1444.

Wingate, M., et al., (2014). *Prevalence of autism spectrum disorder among children aged 8 years-autism and developmental disabilities monitoring network, 11 sites, United States, 2010*. MMWR Surveillance Summaries, *63*(2).

Wolff, J. J., Hazlett, H. C., Lightbody, A. A., Reiss, A. L., & Piven, J. (2013). Repetitive and self-injurious behaviors: associations with caudate volume in autism and fragile X syndrome. *Journal of Neurodevelopmental Disorders, 5*(1), 12.

Wood, J. J., Drahota, A., Sze, K., Har, K., Chiu, A., & Langer, D. A. (2009). Cognitive behavioral therapy for anxiety in children with autism spectrum disorders: A randomized, controlled trial. *Journal of Child Psychology and Psychiatry, 50*(3), 224-234.

Woynaroski, T., Yoder, P., & Watson, L. R. (2016). Atypical cross-modal profiles and longitudinal associations between vocabulary scores in initially minimally verbal children with ASD. *Autism Research, 9*(2), 301-310.

Wu, S., Jia, M., Ruan, Y., Liu, J., Guo, Y., Shuang, M., Gong, X., Zhang, Y., Yang, X. & Zhang, D., (2005). Positive association of the oxytocin receptor gene (OXTR) with autism in the Chinese Han population. *Biological Psychiatry, 58*(1), 74-77.

Wyver, S., Tranter, P., Naughton, G., Little, H., Sandseter, E. B. H., & Bundy, A. (2010). Ten ways to restrict children's freedom to play: The problem of surplus safety. *Contemporary Issues in Early Childhood, 11*(3), 263-277.

Xiong, N., Yang, L., Yu, Y., Hou, J., Li, J., Li, Y., Liu, H., Zhang, Y. & Jiao, Z., (2011). Investigation of raising burden of children with autism, physical disability and mental disability in China. *Research in Developmental Disabilities, 32*(1), 306-311.

Xu, M., Xu, X., Li, J., & Li, F. (2019). Association Between Gut Microbiota and Autism Spectrum Disorder: A Systematic Review and Meta-Analysis. *Frontiers in Psychiatry, 10*, 473.

Xue, J., Ooh, J., & Magiati, I. (2014). Family functioning in Asian families raising children with autism spectrum disorders: the role of capabilities and positive meanings. *Journal of Intellectual Disability Research, 58*(5), 406-420.

Yamada, A., Kato, M., Suzuki, M., Suzuki, M., Watanabe, N., Akechi, T., & Furukawa, T. A. (2012). Quality of life of parents raising children with pervasive developmental disorders. *BMC Psychiatry, 12*(1), 119.

Yamasue, H. (2016). Promising evidence and remaining issues regarding the clinical application of oxytocin in autism spectrum disorders. *Psychiatry and Clinical Neurosciences, 70*(2), 89-99.

Yamasue, H., Okada, T., Munesue, T., Kuroda, M., Fujioka, T., Uno, Y., Matsumoto, K., Kuwabara, H., Mori, D., Okamoto, Y. & Yoshimura, Y. (2018). Effect of intranasal oxytocin on the core social symptoms of autism spectrum disorder: A randomized clinical trial. *Molecular Psychiatry,* 1-10.

Yang, S. Y., Cho, S. C., Yoo, H. J., Cho, I. H., Park, M., Yoe, J., & Kim, S. A. (2010). Family-based association study of microsatellites in the 5' flanking region of AVPR1A with autism spectrum disorder in the Korean population. *Psychiatry Research, 178*(1), 199-201.

Yasuda, H., Yoshida, K., Yasuda, Y., & Tsutsui, T. (2011). Infantile zinc deficiency: association with autism spectrum disorders. *Scientific Reports, 1*, 129.

Yirmiya, N., & Shaked, M. (2005). Psychiatric disorders in parents of children with autism: A meta-analysis. *Journal of Child Psychology and Psychiatry, 46*(1), 69-83.

Yoder, P. J., & Stone, W. L. (2006). A randomized comparison of the effect of two prelinguistic communication interventions on the acquisition of spoken communication in preschoolers with ASD. *Journal of Speech, Language, and Hearing Research, 49*(4), 698–711.

Yoo, H. (2015). Genetics of autism spectrum disorder: current status and possible clinical applications. *Experimental Neurobiology, 24*(4), 257-272.

Yoo, H. J., Cho, I. H., Park, M., Yang, S. Y., & Kim, S. A. (2012). Family based association of GRIN2A and GRIN2B with Korean autism spectrum disorders. *Neuroscience Letters, 512*(2), 89-93.

Yu, Y., McGrew, J. H., Rand, K. L., & Mosher, C. E. (2018). Using a model of family adaptation to examine outcomes of caregivers of individuals with autism spectrum disorder transitioning into adulthood. *Research in Autism Spectrum Disorders, 54*, 37-50.

Yuill, N., Strieth, S., Roake, C., Aspden, R., & Todd, B. (2007). Brief report: Designing a playground for children with autistic spectrum disorders—effects on playful peer interactions. *Journal of Autism and Developmental Disorders, 37*(6), 1192-1196.

Zeedyk, S. M., Rodriguez, G., Tipton, L. A., Baker, B. L., & Blacher, J. (2014). Bullying of youth with autism spectrum disorder, intellectual disability, or typical development: Victim and parent perspectives. *Research in Autism Spectrum Disorders, 8*(9), 1173-1183.

Zeglam, A. M., & Maouna, A. J. (2012). Prevalence of autistic spectrum disorders in Tripoli, Libya: The need for more research and planned services. *Eastern Mediterranean Health Journal, 18*(2), 184.

Zhang, X., Lv, C. C., Tian, J., Miao, R. J., Xi, W., Hertz-Picciotto, I., & Qi, L. (2010). Prenatal and perinatal risk factors for autism in China. *Journal of Autism and Developmental Disorders, 40*(11), 1311-1321.

Zimet, G. D., Dahlem, N. W., Zimet, S. G., & Farley, G. K. (1988). The multidimensional scale of perceived social support. *Journal of Personality Assessment, 52*(1), 30-41.

Zimet, G. D., Powell, S. S., Farley, G. K., Werkman, S., & Berkoff, K. A. (1990). Psychometric characteristics of the multidimensional scale of perceived social support. *Journal of Personality Assessment, 55*(3-4), 610-617.

Zimmerman, D., Ownsworth, T., O'Donovan, A., Roberts, J., & Gullo, M. J. (2016). High-functioning autism spectrum disorder in adulthood: A systematic review of factors related to psychosocial outcomes. *Journal of Intellectual & Developmental Disability, 43*(1), 2-19.

List of Publications

List of publications arising from this thesis:

- Syed, S., Moore, K. A., & March, E. (2017). A review of prevalence studies of Autism Spectrum Disorder by latitude and solar irradiance impact. *Medical Hypotheses, 109*, 19-24.
- Syed, S., Moore, K., & March, E. (2018). Coping strategies of parents of children with Autism in regional Australia. In P. Buchwald, K. Moore (Eds.), *Stress and anxiety: Theories and realities* (pp. 119-127). Logos Verlag.
- Syed, S., & Moore, K. A. (under review). Psychosocial impact on families living with a child with ASD in regional areas.
- Syed, S., & Moore, K. A. (under review). Exploratory model of the impact of living with a child with ASD.

Appendix A: Paper accepted by *Medical Hypotheses*

Medical Hypotheses 109 (2017) 19–24

Contents lists available at ScienceDirect

Medical Hypotheses

journal homepage: www.elsevier.com/locate/mehy

A review of prevalence studies of Autism Spectrum Disorder by latitude and solar irradiance impact

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A B S T R A C T

Autism Spectrum Disorder (ASD) is a lifelong disability with no known cause or cure. Among the suggested etiologies, is Cannell's hypothesis of a deficiency in Vitamin D the main natural source of which is Solar Ultraviolet-B (UVB) radiation. The aim in this paper is to build on this hypothesis and explore the relationship of solar irradiance of which UVB is a component, by latitude with the prevalence rates of ASD. Twenty-five reports published between 2011 and 2016 using comparable diagnostic criteria were reviewed. The results suggest a tendency for the prevalence rates of ASD to be lowest in countries near the equator and for this rate to increase as the latitude increases. These findings provide some support not just for the Vitamin D hypothesis, but also for a new proposition that along with UVB radiation, the entire solar radiation spectrum which reaches the earth, may play a role in ASD. While these results are both novel and encouraging in terms of the potential efficacy of exposure to natural sunlight, further research is warranted before results can be considered definitive, and before the implications of the findings can be implemented clinically.

Introduction

Autism Spectrum Disorder (ASD) is an umbrella term for multiple neurodevelopmental conditions characterized by repetitive or stereotyped behaviors and pervasive deficits in social communications and interactions [1]. ASD is considered a lifelong disability which has an impact on both the individual and the family [2,3], as well as being a cost to society in general [2,4]. Among these costs are additional health care, disability support in school and, in some instances, the loss of a productive working life and the provision of social security. In addition, ASD is associated with several comorbidities [5,6] such as Attention-Deficit/Hyperactivity Disorder [7–10], Obsessive Compulsive Disorder [7,8], anxiety disorders [11–15], sensory over-responsivity [13,16–19], sleep disorders [20–22], and gastrointestinal problems [13,20,23,24].

The prevalence of ASD, or at least reports thereof, have increased substantially from 1 in 500 in 1995, 1 in 250 in 2001 [25], to 1 in 68 in 2010 in the USA in children less than 8 years [26]. However, it is possible that rather than an actual increase in the rate of ASD, these statistics reflect higher prevalence associated with expanded definitions of ASD, increased public awareness and help-seeking. Further changes in the prevalence rates for ASD may also be a product of social de-stigmatization associated with ASD [27].

Despite the efforts of scientists who have investigated a myriad of environmental and genetic factors including air pollution [28–32]; environmental toxins such as mercury, nickel, selenium, lead, cadmium, aluminum, vinyl chloride and trichloroethylene [33–37]; genetic heritability [38–40]; hormonal imbalances such as oxytocin [41], vasopressin [42], and more recently Vitamin D deficiency [43,44] the etiology of ASD remains uncertain.

Following Cannell's proposal [43,44] that Vitamin D deficiency could be a risk factor for ASD several researchers have found low Vitamin D levels in patients with ASD [45,46], their siblings [47] and also maternal deficiencies [4,48,49]. Laboratory research has explored the genes regulated by Vitamin D [50,51]. Overall, these results have pointed towards an association between Vitamin D deficiency and autism-related traits [44,52]. Further support for Cannell's hypothesis comes from reports that some level of improvement in autistic symptoms has been achieved via the administration of Vitamin D supplements [53–55]. Despite this tentative support for an association between Vitamin D and ASD, no conclusive evidence of this relationship

Abbreviations: ADI-R, Autism Diagnostic Interview-Revised; ADOS, Autism Diagnostic Observation Schedule; ASSQ, Autism Spectrum Screening Questionnaire; ASQ, Autism Screening Questionnaires; BSA, Body Surface Area; CAST, Mandarin Childhood Autism Spectrum Test; ICD-10, International Classification of Diseases, 10th edition; ISAA, Indian Scale for Assessment of Autism; M-CHAT, Modified Checklist for Autism in Toddlers; M-CHAT/ES, Spanish version of the Modified Checklist for Autism in Toddlers; VBAS, Vineland Adaptive Behaviour Scale

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E-mail address: k.moore@federation.edu.au (K.A. Moore).

http://dx.doi.org/10.1016/j.mehy.2017.09.012
Received 28 June 2017; Accepted 15 September 2017

Appendix B: Syed et al. Published chapter in *Stress, Anxiety and Resilience*

CHAPTER 11

COPING STRATEGIES OF PARENTS OF CHILDREN WITH AUTISM IN REGIONAL AUSTRALIA

Somayya Syed[1], Kathleen A. Moore[1,2], Evita March[1]
Federation University Australia, Churchill 3842, Australia
Charles Stunt University, Bathurst 2795, Australia
Email: k.moore@federation.edu.au

Abstract

The aim in the current study was to explore the coping strategies of parents of children with Autism Spectrum Disorder (ASD) in regional Australia. Mothers (n=12), fathers (n=3) and one grandmother took part in semi-structured interviews to discuss the problems they faced and their coping styles. All participants reported adopting problem-focused coping strategies. They discussed their innovative solutions to cope with stress and to tackle the lack of support structures and health services in regional Australia. On the other hand, only mothers reported using social support resources as a major coping strategy. These resources included engaging with their friends and accessing local ASD support groups. Another theme that was common to both genders was social withdrawal, which they attributed to society's insensitive attitude towards "Autism" and Autistic children. Greater awareness of ASD in the general populations will reduce this stress on parents. Also, an understanding of the factors behind the coping strategies adopted by parents of children with ASD, and their health service needs, will facilitate better delivery of healthcare and support structures for them.

Introduction

Raising a child with Autism Spectrum Disorder (ASD) is challenging and has an impact on parents and other family members. The pervasive and severe deficits often present in children with ASD are associated with a plethora of difficulties and can contribute to an increase in the mental and physical health problems of parents (Karst & Hecke, 2012). Compared with mothers of typically developing children, mothers of children with ASD report significantly higher fatigue (Giallo, Wood, Jellett, & Porter, 2013). Additionally, the severity of behavioural problems has been found to account for much of the variance in caregivers' psychological and physiological health (Lovell, Moss, & Wetherell, 2015). Consequently, the family has to adapt.

Family adaptation refers to the family's ability to manage change and associated stressors (Olson, Sprenkle, & Russell, 1979) which are considered to be ongoing for families of a child with ASD. The extant literature shows differences in the adaptation of the families of children with ASD with variability in coping strategies and social support linked to individual differences in outcomes (McStay, Trembath, & Dissanayake, 2014; Paynter, Riley, Beamish, Davies, & Milford, 2013). Despite a growing literature on psychosocial studies, the

Approval

Human Research Ethics Committee

Federation UNIVERSITY • AUSTRALIA

Principal Researcher:	Dr Kate Moore
Other/Student Researcher/s:	Dr Somayya Syed Dr Evita March
School/Section:	**School of Health Sciences and Psychology**
Project Number:	**A16-152**
Project Title:	**Psychosocial Impact of Autism Spectrum Disorder on Families.**
For the period:	01/12/2016 **to** 31/08/2017

Quote the Project No: A16-152 in all correspondence regarding this application.

Please note: Ethics Approval is contingent upon the submission of Annual Progress reports and a Final report upon completion of the project. It is the responsibility of researchers to make a note of the following dates and submit these reports in a timely manner, as reminders may not be sent out. Failure to submit reports will result in your ethics approval lapsing

REPORTS TO HREC:

A Final report for this project must be submitted to the Ethics Officer on:
30 September 2017

These report forms can be found at:
http://federation.edu.au/research-and-innovation/research-support/ethics/human-ethics/human-ethics3

Fiona Koop
Ethics Officer
1 December 2016

Please see attached 'Conditions of Approval'.

Annual/Final Project Report

Human Research Ethics Committee

Federation UNIVERSITY • AUSTRALIA

Please indicate the type of report	☐ Annual Report (**Omit 3b & 5b**) ☒ Final Report
Project No:	A16-152
Project Name:	Psychosocial Impact of Autism Spectrum Disorder on Families
Principal Researcher:	Dr. Kate Moore
Other Researchers:	Dr. Somayya Syed Dr. Evita March
Date of Original Approval:	1/12/2016
School / Section:	School of Health Sciences and Psychology
Phone:	(03) 5122 8922
Email:	k.moore@federation.edu.au

Please note: For HDR candidates, it is a requirement of candidature to submit Candidature reports annually to research.degrees@federation.edu.au in addition to Ethics Annual/Final reports.

1) Please indicate the current status of the project:				
1a) Yet to start				☐
1b) Continuing				☐
1c) Data collection completed				☒
1d) Abandoned / Withdrawn:				☐
1e) If the approval was subject to certain conditions, have these conditions been met? (If not, please give details in the comments box below)			☐ Yes	☐ No
Comments:				
1f) Data Analysis	☐ Not yet commenced	☒ Proceeding	☐ Complete	☐ None
1g) Have ethical problems been encountered in any of the following areas:				
Study Design			☐ Yes	☒ No
Recruitment of Subjects			☐ Yes	☒ No
Finance				

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Facilities, Equipment (If yes, please give details in the comments box below)	☐ Yes ☐ Yes	☒ No ☒ No
Comments:		

2a) Have amendments been made to the originally approved project?	
☒ No	☐ Yes
2b) If yes, was HREC approval granted for these changes?	
☐ Yes	**Provide detail:** ☐ Yes **Application for Amendment to an Existing Project** ☐ Yes **Change of Personnel** ☐ Yes **Extension Request**
☐ No	**If you have made changes, but not had HREC approval, provide detail as to why this has not yet occurred:**
2c) Do you need to submit any amendments now?	
☒ No	☐ Yes **Application for Amendment to an Existing Project** ☐ Yes **Change of Personnel** ☐ Yes **Extension Request** *** NB: If 'Yes', download & submit the appropriate request to the HREC for approval:** **Please note: Extensions will not be granted retrospectively. Apply well prior to the project end date, to ensure continuity of HRE approval.**

3a) Please indicate where you are storing the data collected during the course of this project: (Australian code for the Responsible conduct of Research Ch 2.2.2, 2.5 – 2.7)
The audio recordings of interviews are deleted once transcribed (this has been done). Transcriptions of the interviews are stored in a password protected file on a university computer
3b) Final Reports: Advise when & how stored data will be destroyed **(Australian code for the Responsible conduct of Research Ch 2.1.1)**
Transcriptions will be deleted from both the folder and the recycling box after five years

4) Have there been any events that might have had an adverse effect on the research participants OR unforeseen events that might affect continued ethical acceptability of the

Annual/Final Project Report

Human Research Ethics Committee

Federation UNIVERSITY • AUSTRALIA

project?	
☒ No	☐ Yes * **NB: If 'yes', please provide details in the comments box below:**
Comments:	

5a) Please provide a short summary of results of the project so far (no attachments please):
The common themes identified in the interviews include: friends and family support; financial factors; impact on social activities; self-discipline; problem solving; impact on the couple's relationship; love for child; strategies for outdoor activities and insights on coping strategies.
5b) Final Reports: Provide details about how the aims of the project, as stated in the application for approval, were achieved (or not achieved). (Australian code for the Responsible conduct of Research 4.4.1)
Information on parents coping strategies and impact of Autism on the families was gathered during the interviews, thus achieving the aim of the study. This analysis is still ongoing.

6) Publications: Provide details of research dissemination outcomes for the previous year resulting from this project: eg: Community seminars; Conference attendance; Government reports and/or research publications

7) The HREC welcomes any feedback on: • **Difficulties experienced with carrying out the research project; or** • **Appropriate suggestions which might lead to improvements in ethical clearance and monitoring of research.**

8) Signatures

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Principal Researcher:	Kate Moore	**Date:**	17/8/2017
Other/Student Researchers:	Somayya Syed	**Date:**	17/8/2017
	…… Print name:	**Date:**	

Submit to the Ethics Officer, Gippsland or Mt Helen campus, by the due date:
research.ethics@federation.edu.au

Appendix D: Email Invitation to Support Group Members

Advertisement for:

ASD Support Group in Traralgon Noticeboard and Inclusion in their email to members

Morwell support Group,

FaceBook

Psychosocial Impact of Autism Spectrum Disorder on Families

My name is Somayya Syed and in conjunction with Drs Kate Moore and Evita March at Federation University, I am conducting research in the impact of living with a child with Autism Spectrum Disorder (ASD) as part of my PhD.

I would like to invite you to participate in this study. If you agree, please contact me on my email/phone number below to organise a time for us to meet. Or, you might prefer to meet along with other family members of a child with ASD in a focus group. The times for these are:

- TBA
- TBA
- TBA

Interviews and focus will be held at your ASD Support Group Centre. We estimate it will take about 30 minutes of your time.

Thank you for considering this invitation.

Dr. Somayya Syed

Associate Professor Kate Moore

Dr Evita March

School of Health Sciences and Psychology

Federation University Australia

Emails:

somayyasyed@students.federation.edu.au

k.moore@federation.edu.au

e.march@federation.edu.au

Telephone: 03 8922 5122

Appendix E: Semi-Structured Interview Questions

Participants will be thanked for the participation and will be asked to provide demographic data on their gender, age, and age and gender of their child with ASD. These data will be recorded anonymously on a data sheet separate from the audio recordings of interviews and focus groups.

The following open-ended questions will be used as prompts during each session:

- Please tell me about when you child was diagnosed with ASD? How old was your child? What lead to you seeking help at that time? How severe is your child's ASD?
- How has your child having ASD affected
    - other children [if relevant]
    - your relationship
    - your work
    - social life?
- What are the biggest difficulties you face as a result of your child having a diagnosis of ASD?
- Is there any family history of ASD?
- How do friend and family react?
    - Supportive?
    - Available to assist?
- If the child is at school….
    - How is their progress
    - What support does the school offer
- What impact is there on the family's overall lifestyle?
    - Diet
    - Exercise
    - Does your child spend time outdoors
    - Playing with others
- Is your child on medications?
    - Do these affect him/her
- What concerns do you have about your child going forward?

Follow up questions and prompts will be used to explore issues that arise during the discussions. Participants will be encouraged to discuss any issues they considered relevant and to provide examples.

Approval

Human Research Ethics Committee

Federation UNIVERSITY • AUSTRALIA

Principal Researcher:	Dr Kate Moore
Other/Student Researcher/s:	Dr Somayya Syed Dr Evita March
School/Section:	**School of Health Sciences & Psychology**
Project Number:	**A18-072**
Project Title:	**Impact of Autism spectrum disorder on Families.**
For the period:	30/05/2018 **to** 31/03/2019

Quote the Project No: A18-072 in all correspondence regarding this application.

Approval has been granted to undertake this project in accordance with the proposal submitted for the period listed above.

Please note: It is the responsibility of the Principal Researcher to ensure the Ethics Officer is contacted immediately regarding any proposed change or any serious or unexpected adverse effect on participants during the life of this project.

In Addition: Maintaining Ethics Approval is contingent upon adherence to all Standard Conditions of Approval as listed on the final page of this notification

COMPLIANCE REPORTING DATES TO HREC:

Final project report:
30 April 2019

The combined annual/final report template is available at:
http://federation.edu.au/research-and-innovation/research-support/ethics/human-ethics/human-ethics3

Fiona Koop
Ethics Officer
30 May 2018
Please note the standard conditions of approval on Page 2:

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Human Research Ethics Committee

Federation UNIVERSITY • AUSTRALIA

Please indicate the type of report	☐ Annual Report (**Omit 3b & 5b**) ☒ Final Report
Project No:	A18-072
Project Name:	Impact of Autism Spectrum Disorder on Families
Principal Researcher:	Dr. Kate Moore
Other Researchers:	Dr. Somayya Syed Dr. Evita March
Date of Original Approval:	30/5/2018
School / Section:	School of Health Sciences & Psychology
Phone:	(03) 5122 8922
Email:	k.moore@federation.edu.au

Please note: For HDR candidates, this Ethics annual report is a separate requirement, in addition to your HDR Candidature annual report, which is submitted mid-year to research.degrees@federation.edu.au.

1) Please indicate the current status of the project:				
1a) Yet to start				☐
1b) Continuing				☐
1c) Data collection completed				☒
1d) Abandoned / Withdrawn:				☐
1e) If the approval was subject to certain conditions, have these conditions been met? (If not, please give details in the comments box below)			☐ Yes	☐ No
Comments:				
1f) Data Analysis	☐ Not yet commenced	☒ Proceeding	☐ Complete	☐ None
1g) Have ethical problems been encountered in any of the following areas:				
Study Design			☐ Yes	☒ No
Recruitment of Subjects			☐ Yes	☒ No

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Finance Facilities, Equipment (If yes, please give details in the comments box below)	☐ Yes ☐ Yes	☒ No ☒ No
Comments:		

2a) Have amendments been made to the originally approved project?	
☒ No	☐ Yes
2b) If yes, was HREC approval granted for these changes?	
☐ Yes	**Provide detail:** ☐ Yes **Application for Amendment to an Existing Project** ☐ Yes **Change of Personnel** ☐ Yes **Extension Request**
☐ No	**If you have made changes, but not had HREC approval, provide detail as to why this has not yet occurred:**
2c) Do you need to submit any amendments now?	
☒ No	☐ Yes **Application for Amendment to an Existing Project** ☐ Yes **Change of Personnel** ☐ Yes **Extension Request** *** NB: If 'Yes', download & submit the appropriate request to the HREC for approval:** **Please note: Extensions will not be granted retrospectively. Apply well prior to the project end date, to ensure continuity of HRE approval.**

3a) Please indicate where you are storing the data collected during the course of this project: (Australian code for the Responsible conduct of Research Ch 2.2.2, 2.5 – 2.7)
Data collected is stored in a password protected file on a university computer.
3b) Final Reports: Advise when & how stored data will be destroyed (Australian code for the Responsible conduct of Research Ch 2.1.1)
Data will be deleted from both the folder and the recycling box after five years

4) Have there been any events that might have had an adverse effect on the research participants OR unforeseen events that might affect continued ethical acceptability of the

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project?	
☒ No	☐ Yes * **NB: If 'yes', please provide details in the comments box below:**
Comments:	

5a) Please provide a short summary of results of the project so far (no attachments please):
Data analyses are proceeding – to date we have extracted four factors related to the impact of living with a child with ASD: impact on social life, relationships, siblings, finances/work
5b) Final Reports: Provide details about how the aims of the project, as stated in the application for approval, were achieved (or not achieved). (Australian code for the Responsible conduct of Research 4.4.1)
Information on parents coping strategies and impact of Autism on the families was gathered during the interviews, thus achieving the aim of the study. This analysis is still ongoing.

6) Publications: Provide details of research dissemination outcomes for the previous year resulting from this project: eg: Community seminars; Conference attendance; Government reports and/or research publications
NA

7) The HREC welcomes any feedback on: • **Difficulties experienced with carrying out the research project; or** • **Appropriate suggestions which might lead to improvements in ethical clearance and monitoring of research.**

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8) Signatures			
Principal Researcher:	Print name: Kate Moore	**Date:**	2/4/19
Other/Student Researchers:	*Other Researcher* Print name: Somayya Syed	**Date:**	2/4/19
	..... Print name:	**Date:**	

Submit to the Ethics Officer, Mt Helen campus, by the due date:
research.ethics@federation.edu.au

Appendix G: The Plain Language Information Statement

The Plain Language Information Statement

School of Heath Sciences and Psychology

Project Title	Impact of Autism Spectrum Disorder on Families
Project Supervisor	Associate Professor Kate Moore & Dr Evita March
PhD Researcher	Dr. Somayya Syed

Dear Participant,

We are currently seeking parents, guardians, and grandparents who live with a child with Autism to participate in an online survey conducted by Dr. Somayya Syed as part of her PhD under the supervision of Associate Professor Kate Moore and Dr Evita March, in the Faculty of Health at Federation University Australia. This survey has received approval from Federation University's Human Research Ethics Committee. This aim of this survey is to investigate the impact on families living with a child with Autism.

We would like to invite you to participate in this anonymous survey. If you agree, you are requested to complete an online survey. That contains questions about what impact living with a child with ASD has had on yourself, your relationship, work, and other family members. It is anticipated that the survey will take about 15-20 minutes of your time.

Participation in this survey is voluntary, and you have the right to withdraw your consent to participate or discontinue the survey at any time without explanation by simply exiting the website. As the data are anonymous, should you change your mind, we shall not be able to delete your data once you have submitted the questionnaire.

Your anonymous responses to the questions will form part of a larger database. Only the researchers listed above will have access to the anonymous data. Any publications that arise from this online survey will be based on this anonymous data. Please note, that by submitting the completed survey you are deemed to have given your informed consent to participate in this study.

It is unlikely that you will experience distress or become uncomfortable while answering the questions, however if you do, please do not continue. Please close your web browser to exit the questionnaire without any penalty or your data to date being saved, and do not hesitate call contact Lifeline on 13 11 14 or your medical practitioner.

A summary of the results will be available in December, 2018. If you would like a copy of the results, please email Associate Professor Kate Moore and the information will be forwarded to you.

If you have any questions, please do not hesitate to contact Dr Moore, or if you have any concerns about the conduct of the research, please contact the University Ethics Officers, details below.

Thank you for considering participation in this research.

If you have any questions, or you would like further information regarding the project titled **Psychosocial Impact of Autism Spectrum Disorder on Families** please contact the Principal Researcher **Associate Professor Kate Moore**, of the Faculty of Health:
PH: 03 5122 8922
EMAIL: k.moore@federation.edu.au

Should you (i.e. the participant) have any concerns about the ethical conduct of this research project, please contact the Federation University Ethics Officer, Research Services, Federation University Australia, PO Box 663, Mt Helen VIC 3353. Telephone: (03) 5327 9765, Email: research.ethics@federation.edu.au

CRICOS Provider Number 00103D

Appendix H: Recruitment Advertisement

For Facebook and for Autism Support Groups

Hello everyone

My name is Somi Syed and I'm completing my PhD in Psychology at Federation University Churchill campus under the supervision of Associate Professor Kate Moore. For my thesis, I am conducting research into the impact of living with a child with Autism on families.

If you are a parent, grandparent or guardian (18 years of age or older), of a child with Autism - I invite you to participate in my research. Participation in this study is voluntary and the questionnaire will be completely anonymous. It will take approximately 15 minutes to complete. This aim of this survey is to investigate the impact on families living with a child with Autism.

If you are interested, please go to the following link where you will find more information about the study and the questionnaire

(www.TBA)

Your participation would be greatly appreciated. Please feel free to share this post or pass on to any other families with a child with Autism who you think may be interested in participating in this study.

If you have any questions or would like more information, please contact myself on

somayyasyed@students.federation.edu.au or Kate on k.moore@federation.edu.au

Thank you for your time in considering this invitation.