An Examination of Selected Psychophysiological Parameters of Stress in Parents of Children with Autism Spectrum Disorders

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Dissertation submitted in partial fulfilment of the requirements for the
Degree of Doctor of Philosophy in Applied Behaviour Analysis

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November, 2013

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Declaration and Statements

DECLARATION

This work has not been previously accepted in substance for any degree, and is not being concurrently submitted in candidature for any degree.

Signed.. (candidate)

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STATEMENT 1

This thesis is the result of my own investigations, except where otherwise stated. Where correction services have been used, the extent and nature of the correction is clearly marked in a footnote(s).

Other sources are acknowledged by footnotes giving explicit references. A bibliography is appended.

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I hereby give consent for my thesis, if accepted, to be available for photocopying and for inter-library loan, and for the title and summary to be made available to outside organisations.

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Summary

Autism Spectrum Disorders (ASD) are neurodevelopmental disorders that are often associated with a broad range of difficulties and challenges for the individual. Parents of children with ASD often experience higher levels of stress than parents of typically developing children. However, there is a lack of research investigating the physiological effects of stress related to caring for a child with ASD on the health of parents. The purpose of this research was to examine factors that predict stress among parents of children with ASD. Furthermore, this research investigated physiological markers of stress (i.e., blood pressure (BP), heart rate (HR), cortisol, and alpha-amylase (sAA) levels) to determine the effects, if any, of stress related to parenting a child with ASD on these physiological markers. Three interrelated studies were conducted, whereby participants wore an ambulatory blood pressure monitor for 24 hours, collected saliva samples which were analysed for cortisol and sAA levels, and completed a number of questionnaires.

Chapter 2 investigated stress measurements among mothers of children with ASD (N = 74). The aim was to identify factors predictive of stress, and to determine if mothers of children with ASD are at risk of experiencing hypocortisolism (i.e., blunted cortisol responses). A higher quantity of unmet service needs, sleep problems, use of maladaptive coping strategies, adaptive behaviour and socialisation deficits, and oppositional behaviour predicted higher levels of stress. Furthermore, there was evidence of hypocortisolism.

Chapter 3 compared stress measurements between mothers (n = 19) and fathers (n = 19) of children with ASD to determine if mothers and fathers are affected differently by stress related to caring for a child with ASD. Mothers of children with ASD reported higher parenting responsibility, anxiety, depression, and parenting stress than fathers of children with ASD. Mean cardiovascular (CV), cortisol, and sAA levels did not significantly differ between mothers and fathers. However, fathers had significantly higher BP variability than mothers, and mothers had significantly lower HR variability than fathers, both of which are associated with poorer health outcomes. Additionally, fathers' stress was found to be correlated with mothers' anxiety and depression, although mothers' stress was not significantly correlated with fathers' anxiety and depression. There was evidence of hypocortisolism among both mothers and fathers.

Finally, Chapter 4 compared stress measurements between dyads of parents of children with (*n* = 19) and without (*n* = 19) ASD, in order to control for stress related to normative parenting. Parents of children with ASD reported significantly higher levels of anxiety, depression, and parenting stress than parents of typically developing children. The parenting groups did not differ with respect to mean CV, cortisol, or sAA levels. However, parents of children with ASD had significantly lower absolute levels of cortisol on the third awakening response than parents of typically developing children, suggesting a risk of hypocortisolism. Fathers of children with ASD and mothers of typically developing children were found to have the highest sleeping BP variability, suggesting that the risk of CV disease for mothers and fathers may depend on the relative risk imposed by the impact of CV variability in the respective parenting groups.

The results of the current thesis were discussed in relation to their practical implications, in addition to future research directions. The overall research findings indicate that further supports are needed for parents of children with ASD in order to prevent the development of stress-related health problems.

Acknowledgements

I would like to extend my sincere gratitude to my supervisor, Professor Jack James, for your wonderful support and advice. Thanks in particular for your incredibly helpful feedback and support in the final stages of the thesis. Working with you has been a fantastic learning experience, and it has been a privilege to have had that opportunity. I am extremely grateful for all your support, guidance, and kindness. I would also like to thank my supervisor, Dr. Geraldine Leader, and I would like to thank Dr. Jennifer Holloway and Dr. Olive Healy for your support and guidance throughout the Ph.D. programme. Declan Coogan, I'm very grateful for your technical support, particularly in setting up the bioassay laboratory equipment. You have the patience of a saint! Thanks also to Dr. Siobhan Howard and Dr. Eanna O'Leary for being so helpful and generous with your advice.

Sean, thank you for your patience and support. I appreciate you putting up with me, and I'm looking forward to our future together without the distraction of my thesis. Fergus, Sinead, and Brian, thanks a million for everything you have done for me. You have always inspired me, and your support and encouragement has meant a lot. I couldn't ask for better siblings. Thanks also to Clare and Michael for being great hosts on my data collection treks! To my amazing parents, Pat and Martha – I don't think I have words to express how grateful I am to you both. I don't think I'll ever be able to repay you for all that you have done for me (don't worry though, I will try!). In many ways, this research would not be possible without you. For your unwavering support and all that you continue to do for me, a massive thank you!

I would also like to thank my friends for all the encouragement and distractions along the way. Thank you to those of you who put me up or helped me at any stage of my research. I'm really looking forward to having time to catch up with everyone now. A very special thank you to my Ph.D. friends for your friendship and amazing moral support. I'll miss the office banter!

I would also like to acknowledge and thank the many services and support groups that helped me recruit participants. And last, but definitely not least, I would like to extend my gratitude to all the parents and families who took part in this research. I know many of you are extremely busy, and if you had not been so generous and flexible with your time, this research would not have been possible. Thank you so much!

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List of Acronyms

ABA:	Applied Behaviour Analysis
ABP:	Ambulatory blood pressure
ABPM:	Ambulatory blood pressure monitoring
ACT:	Acceptance and commitment therapy
ADHD:	Attention deficit hyperactivity disorder
ASD:	Autism spectrum disorder
AUC_G:	Area under the curve with respect to ground
AUC_I:	Area under the curve with respect to increase
BMI:	Body mass index
BP:	Blood pressure
BPI:	Behaviour Problems Inventory
bpm:	Beats per minute
CAR:	Cortisol awakening response
CFS:	Chronic fatigue syndrome
CPRS:	Conners Parent Rating Scale
CSHQ:	Child Sleep Habits Questionnaire
CV:	Cardiovascular
DBP:	Diastolic blood pressure
DR:	Defensive responding
DSM:	Diagnostic and Statistical Manual of Mental Disorders
GARS:	Gilliam Autism Rating Scale
HADS:	Hospital Anxiety and Depression Scale
HADS-A:	Hospital Anxiety and Depression Scale – Anxiety subscale
HADS-D:	Hospital Anxiety and Depression Scale – Depression subscale
HPA-axis:	Hypothalamic-pituitary-adrenal axis
HR:	Heart rate
IBS:	Irritable bowel syndrome
ID:	Intellectual disability
MAP:	Mean arterial pressure
mmHg:	Millimetres of mercury
ODD:	Oppositional defiant disorder
OTC:	Over-the-counter

PCDI:	Parent-child dysfunctional interaction
PD:	Parental distress
PDD-NOS:	Pervasive developmental disorder-not otherwise specified
PP:	Pulse pressure
PNS:	Parasympathetic nervous system
PRS:	Parenting Responsibility Scale
PSI:	Parenting Stress Index
PSQI:	Pittsburgh Sleep Quality Index
PTSD:	Posttraumatic stress disorder
sAA:	Salivary alpha-amylase
SBP:	Systolic blood pressure
SCD:	Social Communication Disorder
SIB:	Self-injurious behaviour
SNS:	Sympathetic nervous system
SOS:	Salimetrics oral swab
SSQ:	Social Support Questionnaire
U/mL:	Units per millilitre
μg/dL:	Micrograms per decilitre

Publications and Conference Presentations Resulting from this Thesis

Publications

1. **Foody, C.,** James, J. E., & Leader, G. (2014). Parenting stress, salivary biomarkers, and ambulatory blood pressure in mothers of children with Autism Spectrum Disorders. *Research in Autism Spectrum Disorders, 8,* 99-110. doi:10.1016/j.rasd.2013.10.015 (Chapter 2)
2. **Foody, C.,** James, J. E., & Leader, G. (2013). *Parenting stress, salivary biomarkers, and ambulatory blood pressure: A comparison between mothers and fathers of children with Autism Spectrum Disorders.* Manuscript submitted for publication. (Chapter 3)

Presentations

1. **Foody, C.,** James, J. E., & Leader, G. *Biomarkers of stress in mothers of children with Autism Spectrum Disorders*. Paper presented at 34th Stress and Anxiety Research Society Conference, Faro, Portugal, July 2013.
2. **Foody, C.,** James, J. E., & Leader, G. *Stress and health in parents of children with Autism Spectrum Disorders*. Paper presented at 2nd International Conference on Autism Spectrum Disorders: From Research to Practice, Galway, Ireland, June 2013.
3. **Foody, C.,** James, J. E., & Leader, G. *Biomarkers of stress in parents of children with Autism Spectrum Disorders*. Poster presented at the Psychological Society of Ireland, Annual Conference of the Division of Health Psychology, Dublin, Ireland, May 2013.
4. **Foody, C.,** James, J. E., & Leader, G. *Stress and health in caregivers of children with Autism Spectrum Disorders.* Paper presented at the Psychological Society of Ireland, 7th Annual Conference of the Division of Behaviour Analysis, Galway, Ireland, April 2013.

5. **Foody, C.,** James, J. E., & Leader, G. *Biomarkers of stress in caregivers of children with Autism Spectrum Disorders: A preliminary analysis*. Poster presented at the Association for Behavior Analysis International, 38th Annual Convention, Seattle, Washington, May 2012.
6. **Foody, C.,** James, J. E., & Leader, G. *Biomarkers of stress in caregivers of children with Autism Spectrum Disorders: A preliminary analysis*. Paper presented at the Psychological Society of Ireland, 6th Annual Conference of the Division of Behaviour Analysis, Dublin, Ireland, April 2012.

Chapter 1

Literature Review

Autism Spectrum Disorders and Parenting Stress

Being a parent is inherently stressful, because of normative events that parents can encounter (Glasberg, Martins, & Harris, 2006). However, parenting a child with special needs is likely to be associated with additional challenges. Of all the childhood developmental disabilities, Autism Spectrum Disorders (ASD) have been found to give rise to one of the most complex networks of stressors for parents (Randall & Parker, 1999). ASDs are chronic, neurobiologically-based conditions of childhood onset that are characterised by varying levels of dysfunction in the areas of socialisation, communication, and restricted or stereotyped behaviours or interests (American Psychiatric Association; APA, 2000; APA, 2013). ASDs are extremely prevalent. The Centers for Disease Control and Prevention (2012) recently estimated that the prevalence of ASD in the United States (U.S.) is 1 in 88 children, with diagnoses more common among boys (1 in 54) than girls (1 in 252). Similar prevalence rates have recently been reported in Ireland, with approximately 1% of Irish children believed to have an ASD (Dublin City University, 2013). ASD diagnoses have increased significantly in recent years, though it is unclear if this is due to an actual increase in the prevalence of ASD, or better diagnosis and awareness (Prior, 2003). As a result, it is arguably more important than ever to consider the effects, if any, of stress related to caring for a child with ASD on parental health and wellbeing.

Past research has found that having a child with ASD can be a source of stress for parents. Caregivers of children with ASD, especially mothers, have been found to experience higher levels of stress than both the general population and parents of children with other developmental disabilities (e.g., Bouma & Schweitzer, 1990; Duarte, Borden, Yazigi, & Mooney, 2005; Estes, Munson, Dawson, Koehler, Zhou,

& Abbott, 2009; Hayes & Watson, 2013; Hoffman, Sweeney, Hodge, Lopez-Wagner, & Looney, 2009; McKinney & Peterson, 1987; Rao & Beidel, 2009). Eisenhower, Baker, and Blacher (2005) found that parents of children with ASD experienced higher stress levels than parents of children with Down syndrome, cerebral palsy, undifferentiated developmental delay, and typical development, even after accounting for differences in behaviour problems and cognitive level. There are many potential deficits and additional diagnoses associated with an ASD diagnosis, meaning that there are many different facets of the disorder that may be associated with, or contribute to, parenting stress.

DSM-IV-TR and DSM-5 Differences

Before discussing stress related to parents of children with ASD in further detail, it is important to point out that there has been a recent change in the Diagnostic and Statistical Manual's (DSM) classification of ASD. According to the DSM-IV-TR, ASD referred to three of five pervasive developmental disorders: autism, Asperger's syndrome, and pervasive developmental disorder not otherwise specified (PDD-NOS; APA, 2000). The two other pervasive developmental disorders (i.e., Rett syndrome and childhood disintegrative disorder) are not included under the ASD umbrella. Using the DSM-IV-TR, a diagnosis of an ASD was made based on deficits in three domain areas, known as the triad of impairments: socialisation, communication, and repetitive or stereotyped behaviours or interests. However, in the recently released DSM-5 (APA, 2013), the three ASDs (i.e., autism, Asperger's syndrome, and PDD-NOS) were folded into a single disorder, now known as Autism Spectrum Disorder (ASD). Thus, Asperger's syndrome is now a level of the single diagnostic criteria of ASD, rather than a distinct disorder. Furthermore, the triad of impairments was collapsed into two domains: a new combined social communication domain, in addition to the domain of repetitive or stereotyped behaviours or interests (APA, 2013). The DSM-5 introduced a new social communication disorder (SCD), allowing for the diagnosis of a disability in social communication without the presence of repetitive behaviour (APA, 2013). Furthermore, this means an individual can have a diagnosis of ASD with or without a language disorder (Lai, Lombardo, Chakrabarti, & Baron-Cohen, 2013). Finally, the DSM-5 recommends the use of "specifiers", to record the severity of additional symptoms, current language and intellectual ability, onset age and pattern, and genetic/medical or environmental/acquired conditions (Lai et al, 2013). However, given that the present research was conducted prior to the introduction of the DSM-5, and the fact that ASD diagnoses obtained by the children of the participants in this thesis were based on the DSM-IV-TR criteria, the present thesis will focus on the DSM-IV-TR classification of ASD. The term ASD will be used throughout the thesis to refer to individuals diagnosed with autism, Asperger's syndrome, or PDD-NOS under the DSM-IV-TR criteria.

Core Deficits

Socialisation. Some researchers have suggested that impairments in socialisation may be the primary deficit for individuals with ASD (Fein, Waterhouse, Lucci, & Snyder, 1985). Social deficits are often diverse. They can involve speech, linguistic conventions, and interpersonal interaction, while problems such as eye-contact, turn-taking, speech prosody, interpreting emotions, and nonliteral language are common among individuals with ASD (White, Keonig, & Scahill, 2007). Impairments in socialisation can often result in a failure to develop peer relationships appropriate to developmental level. This may be due to a lack of interest in developing such friendships, or an inability to establish or sustain friendships because of difficulties understanding social conventions or lacking shared interests (APA, 2000). Particularly for individuals with ASD who *desire* to make friendships, socialisation deficits may create loneliness and isolation, with sadness reported to be common among individuals with high-functioning autism (Ghaziuddin, Ghaziuddin, & Greden, 2002). Additionally, poor socialisation skills can contribute to peer rejection, isolation, academic and occupational underachievement, and mood and anxiety problems among individuals with ASD (White et al., 2007). These factors can also be difficult for parents. In a review of parental perceptions and needs, parents of children with ASD rated lack of interaction and play (reported by 94% of parents) and deficits in social skills (reported by 93% of parents) as the most difficult child behaviours (Dillenburger, Keenan, Doherty, Byrne, & Gallagher, 2010). Furthermore, social relatedness has previously been reported to be associated with overall parenting stress, parent-child relationship problems, and parental distress among mothers and fathers of children with ASD (Davis & Carter, 2008).

Communication. A communication deficit is often one of the first warning signs for a parent that their child may have an ASD (Hall & Graff, 2011). Although the DSM-5 removed the need for a communication delay from the ASD diagnostic criteria, communication can be markedly impaired for many individuals with ASD. Many individuals may have a delay in, or lack of, development of oral language, difficulty initiating or sustaining a conversation, or stereotyped or repetitive uses of language (APA, 2000). Individuals with ASD may also have difficulty with receptive language, or may have immature grammatical structures or abnormal pitch (APA, 2000). Problems with communication may also be more subtle. Individuals with ASD often rely on prompts to elicit their communication, and the development of spontaneous speech can be challenging (Greer & Ross, 2004). There may also be difficulties with understanding and expressing emotions, while nonliteral language, such as sarcasm, facial expressions, and metaphors can be problematic (White et al., 2007).

Communication deficits can have a number of significant implications for individuals with ASD. Research has found that a child's pre-intervention language levels may be linked to their progress, with children who have higher pre-intervention language demonstrating higher gains than those with lower pre-intervention language (Magiati, Charman, & Howlin, 2007). Communication deficits may also contribute to the development of challenging behaviour as a result of frustration (Sicile-Kara, 2003). Challenging behaviour may also replace speech as a mode of communication for some individuals with ASD (Chiang, 2008). Communication deficits can also contribute to individuals with ASD requiring more supervision. Difficulties with receptive communication can create safety concerns, because some individuals with ASD may fail to recognise threats and dangers (Norton & Drew, 1994). Communication skills have been reported to predict the extent of independence or supervision that individuals with ASD will require in adulthood (Hall & Graff, 2011). In Dillenburger et al.'s (2010) study on parental perceptions and needs, 93% of parents rated language and communication deficits as a difficulty.

Stereotyped or repetitive behaviour. Another core feature of ASD is the presence of repetitive or stereotyped behaviours or interests. This can include abnormally intense stereotyped and restricted patterns of interest, inflexible adherence to specific, non-functional routines or rituals, stereotyped and repetitive motor mannerisms, or persistent preoccupation with parts of objects (APA, 2000). For instance, many individuals with ASD are inflexible and may have difficulty with changes in their routine (McClannahan & Krantz, 1999). Others may engage in vocal (e.g., humming) or motor stereotypy (e.g., hand flapping or rocking; Singer, 2009). This area of impairment can cause huge difficulty across an individual’s life. As discussed above, although some individuals with ASD may lack the desire to engage in social interactions, others may have the desire, but may lack the skills to initiate or sustain conversation. At times, this may arise from a lack of shared interests or difficulty in diverting from a specific interest that the individual may wish to discuss. Mayes, Calhoun, Mayes, and Molitoris (2012) found that children with ASD had a significantly higher frequency of selective attention (98% of individuals in the sample) than children with ADHD (21% of individuals in the sample). Children with ASD had the ability to “hyper-focus” on activities of interest to them. While this could be a strength in terms of developing a particular vocation or hobby, it also has the potential to create challenges in socialisation given that conversations require a certain amount of give and take.

Higher levels of stereotyped behaviour have been found to affect skill acquisition, decrease opportunities for positive interactions, and can be stigmatising (MacDonald et al., 2007). Furthermore, stereotypy has previously been identified as a potential source of stress for parents of children with ASD (e.g., Davis & Carter, 2008; Gabriels, Cuccaro, Hill, Ivers, & Goldson, 2005). For some individuals, even slight changes in routine may cause serious difficulty, such as tantrums (Norton & Drew, 1994). As disruptions and changes in routine are a common part of everyday life, the need for strict routine can make it difficult for parents to manage a normal daily lifestyle. Difficulty with waiting, which is very common for individuals with ASD, can also create issues for families. For instance, parents may avoid certain public outings or events due to problems with waiting and changes in routines.

Dillenburger et al. (2010) found that 86% of parents they surveyed reported restrictions on social and community activities, and 85% reported restrictions on recreational and leisure activities since having a child with ASD.

ASD severity. Because ASD is a spectrum disorder, individuals on the spectrum can experience different deficits or symptoms, and the severity of symptoms can vary. ASD severity may be a significant predictor of caregiver stress, although there is some discrepancy in the literature. Stuart and McGrew (2009) explored caregiver burden in mothers and fathers after receiving an ASD diagnosis for their child. They found a moderate relationship between symptom severity and both independent and family burden, although not with marital burden. Hastings and Johnson (2001) also explored predictors of stress in parents of young children with ASD in intensive home-based behavioural intervention. They reported that higher ASD severity was associated with higher levels of parental stress. Osborne, McHugh, Saunders, and Reed (2008) found that when a child was younger at baseline, ASD severity was the best predictor of parenting stress, but no strong relationship between ASD severity and stress was found with an older sample. This suggests that the relationship between ASD severity and parenting stress may be restricted to parents of younger children. However, in another study exploring well-being in parents of children with ASD, it was found that neither maternal nor paternal stress was positively correlated with ASD severity (Hastings et al., 2005b). Further research is needed to investigate the role ASD severity plays in stress and well-being among parents of children with ASD.

Associated Difficulties and Diagnoses

In addition to the core deficits of ASD, there are a range of associated symptoms and comorbid diagnoses that are commonly experienced by individuals with ASD.

Comorbidity refers to the occurrence of two or more forms of psychopathology in the same person (Matson & Nebel-Schwalm, 2007). One study reported that 70% of children with ASD in their sample had at least one comorbid diagnosis, with 41% reported to have two or more comorbid diagnoses (Simonoff et al., 2008). Additional difficulties or disorders that individuals with ASD may experience include, but are not limited to, intellectual disability (ID; Malfa, Lassi, Bertelli, Salvini, & Placidi, 2004), attention deficit hyperactivity disorder (ADHD; Gillberg et al., 2004), oppositional defiant disorder (ODD; Simonoff et al., 2008), mood disorders, such as depression (Kim, Szatmari, Bryson, Streiner, & Wilson, 2000), dyspraxia (Mind, Brimacombe, & Wagner, 2007), sensory issues (Smith, Mruzek, & Mozingo, 2010), adaptive skills deficits (Matson, Hattier, & Belva, 2012), challenging behaviour (Murphy, Healy, & Leader, 2009), sleeping problems (Richdale, 2001), gastrointestinal (GI) issues (Valicenti-McDermott, McVicar, Cohen, Wershil, & Shinnar, 2006), and seizure disorders (Tuchman & Rapin, 2002). This long, but not comprehensive, list of symptoms and disorders that are often associated with ASD illustrates the broad range of difficulties that may be experienced by individuals with ASD, as well as their parents.

Though these additional symptoms or disorders are not part of the ASD diagnostic criteria, many of these additional challenges have been linked with parental stress among parents of children with ASD. For instance, it has been widely documented that there is a relationship between child behaviour problems and parental stress. Eisenhower et al. (2005) found that behaviour differences over time were paralleled by differences in maternal stress, such that mothers of children with ASD were found to be at elevated risk of experiencing high stress. Baker et al. (2003) also found an interaction between parenting stress and behaviour problems over time among parents of children with developmental delays. High parenting stress contributed to a worsening of child behaviour problems over time, while higher behaviour problems contributed to a worsening of parenting stress. Additionally, in Dillenburger et al.'s (2010) review of parental perceptions and needs, parents identified a number of issues that were not core features of ASD as difficulties. These included challenging behaviour (reported by 90% of parents), adaptive behaviour deficits (reported by 88% of parents), erratic sleep patterns (reported by 80% of parents), and ADHD (reported by 22% of parents). Comorbid diagnoses can cause significant impairment for individuals with ASD, and can contribute additional burden on parents of children with ASD (Leyfer et al., 2006). Thus, in addition to the core deficits associated with ASD, research indicates that it is important to also consider the effects of additional symptoms and diagnoses on parental stress and wellbeing.

Additional Stressors

In addition to the numerous disorders and symptoms that may be experienced by individuals with ASD, other aspects of the disorder may also make it particularly challenging for both the individual and parent. Issues in detecting and diagnosing ASD can arise, because diagnosis relies exclusively on assessment of behavioural symptoms (National Institute of Mental Health, 2010). There is often a delay of between 2–4 years between a parent first detecting a problem and receiving a diagnosis of an ASD (Howlin & Moorf, 1997). Research has reported that, while parents tend to notice ASD symptoms at a very young age, many parents only seek professional help several months after detecting a problem (Matson, Rieske, & Tureck, 2011). Recognising that a child is not meeting developmental milestones may be particularly difficult if the child is the first in a family, and there is no other child in the family for comparison of developmental milestones. Furthermore, diagnostic overshadowing or misdiagnosis can occur, particularly if the individual has comorbid disorders such as ADHD or ODD (Simonoff et al., 2008). Misdiagnosis of ASD is particularly likely among girls (Wing, Gould, & Gillberg, 2011). The diagnostic process can be a particularly stressful time for parents, and many studies have identified parental dissatisfaction with the diagnostic process and the information and support the parent received following the diagnosis (e.g., Brogan & Knussen, 2003; deAlba & Goldfish, 2011; Moh & Magiati, 2012; Osborne & Reed, 2008).

Research has also found that parents may end up feeling guilt or blaming themselves for the disorder (Casey et al., 2012). The guilt that may arise as a result of a diagnosis of ASD is often due to the fact that there is no known cause for the disorder. As mentioned, this can contribute to difficulties in obtaining a diagnosis, but it can also lead to confusion and guilt, and can result in parents taking ownership of the diagnosis (Casey et al., 2012). Numerous theories have been proposed to explain the origins of ASD, many of which may be upsetting for parents, such as the refrigerator mother theory (Bettelheim, 1967), and the MMR (measles, mumps, and rubella) vaccination theory (Wakefield et al., 1998). The constant barrage of contradictory theories regarding the cause of ASD is likely to be very difficult for parents (Herbert, Sharp, & Gaudiano, 2002).

The lack of a known cause of ASD also means that there is no cure, and this has opened up the ASD field to a multitude of interventions. Many interventions are pseudoscientific (i.e., lacking a scientific basis and empirical support, but giving the appearance that they are scientific) in nature (Vyse, 2010). Some pseudoscientific interventions can cause serious damage (e.g., chelation therapy has caused death in some cases); others have been found to produce little or no gains for individuals with ASD, wasting time that a child could have spent receiving other evidence-based interventions (Jacobson, Foxx, & Mulick, 2010). The bombardment of parents with different treatment options may make selection of the most appropriate intervention very difficult. Miller, Schreck, Mulick, and Butter (2012) studied treatment choices among parents of children with ASD, and slightly less than half the parents reported choosing evidence-based treatments. Many reported choosing unsupported treatments, such as music therapy or vitamin therapy. Additional difficulties can arise for parents if they do decide to utilise evidence-based interventions. Currently, Applied Behaviour Analysis (ABA) is the only empirically supported intervention for individuals with ASD (e.g., Eldevik et al., 2009; Ministers' Autism Spectrum Disorders Reference Group, 2007; U.S. Department of Health and Human Services, 1999). However, many parents may find themselves facing financial burden if they wish to avail of ABA for their child, as there is currently no insurance coverage for ABA in Ireland (Keenan, 2012). This is particularly problematic given that many parents often cannot continue to work full-time when they have a child with ASD due to the commitment required in caring for the child (Dillenburger et al., 2010). These additional factors underscore the enormity and range of challenges that may be associated with parenting a child with ASD.

Stress and Health

As discussed above, research has demonstrated that parents of children with ASD often report higher levels of stress than parents of typically developing children (e.g., Eisenhower et al., 2005; Hayes & Watson, 2013). Thus, it follows logically that parents of children with ASD may be at higher risk of health problems associated with chronic stress related to their parenting roles. Indeed, parents of children with ASD have reported poorer health than parents of typically developing children. Eisenhower et al. (2005) found that mothers of children with developmental delays at age 3 reported poorer concurrent and later physical health than mothers of typically developing children. Allik, Larsson, and Smedje (2006) also found that mothers of children with Asperger’s syndrome or high-functioning autism reported poorer physical health than parents of typically developing children. Similarly, Smith, Seltzer, and Greenberg (2012) found that mothers of children with ASD reported a higher proportion of days with headaches, backache, muscle soreness, fatigue, and hot flashes than mothers of children without ASD. Additionally, parents of children with ASD have been found to report poorer health than parents of children with other disabilities. For example, Mugno, Ruta, D’Arrigo, and Mazzone (2007) found that mothers of children with ASD reported poorer physical health, psychological state, and quality of life than parents of children with cerebral palsy, ID, and typically developing children. However, very little research into physiological indices has been conducted to support the parent-report literature on stress, and to identify the mechanisms by which parents of children with ASD may be at risk of poorer health. In order to understand the importance of investigating physiological measures, it is necessary to understand the physiology of stress and its relevance to health.

Selye (1979) defined stress as the non-specific response of the body to any demand made upon it (as cited in Vedhara, Shanks, Anderson, & Lichtman, 2000). It is a process that consists of stressors (i.e., challenging events), mediators (i.e., constructs that enable us to evaluate the nature of a threat and the emotional and behavioural response elicited by that threat), and the stress-response (i.e., physical and emotional responses elicited by a stressor; Vedhara et al., 2000). Different presentation of stressors can influence the body's ability to deal with such stressors. The human body is extremely effective in responding to acute (i.e., events that require an immediate physiological response to survive) and chronic (i.e., events that require a sustained response) *physical* challenges (Sapolsky, 2004). However, prolonged *psychological* stress can be damaging (Loft et al., 2007). Psychological stressors are events that are generated and dealt with entirely in our heads and do not require a physiological response or reaction, yet these stressors can activate the same physiological stress-response that is triggered in response to physical crises (Sapolsky, 2004). In order to maintain optimal bodily functioning, a process known as *homeostasis* regulates internal stability in response to environmental changes (Lovallo, 2005). However, if a demand or stressor is not removed (i.e., in situations where there is chronic stress), the process of homeostasis may be a source of wear and tear on the system, resulting in *allostatic load* (McEwen & Stellar, 1993). In other words, the system may respond to an *acute* stressor by restoring homeostatic balance with no associated costs, but the process of trying to maintain homeostatic balance in response to a *chronic* stressor can result in physiological exhaustion (Sapolsky, 2004).

The Stress-Response

The stress-response is the body's attempt to restore allostasis (Sapolsky, 2004). The overall function of the stress-response is to quickly mobilise energy from storage sites, and to prevent further storage in order to prepare the body to engage in a fight-or-flight response (Sapolsky, 2004). It consists of numerous physiological reactions that include the nervous, endocrine, and cardiovascular (CV) systems, and this same stress-response is activated in response to both physical and psychological stressors (Pinel, 2003). In fact, appraisal of a stimulus as either a stressor or a nonstressful stimulus is an important component in the activation of the stress-response (Sapolsky, 2004).

The nervous system. An important function of the nervous system is to control perception. Appraisal of demands is an important element of the stress-response, and can be affected by many factors, such as an individual's emotional state or the meaning they attach to a stressor (Aiello & Kaplan, 2009). Lazarus and Folkman (1984) devised a two-level appraisal model of psychological stress, which posits that we first evaluate events for their threat value, and then evaluate the options for coping with any perceived threat (as cited in Lovallo, 2005). This in turn determines our cognitive, behavioural, emotional, neurophysiological, autonomic, and endocrine responses to external events. These stress responses that are based on our perceptions of an event as being stressful are referred to as psychological stress responses, because the threat primarily depends on our interpretation of the event (Lovallo, 2005). Psychological stressors involve primary appraisals, emotions, physiological responses, and coping mechanisms (Lovallo, 2005).

Once we perceive a situation as stressful, the autonomic nervous system (ANS) responds (Turner, 1994). The ANS is separated into two branches, which have opposing roles: the sympathetic nervous system (SNS) is activated in response to stress, while the parasympathetic nervous system (PNS) is suppressed (Sapolsky, 2004). The role of the PNS is to calm the body, absorb nutrients, conserve energy, and promote growth and other important processes (Turner, 1994). The SNS is the branch that is responsible for mobilising the body for action, and has a crucial role to play in the fight-or-flight response during times of stress (Lovallo, 2005). This system promotes vigilance, arousal, activation, and mobilisation (Sapolsky, 2004). Activation of the SNS triggers the release of epinephrine (adrenaline) and norepinephrine (noradrenaline), both of which induce increased heart rate (HR) and myocardial contractile force (Turner, 1994). During the stress-response, hormones are also secreted by the endocrine system.

The endocrine system. The other major system activated during a stress-response is the hypothalamic-pituitary-adrenal (HPA) axis, which triggers the production of the hormone adrenocorticotropic hormone (ACTH), which then stimulates the production of the stress hormone, cortisol (Kalat, 2004). Cortisol plays an important role in both normal states and during stress, and stress levels of cortisol support SNS activities that increase the release of stored glucose and fats (Lovallo, 2005). However, prolonged elevation of cortisol can become harmful (Kalat, 2004). High, repeated, or prolonged levels of cortisol can increase the sensitivity of the amygdala to potential stressors, thus increasing stress reactivity (Lovallo, 2005). This may help to explain psychosomatic illnesses that are associated with periods of stress, such as irritable bowel syndrome (IBS; Lovallo, 2005). The combined effects of the hormones released during a stress-response generate energy in the body by increasing blood pressure (BP), HR, and blood flow, thereby converting and releasing stored sugar from the liver into the bloodstream and increasing the body’s metabolism (Turner, 1994).

The cardiovascular system. The actions of the nervous and endocrine systems combine with the CV system to produce the pattern of activation referred to as the fight-or-flight response to stressors (Turner, 1994). During the stress-response, CV functioning is altered: HR is increased by suppressing parasympathetic tone and activating the SNS; glucocorticoids stimulate sympathetic arousal and enhance the effects of epinephrine on heart muscle; the force of the heart beat is increased by the SNS constricting veins; blood flow to muscles is increased while blood flow to nonessential body parts such as the digestive tract is decreased; and water is conserved by decreasing blood flow to the kidneys (Sapolsky, 2004). Once the stressor has passed, functioning in all of the systems should return to normal.

Stress and immunity. The immune system has an important role in defending the body against infectious agents such as parasites, bacteria, viruses, and fungi (Sapolsky, 2004). The immune system must recognise antigens, neutralise them and remember them for future attacks, and must also be capable of distinguishing between normal parts of the body (self) and invading cells (non-self; Lovallo, 2005; Sapolsky, 2004). Early in the stress-response, the immune system is activated and enhanced by glucocorticoid and SNS activation; but, because of the dangers of developing autoimmunity if the immune system is enhanced for too long, glucocorticoid exposure then suppresses immunity and brings it back to baseline (Sapolsky, 2004). Problems arise when stressors are prolonged, because the system then plummets to immunosuppression (Sapolsky, 2004). Thus, prolonged activation of the stress-response and the associated increase in cortisol and SNS activity can suppress immune system activation, consequently lowering the body's response to viral challenge (Lovallo, 2005). Psychological stress has been identified as an important risk factor for developing upper respiratory tract infections and has been linked to incidences of colds and flus (Reid, Mackinnon, & Drummond, 2001). Previous research has reported that parents of children with developmental disabilities (DD) have poorer antibody responses to vaccination than control participants (Gallagher, Phillips, Drayson, & Carroll, 2009).

Parental Stress and Health

Attempts to maintain allostasis long-term as a result of chronic psychological stress can produce wear and tear, and can become damaging to the body. In fact, with prolonged stress, the stress-response can become more damaging than the stressor itself, particularly when the stressor is purely psychological in nature (Sapolsky, 2004). Chronic activation of the stress-response can increase the risk of developing fatigue, hypertension (i.e., high BP), CV disease, peptic ulcers, reproductive disorders, cognitive decline, and damage to immune function, resulting in vulnerability to infectious diseases and less capability of combating them once they are acquired (Sapolsky, 2004). This gives some indication of the physiological responses that could potentially occur among parents of children with ASD, who are known to be at high risk of experiencing chronic stress. However, self-reported stress may not positively correlate with physiological measures, and individuals who report the highest levels of stress may not necessarily be those demonstrating the greatest negative outcomes (Romanczyk & Gillis, 2006). Therefore, it is important to directly measure physiological parameters of stress when investigating the physiological effects of caring for a child with ASD. There are a number of areas, in particular, that may be important to consider for these parents.

Cardiovascular activity. When discussing stress-related disease, the link between stress, hypertension, and CV disease is of particular health concern. There is some discrepancy about the ideal BP. Some people consider BP of below 140 mmHg/90 mmHg to be normotensive and above 150 mmHg/90 mmHg to be hypertensive, but any elevation in BP has been found to decrease life expectancy (Turner, 1994). Stress is a significant risk factor for hypertension, which is the single most important risk factor for stroke and is an extremely important predictor of CV disease (World Heart Federation; WHF, 2013). CV diseases are the number one group of conditions that cause death globally (World Health Organization; WHO, 2008). CV disease is a wide-ranging immune-mediated condition that includes coronary heart disease and atherosclerosis (Sapolsky, 2004). While chronic stress can contribute to the development of conditions such as hypertension and atherosclerosis, acute stressors can trigger events such as myocardial infarction and angina pectoris in a person already suffering from myocardial ischemia (Sapolsky, 2004). Therefore, it is vital to identify individuals that are at risk of developing such life-threatening diseases.

As there is a great deal of evidence to suggest a link between large stress responses and later development of CV disease, there is an emphasis in the literature on measuring CV responses to psychological stressors in a laboratory setting (Turner, 1994). Cardiovascular reactivity (CVR) to stress refers to the change in physiological activity from that observed at rest to that observed during a stressful challenge, which may be either psychological or physical in nature (Obrist, 1981). Research has shown that CVR to psychological stress is a significant independent risk factor for developing CV disease (Blascovich & Katkin, 1993). People are generally regarded as being at higher risk for developing CV disease if they show stress-induced hemodynamic changes that are of relatively large magnitude and are prolonged (Gianaros & Sheu, 2009). However, because of the structured nature of laboratory-based studies, they may not be representative of how people typically respond to stressors (Turner, 1994). Furthermore, as the crucial link between stress and health may be related to processes of adaptation or exhaustion that can take up to hours or days to emerge, is has been suggested that ambulatory blood pressure (ABP) monitoring over a 24 hour period may be useful for uncovering these processes in real-life situations (Klaver, deGeus, & deVries, 1994).

ABP monitoring is an extremely useful tool for determining the impact of real-life stressors on an individual's CV activity. It enables repeated observations of BP and HR under natural conditions. BP and HR can fluctuate over a 24-hour period for a number of reasons. They are regulated by complex physiological mechanisms, and external stressors and activities can directly impact on BP and HR (Chau et al., 1989). An additional reason for fluctuation is that at day-time and night-time, BP and HR may become synchronised to the person's own circadian rhythm (Chau et al., 1989). The diurnal profile usually includes a 10–20% fall in BP during sleep, which is often referred to as "dipping" (Hansen et al., 2011). There is evidence to suggest that "non-dippers" (i.e., those who do not experience a night-time fall in BP) may be at increased risk of end-organ damage and CV complications (O'Brien et al., 2000). ABP monitoring is the only non-invasive technique that permits BP to be measured at night-time. Furthermore, ABP monitoring has been found to be superior to clinical BP in predicting CV mortality (Dolan et al., 2005).

However, although it is known that parents of children with ASD are at high risk of experiencing chronic stress, and chronic stress is linked to the development of hypertension and CV disease, very little research has investigated CV activity among parents of children with ASD. To our knowledge, only one such study has been conducted to date. Gallagher and Whiteley (2012) reported higher ambulatory CV activity among caregivers of children with developmental disabilities (DD), including ASD, than among a control group of parents of typically developing children. This study requires replication, particularly given that less than half of the parents in the DD group were parents of children with ASD. Thus, ambulatory CV activity among parents of children with ASD remains a largely unexplored area.

HPA-axis activity. As mentioned previously, the HPA-axis is activated in response to a stressor. Chronic activation of the HPA-axis is associated with a variety of health problems, such as depression, risk of CV disease, cognitive decline, and metabolic syndrome (Adam & Kumari, 2009). Activation of the HPA-axis triggers the production of the stress hormone cortisol, which is a biomarker of HPA-axis activity. Cortisol has a strong basal diurnal profile: levels are high in the morning; increase approximately 50-60% in the first 30-45 minutes after awakening (known as the cortisol awakening response or CAR); drop rapidly over the first few hours after waking; and gradually decline throughout the rest of the day, to reach a low point around midnight (Adam, Hawkley, Kudielka, & Cacioppo, 2006). There is great individual difference in cortisol diurnal profiles, and the CAR is a commonly used measure of these differences. Dysregulation of the CAR is associated with negative psychological and somatic consequences, such as depression (Clow, Thorn, Evans, & Hucklebridge, 2004). A larger CAR is frequently associated with higher current chronic stress, while low cortisol levels (i.e., hypocortisolism) or flatter diurnal rhythms can be associated with a history of exposure to stress, and disorders such as chronic fatigue syndrome (CFS) or fibromyalgia (Adam et al., 2006). For these reasons, cortisol is an important measure of HPA-axis activity and an important biomarker of potential health problems. Cortisol levels can be reliably assessed using salivary measurements. In blood, only 1-15% of cortisol is unbound to protein, which is the cortisol of interest when measuring stress (Romanczyk & Gillis, 2006). Unbound serum cortisol enters the saliva and largely remains unbound to protein (Salimetrics, 2012a). Thus, salivary cortisol is a very reliable measure and has been consistently found to be highly correlated with serum cortisol (Vining, McGinley, Maksvytis, & Ho, 1983).

Recent research has begun investigating cortisol activity among parents of children with ASD. One of the first studies did not observe any differences in cortisol levels between parents of children with ASD and parents of typically developing children (Marinovic-Curin et al., 2008). However, the majority of subsequent research has found evidence of hypocortisolism among parents of children with ASD (Dykens & Lambert, 2013; Lovell, Moss, & Wetherell, 2012, 2013; Ruiz-Robledillo, Bono, & Albiol, 2013; Seltzer et al., 2010; Wong et al., 2012). Hypocortisolism is a profile similar to that observed among individuals with posttraumatic stress disorder (PTSD; Heim, Ehlert, & Hellhammer, 2000). As mentioned previously, hypocortisolism is associated with some negative symptoms such as pain, fatigue, and high stress sensitivity, although it may also have some protective effects (Fries, Hesse, Hellhammer, & Hellhammer, 2005). Many of these studies among parents of children with ASD were based on older samples, and hypothesised that hypocortisolism may develop as a result of chronic stress related to parenting a child with ASD across the lifespan. Further research investigating levels of cortisol activity among a younger sample could add to knowledge of the effects of chronic stress on the physiology of parents. Furthermore, the majority of the studies focused either exclusively, or predominantly, on cortisol activity among mothers. Thus, further investigation and comparison of cortisol activity between mothers and fathers of children with ASD could further understanding of the different effects stress may have on mothers and fathers.

SNS activity. As mentioned previously, the SNS also has a crucial role to play in the stress-response, as it helps to mediate vigilance, arousal, and mobilisation as part of the fight-or-flight response during stress (Sapolsky, 2004). Chronic activation of the SNS is associated with a variety of health complications (Sapolsky, 2004). For instance, stress-related increases in SNS activity have been found to be associated with immune suppression, which is likely to increase susceptibility to negative health outcomes and impede an individual’s ability to combat illnesses when acquired (Granger, Kivlighan, El-Sheikh, Gordis, & Stroud, 2007). The enzyme alpha-amylase has been proposed as a marker for stress-induced activity of the SNS (e.g., Rohleder & Nater, 2009; Shetty, Zigler, Robles, Elashoff, & Yamaguchi, 2011). Numerous studies have demonstrated that alpha-amylase levels increase in response to physical and psychological stressors such as exercise, heat and cold stress, written examinations, and the Trier Social Stress Test (Granger et al., 2007). As with cortisol, salivary sampling is possible for alpha-amylase, with salivary alpha-amylase (sAA) providing a useful non-invasive measure of SNS activity (Salimetrics, 2010b). To date, however, we are unaware of any research that has investigated sAA levels among parents of children with ASD. Blunted cortisol responses and increased SNS activity have previously been reported among individuals with PTSD (Fries et al., 2005). Thus, given that there is some evidence of hypocortisolism among individuals with ASD, investigation of both cortisol and sAA levels could provide useful insight into the physiological effects of stress related to parenting a child with ASD.

Gender Differences

While this review of the research has primarily referred to parents of children with ASD as a group to this point, it is important to bear in mind that the majority of research that has been conducted in the field thus far has involved mothers. Among the studies that have included both mothers and fathers, some similarities and differences between their experiences have been observed. Hastings (2003) found that child behaviour problems did not affect paternal stress but did affect maternal stress. Mothers also reported higher levels of anxiety than fathers, although mothers and fathers did not differ in their levels of depression and stress (Hastings, 2003). Davis and Carter (2008) found that the social skills deficits of the child was the most consistent predictor of parenting stress for both mothers and fathers. They also found that behaviour problems that are not clinical manifestations of ASD were also associated with stress, but that the types of behaviours differed for mothers and fathers. They found that for mothers, their child's difficulties with self-regulation, such as eating and sleeping problems, were particularly problematic, while child externalising behaviour such as stereotypy was particularly problematic for fathers. It is also possible that the well-being of one partner may influence the other. Partner's mental health has been reported to be correlated with maternal stress, although not with paternal stress (Hastings, 2003). Further research is necessary to explore the differences between mothers and fathers, to ensure parents can be supported appropriately.

Conclusions

The preceding literature review provides an overview of the critical issues underpinning the current research. An overview of ASD and the characteristics associated with ASD provided an important background for understanding the stressors that parents may experience. In addition to the core deficits in socialisation, communication, and the presence of repetitive and stereotyped behaviours and interests, there are many additional challenges that can be experienced by individuals with ASD. Comorbid diagnoses such as ID, ADHD, mood disorders, sensory issues, challenging behaviour, deficits in adaptive skills, sleeping disorders, and medical conditions such as gastrointestinal disturbances and epilepsy are among the challenges known to be experienced by many individuals with ASD. These facets of the disorder have the potential to decrease an individual's quality of life and increase the level of care they require, thereby increasing parental burden. A review of the literature demonstrated that parenting a child with ASD can be highly challenging.

Studies found that parents of children with ASD were at higher risk of stress, depression, anxiety, and marital conflict than parents of typically developing children. Parents of children with ASD also reported poorer health than parents of typically developing children.

The review then turned to the physiological changes involved in the stress-response, which provided an important background for understanding the damaging effects that chronic stress can have on the body. The stress-response is highly adaptive and complex. It has evolved to enable the body to identify and combat stressors and to prepare the body for fight-or-flight reactions in times of physical crises. However, our ability to perceive events or stimuli as stressors has resulted in the activation of this same stress-response as a result of purely psychological stressors. It has repeatedly been shown that chronic activation of the stress-response can have a devastating impact on health, and can increase risk of diseases such as hypertension, CV disease, and peptic ulcers, particularly when the stress is purely psychological.

The review then turned to research that took into consideration the physiological effects of caring for a child with ASD on the parent. This review observed that this literature is very sparse, and there is a particular lack of research including fathers of children with ASD. Furthermore, there is very little research investigating the effects, if any, of caring for a child with ASD on CV activity. Prior research has reported evidence of hypocortisolism among parents of children with ASD. This blunted cortisol response is similar to the cortisol profile observed in individuals with PTSD and, although it may be adaptive in some ways, can be associated with health problems such as chronic fatigue, pain, and fibromyalgia. Additional research investigating cortisol levels, alongside levels of sAA, could provide important information about the physiological effects of stress on parents of children with ASD.

Thesis Objectives

The present thesis aimed to identify psychosocial variables predictive of parent self-report, salivary biomarkers of stress, and level of CV activity in parents of children with ASD. This included factors that could exacerbate (e.g., child sleeping problems) or alleviate (e.g., social support) parenting stress. Regarding salivary biomarkers, the aim was to examine whether parents of children with ASD have blunted cortisol profiles or elevated sAA levels. Furthermore, the thesis aimed to identify differences in parent self-reports and physiological markers of stress between mothers and fathers, and between parents of children with and without ASD. Three inter-related studies were conducted. A within-subjects analysis was conducted to identify predictors of parent self-report, salivary biomarkers of stress, and CV activity (Chapter 2). Subsequently, the effects of gender and level of caregiving responsibility on caregiver stress and wellbeing were investigated, with a comparison of mothers and fathers of children with ASD (Chapter 3). Finally, in order to control for stress related to normative parenting, stress measures were compared between parents of children with ASD and a matched control group of parents of typically developing children (Chapter 4).

Chapter 2: Study 1

Biomarkers of Stress in Mothers of Children with Autism Spectrum Disorders[1]

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[1] Elements of this chapter have been accepted for publication: Foody, C., James, J. E., & Leader, G. (2014). Parenting stress, salivary biomarkers, and ambulatory blood pressure in mothers of children with Autism Spectrum Disorders. *Research in Autism Spectrum Disorders, 8,* 99-100. doi:10.1016/j.rasd.2013.10.015

Parenting is often stressful, but research shows that parents of children with ASD can experience particularly pronounced levels of stress (e.g., Eisenhower et al., 2005; Randall & Parker, 1999). Numerous psychosocial factors associated with stress among parents of children with ASD have been explored. For instance, it has been widely documented that behaviour problems in children can affect parental stress levels. Eisenhower et al. (2005) found that behaviour differences over time were paralleled by differences in maternal stress, such that mothers of children with ASD were at elevated risk for high stress. Other variables found to exacerbate stress levels in this population include earlier diagnosis (Osborne et al., 2008), lower child adaptive functioning (Hall & Graff, 2011), and an increased number of unmet service needs (Taylor & Seltzer, 2010). However, there are inconsistencies in the literature regarding which variables have the most significant impact on parental stress, which may be partly due to over-reliance in previous research on parent self-reports of stress, and parent reports of child and parent characteristics.

Cortisol

Use of physiological measurement has been recommended for studies of stress (Romanczyk & Gillis, 2006). A biomarker of HPA-axis activity, the stress hormone cortisol, can be measured non-invasively from saliva samples. The HPA-axis is activated in response to stress, triggering cortisol production. Chronic activation of the HPA-axis can increase reactivity to stressors and is associated with increased risk of health problems (Lovallo, 2005). Cortisol has a strong diurnal profile, with levels increasing approximately 50–60% in the first 30–45 minutes after waking (known as the cortisol awakening response; CAR), and gradually declining throughout the rest of the day (Adam & Kumari, 2009). Dysregulation of the CAR is associated with negative outcomes, such as depression (Clow et al., 2004). Seltzer et al. (2010) found that mothers of adolescents and adults with ASD had significantly lower levels of daily cortisol than mothers in a control group. Low cortisol, or hypocortisolism, can result in health problems such as decreased immunity and increased vulnerability to stress-related diseases (Heim et al., 2000). Further research is needed to determine if mothers of younger children and adolescents with ASD also experience hypocortisolism.

Alpha-Amylase

The SNS also has a crucial role in the stress-response, especially in the mediation of arousal and physiological mobilisation as part of the fight-or-flight response (Sapolsky, 2004). Chronic activation of the SNS is associated with a variety of health problems, such as immune suppression (Granger et al., 2007). Previous research has reported reduced immunity among parents of children with developmental disability (DD), including ASD (Gallagher et al., 2009). SAA has been identified as a marker for stress-induced activity of the SNS (Granger et al., 2007; Rohleder & Nater, 2009). Thus, saliva samples can be used to assess both major stress systems (i.e., the HPA-axis and SNS) among parents of children with ASD.

ABP Monitoring

The CV system is also activated during the stress-response. During a maximum stress response, the heart’s output of blood can increase by up to five times the resting state (Clow, 2001). Chronic stress is a significant risk factor for the development of hypertension, and hypertension is a risk factor for future development of stroke and coronary heart disease (Sapolsky, 2004). Laboratory-based studies provide useful information about CV responses to stressors. However, they may not be representative of how people typically behave or respond to stressors, because of their highly structured nature (Turner, 1994). ABP monitoring enables repeated measurement of BP and HR under natural conditions. A portable monitor is worn, usually over a 24-hour period, and it measures BP and HR at regular intervals while a person continues their typical daily routine. ABP has also been reported to be superior to clinical BP in predicting CV mortality (Dolan et al., 2005). Thus, ABP monitoring could provide an important naturalistic measure of CV responses to stressors experienced by parents of children with ASD.

Aims of the Present Study

The present study aimed to identify psychosocial variables predictive of parent self-report, salivary biomarkers of stress, and level of CV activity in this population, including factors that could exacerbate (e.g., child behaviour problems) or alleviate (e.g., coping strategies) parenting stress. Regarding salivary biomarkers, in light of previous research (Seltzer et al., 2010), we aimed in particular to examine whether mothers of children with ASD have blunted cortisol profiles.

Method

Participants

The sample initially comprised 80 mothers of children with an independent diagnosis of an ASD. Six participants were excluded for failing to return questionnaires within the allotted time, leaving a final sample of 74. Participants were recruited through ASD support groups, schools, conferences, media, and online support groups and forums.

Mothers. The mean age of the sample was 41.22 years (*SD* = 5.97), ranging from 26–65 years. Fifty mothers (68%) reported that they were not in employment, while 16 (22%) reported being in part-time employment, and eight (11%) reported being in full-time employment. Mothers reported an average of 2.16 *(SD* = 2.21) current illnesses, ranging from 0–9 illnesses.

Children. Fifty-seven (77%) of the children were boys, and 17 (23%) were girls. Children had a mean age of 106.89 months (*SD* = 42.83, range: 43–207 months). Children received their ASD diagnoses when they were on average 52.23 months old (*SD* = 28.87, range: 18–168 months). Autism was reported as the primary diagnosis for 56 (76%) of the children, while 15 (20%) were reported as having Asperger's syndrome, and three (4%) were reported as having PDD-NOS. Children were reported as having a mean of 2.95 diagnoses (*SD* = 1.81), ranging from 1–8 diagnoses. Thirty-three (45%) children were reported as having an ID, with 14 (19%) reported as having a mild ID, 15 (20%) reported as having a moderate ID, and four (5%) reported as having a severe ID.

Ethics

Ethical approval for this research was obtained from the NUI Galway Research Ethics Committee, and all participants completed a consent form (see Appendix D).

Psychometric Measures

The same measures were used in all three studies. Participants completed a questionnaire booklet, which included inventories that measured parent characteristics and child characteristics. Internal reliability (Cronbach's α) is reported for the overall sample from all three studies, and from each individual study (see Tables 1 and 2).

Parenting Stress Index: Short form (PSI/SF; Abidin, 1995). The PSI/SF is a 36-item, self-report instrument that measures stress related to the role of parenting. It contains statements related to parenting such as, "I feel trapped by my responsibilities as a parent". Individuals rate their level of agreement with each

Table 1

Cronbach's Alpha Values for Psychometric Measures (Parent)

Measure	Study			
	All	1	2	3
PSI: Total	.96	.91	.92	.96
PSI: Parent-child dysfunctional interaction	.89	.84	.83	.89
PSI: Difficult child	.93	.87	.82	.94
PSI: Parental distress	.92	.86	.86	.91
PSI: Defensive responding	.88	.78	.82	.86
HADS: Anxiety	.89	.84	.84	.85
HADS: Depression	.83	.74	.76	.80
COPE: Self-distraction	.44[a]	.46[a]	.59[a]	.40[a]
COPE: Active coping	.82	.76	.78	.82
COPE: Denial	.64	.60	.92	.86
COPE: Substance use	.95	.94	.92	.91
COPE: Use of emotional support	.73	.65	.60	.76
COPE: Use of instrumental support	.78	.77	.72	.78
COPE: Behavioural disengagement	.73	.75	.83	.76
COPE: Venting	.40[a]	.35[a]	.11[a]	.29[a]
COPE: Positive reframing	.63	.42[a]	.33[a]	.65
COPE: Planning	.80	.71	.71	.83
COPE: Humour	.73	.72	.74	.72
COPE: Acceptance	.83	.72	.84	.86
COPE: Religion	.90	.90	.89	.88
COPE: Self-blame	.70	.67	.74	.77
SSQ: Social support quantity	.90	.93	.86	.84
SSQ: Social support quality	.92	.90	.82	.92
PSQI: Subjective sleep quality [b]	-	-	-	-
PSQI: Sleep latency	.83	.84	.86	.80
PSQI: Sleep duration [b]	-	-	-	-
PSQI: Habitual sleep efficiency [b]	-	-	-	-
PSQI: Sleep disturbances	.72	.67	.65	.72
PSQI: Use of sleeping medication [b]	-	-	-	-
PSQI: Daytime dysfunction	.65	.59[a]	.21[a]	.44[a]
PSQI: Global sleep problems	.81	.76	.75	.77
Parenting Responsibility Scale (PRS)	.95	.91	.92	.92

[a] Subscale removed from study due to poor reliability (i.e., $\alpha < .60$). [b] See Pg. 36 for details.

Table 2

Cronbach's Alpha Values for Psychometric Measures (Child)

Measure	Study			
	All	1	2	3
GARS: Stereotyped behaviours	.92	.82	.76	.94
GARS: Communication	.91	.83	.83	.94
GARS: Social interaction	.94	.83	.48[a]	.96
GARS: Autism Index	.97	.91	.80	.98
Vineland: Receptive subdomain	.94	.90	.91	.95
Vineland: Expressive subdomain	.99	.98	.98	.99
Vineland: Written subdomain	.97	.97	.95	.98
Vineland: Communication domain	.99	.98	.98	.99
Vineland: Personal subdomain	.96	.94	.93	.97
Vineland: Domestic subdomain	.96	.92	.90	.97
Vineland: Community subdomain	.97	.97	.95	.98
Vineland: Daily living domain	.99	.98	.97	.99
Vineland: Interpersonal relationships subdomain	.97	.94	.95	.98
Vineland: Play and leisure subdomain	.97	.93	.93	.97
Vineland: Coping subdomain	.96	.94	.90	.97
Vineland: Socialisation domain	.99	.98	.97	.99
Vineland: Fine motor subdomain	.93	.88	.83	.94
Vineland: Gross motor subdomain	.93	.87	.88	.94
Vineland: Motor skills domain	.95	.88	.91	.96
Vineland: Composite score	.99	.98	.97	.99
BPI: SIB frequency	.72	.66	.63	.75
BPI: SIB severity	.72	.68	.53[a]	.71
BPI: Stereotyped behaviour frequency	.93	.89	.92	.95
BPI: Stereotyped behaviour severity	.93	.89	.91	.95
BPI: Aggressive/destructive behaviour frequency	.87	.86	.80	.82
BPI: Aggressive/destructive behaviour severity	.89	.88	.79	.83

[a] Subscale removed from study due to poor reliability (i.e., $\alpha < .60$)

Table 2, Continued

Cronbach's Alpha Values for Psychometric Measures (Child, Continued)

Measure	Study			
	All	1	2	3
CSHQ: Total sleep disturbance	.89	.85	.89	.91
CSHQ: Bedtime resistance	.83	.82	.83	.84
CSHQ: Sleep onset delay [b]	-	-	-	-
CSHQ: Sleep duration	.77	.75	.75	.75
CSHQ: Sleep anxiety	.63	.55[a]	.54[a]	.69
CSHQ: Night wakings	.63	.62	.52[a]	.62
CSHQ: Parasomnias	.68	.64	.63	.68
CSHQ: Sleep disordered breathing	.60	.64	.57[a]	.44[a]
CSHQ: Daytime sleepiness	.71	.71	.57[a]	.66
Conners: Oppositional behaviour	.90	.88	.77	.85
Conners: Cognitive problems/inattention	.94	.89	.87	.94
Conners: Hyperactivity	.90	.82	.60	.90
Conners: ADHD index	.96	.91	.87	.97

[a] Subscale removed from study due to poor reliability (i.e., $\alpha < .60$). [b] See Pg. 40 for details.

statement on a 5-point Likert scale from 1 (*strongly agree*) to 5 (*strongly disagree*). It yields an overall parenting stress score from three different subscales: parental distress (PD), parent-child dysfunctional interaction (PCDI), and difficult child (DC). PD examines the extent the parent experiences stress in his/her role as a parent, and PCDI explores the extent to which the parent believes the child meets their expectations and their satisfaction level with their interactions. DC assesses the parent's perception of how difficult their child is to parent. The total parenting stress subdomain measures the overall stress the parent is experiencing in his/her role as a parent. In addition, the PSI/SF includes a defensive responding (DR) subscale to assess the extent to which a respondent may be trying to answer in a way he/she thinks will make them look best, with a score below 10 indicating that the participant may have been responding in a defensive manner (Abidin, 1995). Good internal consistency, test-retest reliability, and validity have been demonstrated (Abidin, 1995). Reliability was strong for all subdomains in the present study (see Table 1).

Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983). The HADS is a self-report instrument that comprises two 7-item subscales to measure perceived anxiety (HADS-A) and depression (HADS-D). It was designed to assess the severity of anxiety and depression without contamination by reports of physical symptoms. Each item is rated on a 4-point Likert scale ranging from 0 to 3, with response options differing for each item. High internal consistency and validity have been reported (Moorey et al., 1991; Zigmond & Snaith, 1983). In the present study, Cronbach's α was strong for both subdomains (see Table 1).

Brief COPE (Carver, 1997). The Brief COPE is a 28-item self-report measure of coping strategies. It is an abbreviated version of the COPE Inventory (Carver, Scheier, & Weintraub, 1989). It includes 14 scales with 2 items per scale. Scales retained from the COPE Inventory are self-distraction, active coping, denial, substance use, use of emotional support, use of instrumental support, behavioural disengagement, venting, positive reframing, planning, humour, acceptance, and religion, with a self-blame scale added. Respondents are presented with statements relating to different coping strategies (e.g., "I've been turning to work or other activities to take my mind off things"), and rate the extent to which they use the strategy on a 4-point Likert scale ranging from 1 (*I haven't been doing this at all*) to 4 (*I've been doing this a lot*). The factor structure was found to be consistent with the COPE Inventory, and good internal reliability has been reported (Carver, 1997). For the present study, Cronbach's α was poor for three subscales (i.e., self-distraction, venting, and positive reframing), so these subscales were excluded from the study (see Table 1). Acceptable Cronbach's α was found for denial, emotional support, and self-blame, so these subscales were included with caution. Internal reliability estimates were good for all other subscales.

Short form Social Support Questionnaire (SSQ6; Sarason, Shearin, Pierce, & Sarason, 1987). The SSQ6 is a measure of perceived social support, which assesses two aspects of social support: social support quantity, and social support quality. The SSQ6 is an abbreviated version of the original 27-item Social Support Questionnaire (Sarason, Levine, Basham, & Sarason, 1983). Respondents are presented with six questions (e.g., “Who can you count on to console you from your worries when you feel under stress?”), and are asked to list up to nine individuals that provide this type of support. They are then asked to rate their level of satisfaction with this type of support on a 6-point Likert scale ranging from 0 (*very dissatisfied*) to 6 (*very satisfied*). Both subscales have been found to have high internal consistency and test-retest reliability, and strong factorial validity has also been reported (Sarason et al., 1987). In the present study, strong internal reliability was observed (see Table 1).

Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI is a 19-item scale that assesses sleep quality and disturbances over a 1-month period. It generates a global score and seven component subscales: subjective sleep quality, sleep latency, sleep duration, sleep disturbances, habitual sleep efficiency, use of sleeping medication, and daytime dysfunction. Acceptable internal consistency, test-retest reliability, and validity have been reported (Buysse et al., 1989). In the present study, however, poor internal consistency was found for daytime dysfunction, so this subscale was excluded from the study (see Table 1). Cronbach’s α could not be calculated for subjective sleep quality, sleep duration, habitual sleep efficiency, or use of sleeping medication, as these are single item subscales. However, we inspected the Cronbach’s α for the global index if the items were deleted. Deletion of any of these subscales would have only minimally adjusted the alpha value. Furthermore, previous research has reported acceptable reliability and validity, and good correlation between subscales and the global index (Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002; Beck, Schwartz, Towsley, Dudley, & Barsevick, 2004). Thus, these subscales were included in the research with caution. Acceptable reliability was found for the remaining subscales.

Parental Responsibility Scale (PRS; McBride & Mills, 1993). The PRS is a 14-item measure of parental responsibility. Common childcare tasks are listed (e.g., “Spending special time with the child at bedtime”). Respondents designate which parent has the primary responsibility for that task on a 5-point Likert scale ranging from 1 (*almost always completed by me*) to 5 (*almost always completed by my partner*). In the original scale by McBride and Mills (1993), 1 represented “*mother almost always*” and 5 represented “*father almost always*”. The wording was changed slightly to meet the needs of the present study. Responsibility was defined as remembering, planning, and scheduling the task, so it was possible for the parent to be responsible for a task without actually completing it. Moderate internal consistency, test-retest reliability, and validity have been reported (McBride & Mills, 1993). A strong Cronbach’s α of .91 was observed in the present study.

Gilliam Autism Rating Scale (GARS-2; Gilliam, 2006). The GARS-2 is a 42-item, norm-referenced instrument, which is completed by parents to assess the severity of their child’s ASD symptoms. It includes three subscales (i.e., stereotyped behaviours, communication, and social interaction), which are combined to create an overall ASD severity score, the Autism Index. Each subscale includes 14 items. A number of behaviours commonly demonstrated by individuals with ASD are listed, and respondents rate the items according to the frequency of occurrence on a 4-point Likert scale, ranging from 0 (*never observed*) to 3 (*frequently observed*). A structured interview form is also available for diagnostic purposes, but this was not included in the present study. Strong reliability and validity have been reported (Gilliam, 2006). In the present study, internal reliability estimates were strong for all subscales (see Table 2).

Vineland Adaptive Behavior Scales (Vineland-II; Sparrow, Cicchetti, & Balla, 2005). The Vineland-II is a standardised, norm-referenced measure of adaptive skills. The Parent/Caregiver Rating Form was used in the present study. An overall adaptive behaviour score (i.e., the composite score) is created from 3 domains: communication, daily living, and socialisation. These domains include a total of 307 items. For children aged 0-7 years, the 76-item motor skills domain is also required to compute the composite score, so this domain was included for children within this age range only. The optional ‘Maladaptive Behaviour Index’ was not included. Each domain has a number of subdomains: receptive, expressive, and written (which form the communication domain); personal, domestic, and community (which form the daily living skills domain); interpersonal relationships, play and leisure time, and coping skills (which form socialisation domain); and fine motor and gross motor (which form the motor skills domain). A number of behaviours are listed (e.g., “Turns eyes and head toward sound”), and respondents rate how often their child performs the behaviour without help, when the behaviour is needed. Items are rated on a 4-point Likert scale ranging from 2 (*usually performs the behaviour without help*) to 0 (*never performs the behaviour without help*), with an optional DK (*don’t know*) response if the parent is unsure if the child can perform the behaviour. For 15 of the items, there is also an N/O (*no opportunity*) option if the child has never had the opportunity to perform the behaviour (e.g., if there is no computer in the home). Excellent internal consistency, test-retest reliability and validity have been reported for all domains and subdomains (Sparrow et al., 2005). Cronbach's α for all domains and subdomains was strong in the present study (see Table 2).

Behaviour Problems Inventory (BPI-01; Rojahn, Matson, Lott, Esbensen, & Smalls, 2001). The BPI-01 is a 52-item assessment tool that measures problem behaviours in individuals with developmental disabilities. It consists of 3 subscales: self-injurious behaviour (SIB), stereotyped behaviour, and aggressive or destructive behaviour. Frequency and severity scores are provided for each of these subscales. Common behaviours are listed (e.g., self-biting) and respondents rate how frequently the individual engaged in this behaviour in the past two months, using a 5-point Likert scale ranging from 0 (*never*) to 4 (*hourly*). Severity of the behaviour is also rated on a 4-point Likert scale, ranging from 0 (*never*) to 4 (*severe*). High internal consistency, test-retest reliability, inter-observer agreement, and factor and criterion validity were reported (Rojahn et al., 2001). In the present study, there was acceptable internal reliability for the SIB subscales, and strong internal reliability for the remaining subscales (see Table 2).

Child's Sleep Habits Questionnaire (CSHQ; Owens, Spirito, & McGuinn, 2000). The CSHQ is a 35-item scale that assesses child sleep problems. Statements relating to sleep habits are listed (e.g., "Child falls asleep in parent's or sibling's bed"). Respondents rate how frequently this has occurred for the child in the previous week on a 3-point Likert scale, ranging from 3 (*usually, 5-7 times*) to 1 (*rarely, 0-1 times*). Six items are presented in reverse format, with a desirable sleep habit presented (e.g., "Child falls asleep in own bed"), and these items are reverse-coded after completion. Items 10 and 27 are open-ended questions, and items 34 and

35 are rated on a 3-point Likert scale ranging from 1 (*not sleepy*) to 3 (*falls asleep*). The CSHQ provides an overall sleep disturbance score and 8 subscales: bedtime resistance, sleep onset delay, sleep duration, sleep anxiety, night wakings, parasomnias, sleep disordered breathing, and daytime sleepiness. Acceptable internal consistency, test-retest reliability, and validity have been reported (Owens et al., 2000). In the present study, poor internal reliability was found for the sleep anxiety subscale (see Table 2), so this was excluded from the data analysis. The parasomnias and night wakings subscales had acceptable internal reliability, so they were included with caution. A Cronbach's α value could not be calculated for sleep onset delay, because it is a single-item subscale. However, deletion of the sleep onset delay subscale was found to have minimal effect on the Cronbach's α value for the CSHQ total score, so this subscale was retained with caution. The remaining subscales had good internal reliability.

Conners' Parent Rating Scale (CPRS-R:S; Conners, 1997). The CPRS is an instrument for assessing Attention-Deficit/Hyperactivity Disorders (ADHD) and related problems in children aged 3–17 years old. It is norm-referenced by age and gender. The 27-item short-form version of the parent rating scale was used in the present study. Common problems that children may have are listed (e.g., "Inattentive, easily distracted"). The respondent rates how much of a problem this has been for their child in the past month using a 4-point Likert scale ranging from 0 (*not true at all, never*) to 3 (*very much true, very often*). This version includes four subscales: oppositional, cognitive problems/inattention, hyperactivity, and the ADHD index. Strong internal consistency, test-retest reliability, and factorial validity have been reported (Conners, 1997). Strong internal reliability was found in the present study for all subscales (see Table 2).

Child and personal information. The questionnaire booklet also included child and personal information questionnaires (see Appendix A). The child questionnaire collected information on child and family characteristics, including child age, gender, residential status, ASD and comorbid diagnosis details, education, interventions, unmet service needs, number of additional children in the family, number of additional children with diagnoses, and the child's birth order. The participant information questionnaire contained questions about participant age, employment status, and supports received (e.g., housekeeping assistance, caregiving support from friends or family, or access to services). Health-related details were also collected, including body mass index (BMI), oral health problems, smoking status, activity levels, alcohol and caffeine intake, illnesses, medication usage, weight loss, use of psychotherapy, and menopause status.

Saliva Collection

As mentioned above, the HPA-axis is activated in response to stressors, triggering activation of the stress hormone, cortisol. In blood, only 10% of the cortisol is unbound to protein, which is the cortisol of interest when measuring stress (Romanczyk & Gillis, 2006). Salivary cortisol, which is in its unbound form, is a more effective measure of HPA-axis activity than serum cortisol. Cortisol has a strong basal diurnal profile, consisting of an increase of approximately 50–60% in the first 30–45 minutes after waking (known as the cortisol awakening response; CAR), a rapid drop over the first few hours after waking, and a gradual decline throughout the rest of the day (Adam et al., 2006). The CAR can used to measure dysregulation of the cortisol diurnal profile.

Similarly, sAA is a non-invasive measure of SNS activity (Salimetrics, 2012c). Studies have reported increases in alpha-amylase levels in response to a variety of physical and psychological stressors, such as the Trier Social Stress Test, and associations have been found between sAA and SNS activation (Granger et al., 2007). Thus, sAA can be used as a marker for stress-induced activation of the SNS (Rohleder & Nater, 2009).

In the present study, saliva samples were collected from under the front of the tongue using Salimetrics Oral Swabs (SOS; Salimetrics Europe, Suffolk, UK). The SOS is a small polymer swab that absorbs saliva from the mouth (Salimetrics, 2012b). The SOS was chosen to collect the samples to encourage compliance, as it was expected that it would be preferred by research participants over drooling into a vial. Additionally, collecting saliva using the SOS has an advantage over passive drool, in that it helps to filter mucus and other matter from the sample (Salimetrics, 2012b). Participants were given written saliva collection guidelines (see Appendix B), which were also explained by phone. Participants were advised not to schedule data collection for at least 2 days after any dental work. Participants were also advised to avoid alcohol for 24 hours before collecting saliva, and to avoid tooth-brushing, smoking, eating, drinking, and exercising in the 60 minutes before collecting saliva samples.

CV Assessment

CV measures were taken using a battery-operated Oscar 2™ ABP monitor with an Orbit™ cuff (SunTech Medical Ltd., Oxfordshire, England). The Oscar 2™ measures BP using the oscillometric method, which senses waves of pressure in the artery when occluded by pressure in the cuff, and HR is measured by measuring the frequency of the pressure waves (SunTech Medical, 2010). The reliability and validity of this monitor have been demonstrated (Goodwin, Bilous, Winship, Finn, & Jones, 2007).

In the present study, the Oscar 2™ unit was worn by participants on a waist belt and connected to a cuff worn around the non-dominant upper arm. The cuff was programmed to inflate on a random schedule (+/- 5 minutes) at 20 minute intervals between 8:30 and 22:30 ("daytime"), and at 45 minute intervals between 22:30 and 8:30 ("night-time"). Systolic blood pressure (SBP), diastolic blood pressure (DBP), HR, mean arterial pressure (MAP), and pulse pressure (PP) were recorded for each reading. The cuff was programmed to inflate to 30mmHg above the previous SBP, with maximum pressure set to 220mmHg. CV data were downloaded from the monitor using the SunTech AccuWin Pro v3 ABPM software (SunTech Medical Ltd., Oxfordshire, England).

Diaries

A diary was provided to each participant (see Appendix C). Participants were asked to report their location, activity, and mood each time the BP cuff inflated. The rating of mood states used a 4-point scale, ranging from 1 (*not at all*) to 4 (*extremely*), and contained the following moods: "sad", "active", "interested", "stressed", "upset", "excited", "frustrated", and "alert". In order to obtain information on adherence to saliva collection guidelines, diary entries were also completed after saliva samples were collected. A summary diary entry was also completed when the ABP monitor was removed. Diaries also included questions on predicted and perceived stress, ratings of child behaviour problems, caffeine and alcohol intake, smoking, medication usage, exercise, and supports.

Procedure

The morning saliva collection tubes and instructions were posted or hand-delivered to participants. Instructions on how to collect the morning saliva samples were then explained to participants by phone or in person. The researcher and participant agreed on a suitable day to conduct the study. The study was then carried out on that day in the participant's natural environment, as she continued her typical daily routine.

On the morning of the study, the participant independently collected four saliva samples: immediately, 15, 30, and 45 minutes after waking. Completed samples were then placed by the participant into her domestic freezer, and a diary entry was completed. The researcher then met with the participant later that morning, usually in the participant's home, at a previously agreed time (usually approximately 11am). The researcher attached an ABP monitor to the participant, ensured that the initial reading was valid, and provided written instructions which were also verbally explained to the participant. The researcher also provided the daily diary to the participant at this time, and explained how it should be completed. The participant then proceeded with the rest of the study independently until the researcher returned 24 hours later. The researcher's telephone number was provided in case of queries during the study. The BP cuff inflated approximately every 20 minutes during the day, and every 45 minutes at night. When the cuff inflated during waking hours, the participant made relevant entries into the cuff-inflation diary. An additional saliva sample was collected at 12:00, and a corresponding diary entry was completed.

Twenty-four hours after the ABP monitor had been attached, the researcher returned to remove the monitor, and to collect the diary and saliva samples. A summary diary entry was provided at this time for the participant to complete. The participant was given a questionnaire booklet and a stamped addressed envelope, and asked to return the completed questionnaire within 1–2 months. Participants were given this time due to the size of the questionnaire booklet, and the sensitive nature of some of the questions (e.g., questions about the child's diagnosis).

Salivary Analysis

Saliva samples were retrieved from participants, and stored frozen at -20℃ until they were assayed for cortisol and sAA. On the day of testing, samples were thawed, vortexed, and centrifuged at 3000rpm for 15 minutes.

Cortisol. The four morning saliva samples were assayed in duplicate for salivary cortisol by enzyme immunoassay using the Salimetrics salivary cortisol assay kits (Salimetrics Europe, Suffolk, UK). The test requires 25µL of saliva, and it has a range of sensitivity from 0.003–3.0 µg/dL. Average intra- and inter-assay coefficients of variability were less than 4 and 7%, respectively. The coefficient of variability refers to the standard deviation of a set of measurements divided by the mean of the set (Salimetrics, 2013). Two area under the curve calculations were made: area under the curve with respect to ground (AUC_G), and area under the curve with respect to increase (AUC_I), using Pruessner, Kirschbaum, Meinlschmid, and Hellhammer's (2003) formulas. Both the AUC_I (a measure of the dynamic increase in cortisol secretion), and the AUC_G (which represents overall cortisol secretory activity), have been found to be differentially associated with psychological health and well-being (Clow et al, 2004). To control for noncompliance, samples were excluded if there was more than a 10-minute difference between reports of waking and collecting the first sample (Steptoe, Siegrist, Kirschbaum, & Marmot, 2004). Samples were also excluded if there was not enough saliva in the sample to perform the assay. Applying these criteria, sufficient data were available to conduct cortisol analyses for 41 participants.

Alpha-amylase. The 12:00 samples were assayed by kinetic measurement with the Salimetrics sAA assay kits (Salimetrics Europe, Suffolk, UK). The test used 10µL of saliva. Average inter-assay coefficients of variability were less than 11%.

To control for noncompliance, samples were excluded if they were not reported as being collected within 60 minutes of the target time (i.e., 12:00; Rohleder, Chen, Wolf, & Miller, 2008). Samples were excluded if there was insufficient quantity of saliva to perform the assay. SAA data were available from 49 participants for inclusion in subsequent analyses.

Data Analysis

All statistical analyses were conducted using the IBM SPSS statistical package (Version 20 for Windows). Cortisol and sAA data were positively skewed, and were natural-log and square-root transformed, respectively (Adam & Kumari, 2009; Granger et al., 2007). Outliers identified on boxplots were then recoded to the nearest value in the distribution. Skewness statistics were less than 0.35 for all of the cortisol and sAA distributions after data transformation. For ease of interpretation, raw values are presented in tables and graphs.

Pearson's product moment correlations (Pearson's *r*; with interval variables) or Spearman's rank order correlations (Spearman's rho; with nominal or ordinal variables) were conducted to test for associations with the following stress measures: perceived anxiety, depression, total parenting stress, parent-child dysfunctional interaction, difficult child scores, parental distress, mean CV measures (mean awake, asleep, and 24-hour SBP, DBP, HR, MAP, and PP, and mean night-time SBP and DBP reduction), AUC_G, $AUC_{I,}$ and sAA. The correlation results (see Appendix E for significant correlations) and past research were used to inform the structure of multiple regression models. Separate multiple regression analyses were conducted for each outcome variable. None of the psychosocial variables were significantly correlated with sAA or awake DBP, which were not included in regression analyses as a result. A maximum of seven variables were entered into the HADS, PSI, and

CV multiple regression analyses, and a maximum of four into the cortisol analyses, to ensure that the sample size for each met the criterion of $n \geq 20 + 5m$ (where m refers to the number of predictor variables; Khamis & Kepler, 2010).

Results

Descriptive Statistics

As shown in Table 3, group means for anxiety fell between the mild (8–10) to moderate (11–14) ranges, while group means for depression fell between the normal (0–7) to mild ranges (8–10; Zigmond & Snaith, 1983). Anxiety scores for 14 participants (19%) fell within the severe range (15–21), although only one participant had a depression score in the severe range. Group means also indicated high levels of overall parenting stress, difficult child scores, parent-child dysfunctional interactions, and parental distress (see Table 3). Group means for all PSI subscales fell above the 85th percentile, which indicates clinically significant levels of parenting stress (Abidin, 1995). Mean scores indicated that defensive responding was not a significant problem (scores below 10 suggest possible defensive responding on the PSI).

Mean group cortisol values are illustrated in Figure 1, alongside average cortisol values reported by Wust et al. (2000). One-sample *t*-tests were conducted to compare cortisol values of mothers of children with ASD to Wust et al.'s (2000) normal cortisol values. Alpha values were corrected using family-wise Bonferroni adjustment ($\alpha = .0125$). For the following measures, cortisol values were significantly lower among mothers of children with ASD than the reported averages: immediately after waking, t (50) = -2.82, p = .01; 30 minutes after waking, t (49) = -6.87, p < .001; and 45 minutes after waking, t (48) = -8.91, p < .001. Although mean sAA levels (M = 109.26 U/mL, SD = 66.68) were somewhat higher than Salimetrics'

Table 3

Descriptive Information for Outcome Variables (*N* = 74)

Measure	M *(SD)*	Range
Parenting responsibility	50.96 (7.53)	25 – 60
Anxiety	10.77 (4.08)	3 – 19
Depression	7.89 (3.59)	1 – 15
Total parenting stress	109.12 (20.59)	63 – 154
Parent-child dysfunctional interaction	30.35 (8.26)	13 – 48
Difficult child	41.00 (8.47)	20 – 58
Parental distress	37.77 (9.48)	15 – 60
Defensive responding	23.12 (5.68)	8 – 35
Mean 24-hour SBP (mmHg)	127.81 (14.05)	99 – 170
Mean night-time SBP reduction (%)	13.29 (8.89)	-3 – 44
Mean 24-hour DBP (mmHg)	76.07 (10.31)	51 – 123
Mean night-time DBP reduction (%)	19.93 (8.15)	4 – 38
Mean 24-hour HR (bpm)	74.61 (9.50)	49 – 94
Mean 24-hour MAP (mmHg)	92.82 (10.17)	67 – 123
Mean 24-hour PP (mmHg)	52.56 (7.66)	42 – 71
Cortisol: Sample 1 (µg/dL)	0.43 (0.30)	0.1 – 2. 1
Cortisol: Sample 2 (µg/dL)	0.55 (0.31)	0.2 – 2.0
Cortisol: Sample 3 (µg/dL)	0.56 (0.28)	0.2 – 1.8
Cortisol: Sample 4 (µg/dL)	0.48 (0.24)	0.1 – 1.2
Alpha-amylase (U/mL)	110.71 (70.31)	18 – 308

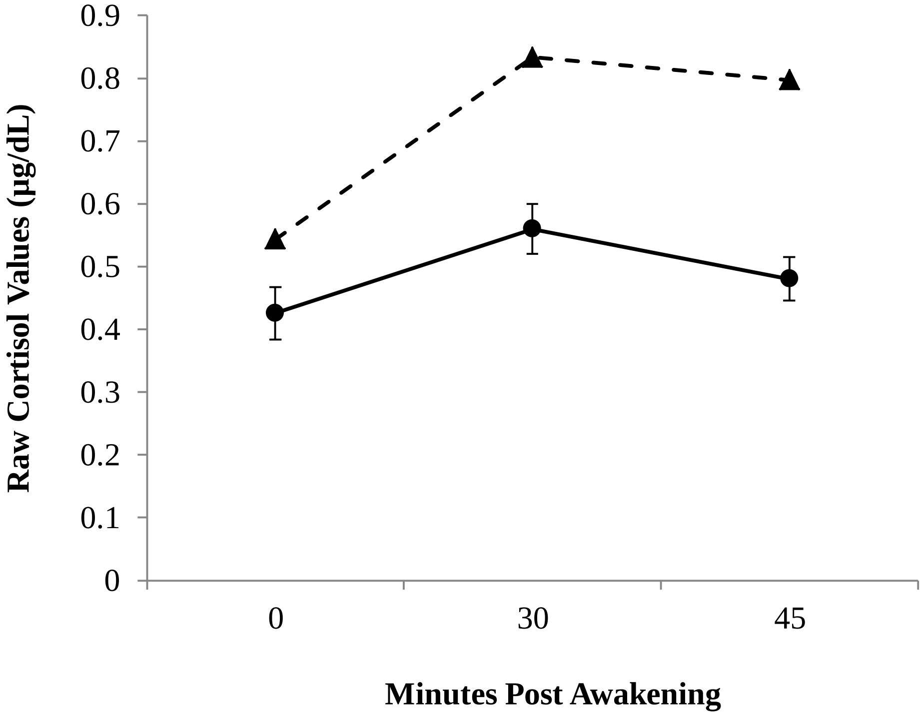

Figure 1. Post-awakening mean group cortisol values (µg/dL) of mothers of children with ASD in the present study (circles) and as published by Wust et al. (2000; triangles and dashed line)

Note. Standard error bars are shown for cortisol values of mothers of children with ASD in the present study, but are not available for results published by Wust et al. (2000)

expected adult mean of 92.40 U/mL (Salimetrics, 2012c), this difference was not statistically significant, *t* (35) = 1.50, *p* = .14.

As shown in Table 3, group mean CV responses fell within the normotensive range, defined as a 24-hour SBP ≤ 135 mmHg and a 24-hour DBP ≤ 85 mmHg (O’Brien et al., 2000). However, there was a wide range of values recorded, with mean 24-hour SBP (range: 99–170 mmHg) and mean 24-hour DBP (range: 51–123 mmHg) values for some participants falling within the hypertensive range.

Regression Analysis: Predictors of Perceived Anxiety, Depression, and Parenting Stress

As shown in Table 4, high levels of self-blame, parental sleep dysfunction, and child parasomnias predicted higher levels of anxiety among mothers, after controlling for quantity of illnesses, depression, and alcohol intake. In contrast, behavioural disengagement was not a significant predictor of anxiety. Use of self-blame as a coping strategy also predicted higher levels of depression after controlling for quantity of illnesses, BMI, and use of antidepressants, as did quantity of unmet service needs (see Table 4). A higher quantity of social supports, on the other hand, predicted lower levels of depression. However, parental sleep dysfunction did not significantly predict depression.

Higher socialisation deficits and oppositional behaviour significantly predicted higher overall parenting stress, after controlling for BMI (see Table 5). However, adaptive behaviour, aggression severity, overall child sleep problems, and overall maternal sleep dysfunction did not significantly predict parenting stress levels. As shown in Table 5, higher socialisation deficits and oppositional behaviour also predicted higher levels of parent-child dysfunctional interactions, after controlling for quantity of illnesses and exercise duration. Higher levels of adaptive behaviour also predicted lower levels of parent-child dysfunctional interaction, although overall maternal sleep dysfunction and child sleep problems were not significant predictors.

Higher levels of adaptive behaviour predicted lower difficult child scores, after controlling for quantity of illnesses (see Table 5). Higher levels of oppositional behaviour, on the other hand, predicted higher levels of difficult child scores. In contrast, overall child sleep problems, ASD severity, and aggression frequency and severity did not significantly predict difficult child scores. As shown in Table 5,

Table 4

Summary of Multiple Regression Analyses for Perceived Anxiety and Depression (*N* = 74)

Variable	B	Standard Error	β	ΔR^2
Predictors of perceived anxiety				
1. Current quantity of illnesses	.223	.234	.119	.242
Current depression	3.386	1.238	.327**	
Units of alcohol consumed	.904	.237	.418***	
2. Behavioural disengagement	.149	.345	.048	.470
Self-blame	.711	.252	.323**	
PSQI global score	.255	.119	.239*	
Child parasomnias	.366	.142	.246*	
Predictors of perceived depression				
1. Current quantity of illnesses	-.089	.222	-.055	.155
Body mass index (BMI)	.109	.066	.207	
Current depression	3.366	1.225	.369**	
2. Social support quantity	-.089	.035	-.299*	.229
3. Self-blame	.829	.182	.440***	.505
PSQI global score	.129	.105	.135	
Unmet service needs	.319	.153	.216*	

Note. *$p < .05$. **$p < .01$. ***$p < .001$.

Table 5

Summary of Multiple Regression Analyses for Parenting Stress Subscales (*N* = 74)

Variable	B	Standard Error	β	ΔR^2
Predictors of total parenting stress				
1. BMI	.766	.258	.351**	.109
2. Vineland composite	-.207	.138	-.164	.424
Socialisation (GARS)	1.799	.874	.252*	
Aggression severity (BPI)	.256	.392	.079	
Oppositional behaviour (CPRS)	.531	.200	.348*	
Overall sleep problems (CSHQ)	.039	.225	.020	
3. PSQI global score	.738	.585	.133	.429
Predictors of parent-child dysfunctional interactions				
1. Quantity of illnesses	.157	.469	.040	.064
Exercise duration	.017	.007	.293*	
2. Vineland composite	-.210	.057	-.404***	.218
3. Overall sleep problems (CSHQ)	-.003	.095	-.004	.384
Oppositional behaviour (CPRS)	.169	.069	.266*	
PSQI global score	.342	.241	.159	
Socialisation deficits (GARS)	.840	.361	.281*	
Predictors of difficult child scores				
1.Quantity of illnesses	.873	.476	.220	.034
2.Vineland composite	-.134	.062	-.253*	.085
3. Aggression severity (BPI)	.119	.406	.088	.330
Aggression frequency (BPI)	.154	.330	.141	
Oppositional behaviour (CPRS)	.235	.086	.374**	
Overall sleep problems (CSHQ)	.037	.098	.045	
ASD severity (GARS)	-.004	.065	-.007	
Predictors of parental distress				
1. BMI	.481	.153	.362**	.254
Use of antidepressants	7.495	2.743	.315**	
2. Inattention (CPRS)	.105	.086	.151	.318
Unmet service needs	.917	.448	.243*	
3. PSQI global score	.296	.266	.120	.487
Behavioural disengagement	-.435	.799	-.068	
Self-blame	2.092	.553	.446***	

Note. **p* < .05. ***p* < .01. ****p* < .001.

a higher quantity of unmet service needs and use of self-blame predicted higher parental distress, after controlling for BMI and use of antidepressants. However, inattention, overall maternal sleep dysfunction, and behavioural disengagement were not significant predictors of parental distress.

Regression Analysis: Predictors of Cortisol Levels

As shown in Table 6, higher child parasomnias predicted a lower AUC_G, after controlling for fatigue. However, neither frequency nor severity of SIB significantly predicted AUC_G. Higher fine motor skills also predicted a lower AUC_I, after controlling for past quantity of illnesses, maternal age, and family history of endocrine disorder (see Table 6).

Table 6

Summary of Multiple Regression Analyses for Cortisol (AUC_G and AUC_I; n = 41)

Variable	B	Standard Error	β	ΔR^2
Predictors of AUC_G				
1. Fatigue	-.310	.149	-.287*	.063
2. Parasomnias (CSHQ)	-.062	.024	-.338*	.264
SIB frequency (BPI)	-.057	.038	-.560	
SIB severity (BPI)	.047	.053	.331	
Predictors of AUC_I				
1. Past quantity of illnesses	.095	.060	.322	.335
Participant age	.046	.025	.393	
Family history of endocrine disorder	.364	.312	.257	
2. Fine motor skills (Vineland)	-.062	.024	-.478*	.523

Note. $*p < .05$.

Regression Analysis: Predictors of CV Activity

Higher quantity of unmet service needs predicted higher mean awake SBP, after controlling for reported pre-existing hypotension (i.e., low BP), pre-existing hypertension, and caffeine intake (see Table 7). However, child daytime sleepiness, overall maternal sleep dysfunction, and behavioural disengagement did not significantly predict mean awake SBP. As shown in Table 7, a higher quantity of unmet service needs was also found to predict higher asleep SBP, after controlling for BMI and pre-existing hypotension, while overall maternal sleep dysfunction, perceived anxiety, parental distress, and behavioural disengagement did not significantly predict mean asleep SBP. Higher unmet service needs, along with higher behavioural disengagement and lower child daytime sleepiness, also predicted higher 24-hour SBP, after controlling for pre-existing hypotension and hypertension. However, overall maternal sleep dysfunction and quantity of unmet supports did not significantly predict 24-hour SBP. None of the psychosocial variables significantly predicted mean SBP night-time reduction.

As shown in Table 8, later detection of a problem (i.e., the parent noticing a problem when the child was older) was found to predict higher asleep DBP, after controlling for pre-existing hypotension and hypertension. However, overall maternal sleep dysfunction, emotional support, and behavioural disengagement were not significant predictors of mean asleep DBP. None of the psychosocial variables significantly predicted mean 24-hour DBP. However, later detection of a problem predicted a smaller night-time drop in DBP, after controlling for pre-existing hypotension and hypertension (see Table 8). Needing to use sleeping medication (PSQI) significantly predicted mean awake HR, after controlling for participant age, cigarettes smoked, use of antidepressants, current diabetes, and past

Table 7

Summary of Multiple Regression Analyses for Systolic Blood Pressure (*N* = 74)

Variable	B	Standard Error	β	ΔR^2
Predictors of mean awake SBP				
1. Hypotension	-11.892	4.354	-.310**	.284
Caffeine intake	.956	.576	.196	
Hypertension	11.381	4.494	.297*	
2. Unmet service needs	1.795	.669	.291*	.371
Daytime sleepiness (CSHQ)	-.637	.523	-.133	
3. PSQI global score	.613	.416	.169	.410
Behavioural disengagement	1.815	1.157	.185	
Predictors of mean asleep SBP				
1. Hypotension	-17.465	5.854	-.362**	.223
BMI	.704	.317	.269*	
2. Unmet service needs	2.278	.828	.312**	.307
3. PSQI global score	.566	.600	.119	.318
Perceived anxiety	.738	.611	.176	
Parental distress	-.220	.306	-.115	
Behavioural disengagement	1.258	1.544	.107	
Predictors of mean 24-hour SBP				
1. Hypotension	13.994	5.850	.325*	.206
Hypertension	-13.039	5.408	-.327*	
2. Unmet service needs	2.495	.668	.432**	.438
Daytime sleepiness (CSHQ)	*-1.017	.465	-.250*	
3. PSQI global score	.663	.413	.185	.519
Unmet supports	.588	1.009	.073	
Behavioural disengagement	2.989	1.363	.263*	
Predictors of mean SBP night-time reduction				
1. Body mass index (BMI)	-.335	.100	-.369**	.308
Recent weight loss	.355	.097	.414**	
Past hypertension	-9.267	3.449	-.306*	
Chronic headaches	4.257	2.729	.173	
2. Daytime sleepiness (CSHQ)	.506	.291	.190	.327
PSQI global score	-.177	.266	-.077	

Note. *$p < .05$. **$p < .01$.

Table 8

Summary of Multiple Regression Analyses for Diastolic Blood Pressure ($N = 74$)

Variable	B	Standard Error	β	ΔR^2
Predictors of mean asleep DBP				
1. Current hypertension	.845	3.428	.029	.124
Current hypotension	-12.401	3.866	-.383**	
2. Age of child when problem detected	.269	.104	.295*	.198
3. PSQI global	.345	.330	.124	.239
Emotional support	-1.159	.814	-.166	
Behavioural disengagement	1.373	.957	.171	
Predictors of mean 24-hour DBP				
1. Current hypertension	3.247	3.777	.107	.130
Current hypotension	-11.650	3.770	-.365**	
Current diabetes	2.268	4.488	.063	
2. Behavioural disengagement	1.536	.983	.199	.164
PSQI global score	.351	.341	.125	
Predictors of mean DBP night-time reduction				
1. Current hypertension	3.179	2.679	.151	.021
Current hypotension	4.747	3.021	.200	
2. Age of child when problem detected	-.227	.081	-.340**	.122

Note. *$p < .05$. **$p < .01$.

Table 9

Summary of Multiple Regression Analyses for Heart Rate (*N* = 74)

Variable	B	Standard Error	β	ΔR^2
Predictors of mean awake HR				
1. Participant age	-.524	.191	-.355**	.127
Quantity of cigarettes smoked	.056	.038	.190	
Use of antidepressants	-.586	3.039	-.025	
2. Current diabetes	4.445	4.848	.132	.147
Past hypertension	6.914	4.366	.206	
3. Needing to use sleeping medication (PSQI)	2.961	1.354	.278*	.209
Predictors of mean asleep HR				
1. Participant age	-.276	.218	-.178	.256
Use of anti-hypertensive medication	-4.603	3.722	-.176	
Current hypotension	-.435	4.530	-.014	
Current diabetes	10.008	5.013	.282	
Current circulation problems	-19.320	6.225	-.450**	
2. Bedtime resistance (CSHQ)	-.252	.439	-.083	.267
Child behaviour problems (Day 1)	5.112	3.437	.208	
Predictors of mean 24-hour HR				
1. Participant age	-.587	.249	-.339*	.120
Number of cigarettes smoked	.063	.040	.225	
Use of antidepressants	.618	3.375	.026	
2. Unmet service needs	.208	.552	.057	.163
Needing to use sleeping medication (PSQI)	3.442	1.739	.307	

Note. **p* < .05. ***p* < .01.

Table 10

Summary of Multiple Regression Analyses for Mean Arterial Pressure ($N = 74$)

Variable	B	Standard Error	β	ΔR^2
Predictors of mean awake MAP				
1. Current hypertension	8.793	3.339	.302*	.214
Current hypotension	-9.957	3.339	-.342**	
2. Unmet service needs	1.446	.473	.332**	.323
Child behaviour problems (Day 1)	.489	.543	.102	
3. Parental distress	.063	.142	.058	.357
PSQI global score	.632	.313	.230*	
Behavioural disengagement	.327	.931	.047	
Predictors of mean asleep MAP				
1. Current hypertension	2.865	3.788	.089	.212
Current hypotension	-15.690	3.979	-.466***	
2. Unmet service needs	1.273	.535	.260*	.353
Age of child when problem first detected	.298	.110	.292**	
3. PSQI global score	.408	.373	.134	.383
Perceived anxiety	.188	.365	.065	
Behavioural disengagement	1.436	1.036	.176	
Predictors of mean 24-hour MAP				
1. Current hypertension	7.649	3.263	.276*	.240
Current hypotension	-11.619	3.453	-.397**	
2. Unmet service needs	1.475	.476	.350**	.366
Education duration	.072	.076	-.109	
Child behaviour problems (Day 1)	.197	.533	.042	
3. PSQI global score	.750	.286	.278*	.458
Behavioural disengagement	1.146	.801	.170	

Note. $*p < .05$. $**p < .01$. $***p < .001$.

Table 11

Summary of Multiple Regression Analyses for Pulse Pressure (*N* = 74)

Variable	B	Standard Error	β	ΔR^2
Predictors of mean awake PP				
1. BMI	.144	.255	.116	.359
Current hypertension	14.776	5.020	.527**	
Current depression	6.937	4.072	.335	
2. Gross motor skills	.535	.428	.190	.458
Unmet service needs	-.001	.601	.000	
Daytime sleepiness (CSHQ)	-1.193	.497	-.391*	
3. PSQI global score	.314	.401	.132	.446
Predictors of mean asleep PP				
1. Current hypertension	8.804	3.931	.318*	.112
Current hypotension	-5.029	3.931	-.182	
2. Unmet service needs	1.868	.459	.492***	.295
Daytime sleepiness (CSHQ)	-.722	.331	-.256*	
3. Subjective sleep quality (PSQI)	3.492	1.713	.277*	.458
Unmet supports	.487	.760	.088	
Parental distress	.096	.157	.091	
Predictors of mean 24-hour PP				
1. Current hypertension	15.875	4.221	.614**	.453
Current depression	8.875	3.146	.461*	
2. Daytime sleepiness (CSHQ)	-.807	.480	-.289	.499
Gross motor skills (Vineland)	.548	.487	.176	
3. PSQI global score	.341	.331	.161	.604
Unmet supports	.255	.745	.060	
Parenting responsibility	.376	.178	.401	

Note. *$p < .05$. **$p < .01$. ***$p < .001$.

hypertension (see Table 9). However, none of the psychosocial variables significantly predicted mean asleep or 24-hour HR.

As shown in Table 10, quantity of unmet service needs and overall sleep dysfunction (PSQI global) significantly predicted mean awake MAP, after controlling for current hypertension and hypotension. However, child behaviour problems, parental distress, and behavioural disengagement were not significant predictors of mean awake MAP. Unmet service needs and the age of the child when the parents first detected a problem also significantly predicted mean asleep MAP, after controlling for current hypertension and hypotension. However, overall sleep dysfunction, perceived anxiety, and behavioural disengagement were not significant predictors of mean asleep MAP. Unmet service needs, along with overall sleep dysfunction, also significantly predicted mean 24-hour MAP, after controlling for current hypertension and hypotension. Neither education duration, child behaviour problems the day before the study, nor behavioural disengagement significantly predicted mean 24-hour MAP.

Child daytime sleepiness inversely predicted mean awake PP, after controlling for current hypertension, depression, and BMI (see Table 11). Gross motor skills, unmet service needs, or overall sleep dysfunction did not significantly predict mean awake PP. However, unmet service needs, child daytime sleepiness, and subjective sleep quality significantly predicted mean asleep PP, after controlling for current hypertension and hypotension. Unmet supports and parental distress were not significant predictors of mean asleep PP. None of the psychosocial variables significantly predicted mean 24-hour PP.

Discussion

This study aimed to examine relationships between psychosocial factors and parent-reports, salivary biomarkers of stress, and level of CV activity among mothers of children with ASD. Mothers reported high levels of parenting stress, moderately high levels of anxiety, and low levels of depression. Although somewhat high levels of sAA were observed, cortisol levels were lower than average (Wust et al., 2000).

Salivary Biomarkers

Maternal cortisol levels were lower than averages reported by Wust et al. (2000), suggesting that mothers of children with ASD may have a blunted or dysregulated cortisol profile. This is consistent with Seltzer et al.'s (2010) findings that mothers of adolescents and adults with ASD (*M* = 24.7 years, *SD* = 7.24, range: 18–53 years) had significantly lower levels of daily cortisol than a control group. The present study extends this finding to mothers of younger children with ASD (*M* = 8.91 years, *SD* = 3.57, range: 3–17 years). Hypocortisolism, which has also been reported among individuals with PTSD, can be associated with decreased immunity, fatigue-like symptoms, and increased vulnerability to stress-related diseases (Heim et al., 2000). The present findings suggest that mothers of children with ASD of all ages, and not just mothers of adolescents and adults with ASD, may be at risk of dysregulated cortisol profiles and health problems associated with hypocortisolism as a result of chronic stress.

Unmet Service Needs

Unmet service needs have previously been reported to negatively affect parent-child relationships for mothers of children with ASD (Taylor & Seltzer, 2010). In the present study, increased number of unmet service needs was also found to predict higher maternal depression, parental distress, and SBP, MAP, and PP. This is of particular significance given that hypertension is a known risk factor for the development of coronary heart disease and stroke (WHF, 2013). Furthermore, a higher quantity of social support predicted lower levels of depression. These findings emphasise the importance of appropriate and adequate service provision for individuals with ASD, not only to ensure optimal outcomes for those individuals, but also to prevent stress-related health problems for parents.

Sleep Problems

Although sleep problems have been identified as a potential source of parenting stress (e.g., Davis & Carter, 2008), little research has investigated the physiological effects of sleep problems for mothers of children with ASD. In the present study, a higher number of child parasomnias (i.e., sleep disturbances) predicted a lower AUC_G, suggesting a link between sleep disruption and dysregulation of maternal cortisol awakening responses. Furthermore, higher child daytime sleepiness predicted lower 24-hour SBP and PP. Children who were sleepier during the day may have been less active, resulting in lower SBP and PP; previous research has reported lower stress among parents of less active children (McBride, Schoppe, & Rane, 2004). Higher parental sleep dysfunction also predicted higher anxiety and awake MAP. This highlights the importance of respite services for parents. In a study of parental needs, only 38% of parents of children with ASD were in receipt of home or respite support, and 93% rated it as a future need (Dillenburger et al., 2010). Also, behavioural interventions and interventions related to the circadian regulation of the sleep-wake cycle (e.g., melatonin) have documented success in improving child sleep habits (Malow et al., 2012; Richdale, 1999). Based on the present findings, respite services, routine sleep assessments, and greater supports in managing sleep problems could have positive psychological and physical effects for parents of children with ASD.

ASD Diagnosis and Comorbid Diagnoses

In the present study, overall ASD severity did not significantly predict maternal stress. However, higher socialisation deficits significantly predicted higher parenting stress and parent-child dysfunctional interactions. Thus, of the core deficits in ASD (i.e., socialisation deficits, communication deficits, and stereotyped behaviours), the present study found that socialisation deficits were the most significant source of stress for mothers. With respect to the ASD diagnosis, earlier detection of a problem predicted lower asleep DBP and MAP, and a higher night-time reduction in DBP. Osborne et al. (2008) previously reported a possible contra-indication of early ASD diagnosis. Parenting stress declined from parents first detecting a problem to the time they received the diagnosis and then remained static, such that earlier diagnosis was associated with higher parenting stress. In the present study, mothers first noticed a problem when their child was on average 21.12 months old (*SD* = 12.63, range: 0–66 months), and waited an average of 31.30 months (*SD* = 30.51, range: 4–156 months) to obtain a diagnosis. Similar to Osborne et al.’s (2008) study, the present findings may suggest that earlier detection of a problem gave mothers a chance to come to terms with their child’s ASD before obtaining an official diagnosis, such that earlier detection predicted lower maternal DBP and a higher night-time DBP reduction. However, it is also possible that later detection of a problem by parents was linked to their use of self-blame as a coping strategy, which was found to predict higher maternal anxiety, depression, and parental distress in the present study. Further research would be useful to determine the relationship between the age of the child when a problem is first detected, the duration it took to obtain a diagnosis, and maternal stress and wellbeing.

The majority of comorbid diagnoses did not predict maternal stress. However, higher levels of oppositional behaviour, one of the most common comorbid diagnoses for individuals with ASD (Simonoff et al., 2008), significantly predicted higher parenting stress, parent-child dysfunctional interactions, and difficult child scores. Consequently, parents of children with higher levels of oppositional behaviour may benefit from additional supports. Present findings with respect to ASD and comorbid diagnoses highlight the importance of supporting parents around the diagnostic process, and managing their child’s behavioural deficits (i.e., socialisation) and excesses (i.e., oppositional behaviour).

Adaptive Behaviour

Higher adaptive behaviour predicted lower parent-child dysfunctional interactions and difficult child scores in the present study, while higher fine motor skills predicted a lower AUC_I. Previous findings have been mixed regarding the role of adaptive behaviour deficits as a source of parenting stress. The present findings are consistent with reports that higher adaptive behaviour is associated with lower parental stress (e.g., Hall & Graff, 2011). High levels of adaptive deficits often experienced by individuals with ASD can affect all areas of their lives, including self-help, leisure, and hygiene (Matson et al., 2012), and can result in greater dependency on parents. The present study extends earlier findings, suggesting that adaptive functioning, particularly fine motor skills, can affect maternal cortisol levels, in addition to parenting stress. Many behavioural interventions can improve adaptive skills among individuals with ASD (Matson et al., 2012), and these findings suggest this is an important skillset to teach individuals with ASD, not only to improve quality of life for individuals with ASD, but also for their parents.

Coping Strategies

In the present study, only the maladaptive coping strategies of self-blame and behavioural disengagement were significant predictors of maternal outcomes. Greater use of self-blame predicted higher levels of anxiety, depression, and parental distress. It is known that many parents of children with ASD experience feelings of guilt (Weiss, 1991), and the present findings suggest that self-blame may exacerbate anxiety, depression, and parental distress. Additionally, higher levels of behavioural disengagement predicted higher 24-hour SBP, a risk factor for the development of CV disease (Sapolsky, 2004). This link between maladaptive coping strategies and negative psychological and physical outcomes suggests that parents of children with ASD may benefit from interventions that focus on emotional and cognitive-control strategies, such as Acceptance and Commitment Therapy (ACT; Blackledge & Hayes, 2006). Blackledge and Hayes (2006) reported that an ACT intervention reduced psychological avoidance and improved psychological outcomes for parents of children with ASD. Their ACT intervention focused on accepting unpleasant emotions and defusing from difficult thoughts while focusing on personal values. Interventions such as ACT may help parents with managing their emotions, reducing negative outcomes that were associated with the use of self-blame and behavioural disengagement in the present study.

Limitations and Future Research Directions

Sampling bias is a potential issue in the present study. Because participants were predominantly recruited from support groups and online organisations, they may have been more proactive than other parents in seeking help and supports. However, participants were also recruited from other locations, such as schools, in an attempt to recruit a representative sample. Salivary data were only available for a subset of the sample, possibly contributing to selection bias, as well as contributing to reduced statistical power. Also, compliance with saliva collection guidelines was not measured directly (e.g., using MEMS® TrackCaps technology), although salivary data were screened for compliance based on parent-reports. Finally, the study included only mothers, and as such, findings may not generalise to fathers of children with ASD. Research is needed to investigate physiological stress markers among fathers of children with ASD, which may result in more effective delivery of supports for parents. Moreover, given the findings of low cortisol levels among mothers of children with ASD, future research should determine whether fathers of children with ASD also have dysregulated cortisol function. Chapter 3 will replicate the present study with mother-father dyads of children and adolescents with ASD, in order to identify differences in maternal and paternal physiological responses to chronic stress.

Chapter 3: Study 2

Parent-Report and Physiological Measurements of Stress in Mothers and Fathers of Children with Autism Spectrum Disorders

As reported in Chapter 2, parents of children with ASD often experience high levels of stress (e.g., Eisenhower et al., 2005), and may be at risk of health problems as a result. However, the majority of the research to date has focused on mothers. Because mothers and fathers do not necessarily experience the same effects as a result of parenting a child with ASD, research investigating stress among mothers of children with ASD may not be generalisable to fathers.

Research among Mothers and Fathers

Fathers are under-represented in research on developmental psychopathology (Phares, Fields, Kamboukos, & Lopez, 2005) and paediatric research (Phares, Lopez, Fields, Kamboukos, & Duhig, 2005). The focus on mothers may reflect the assumption, based on traditional societal norms, that fathers do not spend a significant amount of time caring for their child, and that mothers are always the primary caregivers (Phares, 1992). Some previous research has supported these assumptions, reporting higher levels of parental involvement for mothers than fathers (e.g., Heller, Hsieh, & Rowitz, 1997; Konstantareas & Homatidis, 1992). However, Simmerman, Blacher, and Baker (2001) found that fathers of children with severe ID tended to be substantially involved in caregiving, and mothers reported high levels of satisfaction with the extent of fathers’ help within the family. Given that relevant evidence suggests that many fathers are substantially involved in caring for their children, it is important that fathers are also included in parental stress research.

Some research has reported high levels of stress, anxiety, and depression among fathers of children with ASD (MacDonald, Hastings, & Fitzsimons, 2010). However, mothers have been reported to have higher levels of stress than fathers (e.g., Tehee, Honan, & Hevey, 2009), which may reflect different levels of parental involvement. Additionally, in studies that have included both mothers and fathers of children with

ASD, different sources of stress have sometimes been identified for each. Hastings (2003) found that child behaviour problems did not affect paternal stress, but did affect maternal stress levels. They also found that mothers reported higher levels of anxiety than fathers, although mothers and fathers did not differ in their reports of depression and stress. However, Davis and Carter (2008) identified different aspects of behavioural problems that were sources of stress for mothers and fathers. The child's difficulties with self-regulation such as sleeping problems were associated with stress for mothers, while externalising behaviours such as stereotypy were associated with stress for fathers. Furthermore, partners' mental health has also been identified as a potential source of stress (Hastings, 2003; Hastings et al., 2005b). The provision of appropriate support, including interventions to reduce stress, requires that differences between mothers and fathers are taken into consideration. Relying on maternal stress research to develop supports and interventions for fathers may result in fathers being ineffectively supported.

Salivary Biomarkers of Stress

One neglected area of potential important difference between mothers and fathers concerns physiological markers of chronic stress. Salivary cortisol (Adam & Kumari, 2009) and sAA (Rohleder & Nater, 2009) can provide valuable information about HPA-axis and SNS activity, respectively, which are activated in response to stress. There is growing evidence that parents of children with ASD may experience hypocortisolism (e.g., Lovell et al., 2012; Lovell, Moss, & Wetherell, 2013; Ruiz-Robledillo et al., 2013; Seltzer et al., 2010). However, previous studies have focused primarily on mothers. Seltzer et al. (2010) included mothers only, Lovell et al. (2013) included only one father, and Lovell et al. (2012) included only six (13%) fathers. Furthermore, no mother–father comparisons were conducted in those studies.

Ruiz-Robledillo et al. (2013) included a similar number of mothers (*n* = 7) and fathers (*n* = 5) in the supported caregiver group, but a higher number of mothers (*n* = 9) than fathers (*n* = 3) in the non-supported group, and found no significant gender differences in cortisol levels. Further research is necessary to investigate possible differences in cortisol activity between mothers and fathers of children with ASD.

CV Assessment

As discussed previously, ABP monitoring, which enables repeated observations of BP and HR in the natural environment, can provide useful information about CV responses to chronic stressors experienced in real-life situations (Klaver et al., 1994). To our knowledge, only one study has used ABP monitoring among parents of children with ASD. Gallagher and Whiteley (2012) used ABP monitoring to compare CV activity between caregivers of children with developmental disabilities (DD), including ASD, and a control group of caregivers of typically developing children. They reported that caregivers of children with DD had higher mean SBP and poorer psychosocial functioning than caregivers of typically developing children. However, only 13 of the 35 caregivers included in the study were caring for a child with ASD, and only four of the caregivers were fathers. Thus, CV responses to daily stressors among parents of children with ASD still remain relatively unexplored.

There are two important aspects of ABP, in particular, that have not yet been explored in parents of children with ASD to our knowledge. The diurnal CV profile usually includes a 10–20% fall in BP during sleep, known as “dipping”. Non-dipping (i.e., no fall in nocturnal BP), extreme dipping (i.e., a larger than 20% fall in nocturnal BP), and reverse dipping (i.e., an increase in nocturnal BP) are reported to be associated with increased risk of end-organ damage, stroke, and CV complications (e.g., Kario et al., 2001; Kario & Shimada, 2004; O'Brien et al., 2000). ABP monitoring is the only non-invasive way to measure dipping.

BP and HR variability are also of potential significance. BP variability can be measured using standard deviations and variation coefficients based on invasive intra-arterial BP monitoring (Mancia et al., 1983). HR variability can also be measured by using 24-hour electrocardiograms (ECGs) to measure beat-to-beat changes in HR (Ewing, Neilson, Shapiro, Stewart, & Reid, 1991). Higher BP variability and lower HR variability have been found to independently predict end-organ damage and CV mortality using these methods (Algra, Tijssen, Roelandt, Pool, & Lubsen, 1993; Frattola, Parati, Cesare, Albini, & Mancia, 1993; Tsuji et al., 1994). It is unclear what mechanism is responsible for the correlation between HR variability and CV mortality, but it has been hypothesised that reduced HR variability reflects increased sympathetic activity, a risk factor for the development of myocardial ischemia (Tsuji et al., 1994). Links between BP and HR variability and CV mortality have also been reported in research using standard deviations of BP and HR measured by non-invasive ABP monitoring. Kikuya et al. (2000) reported that higher BP variability and lower HR variability, using standard deviations measured every 30 minutes by ABP monitoring, predicted higher CV mortality. BP variability measured using non-invasive ABP monitoring has also been reported to be associated with hypertension-related target organ damage, and higher incidence of CV morbid complications of hypertension (Frattola et al., 1993; Verdecchia et al., 1996). Thus, using standard deviations based on readings from ABP monitoring provides a way to measure BP and HR variability that is less invasive than intra-arterial BP monitoring or ECG, but still has prognostic significance. Examining CV activity, particularly with respect to nocturnal dipping and CV variability, may provide useful insights into different physiological effects of parenting a child with ASD for mothers and fathers.

Aims of the Present Study

The present study compared levels of parenting involvement, parent-reported anxiety, depression, parenting stress, salivary biomarkers, and ambulatory hemodynamic activity between mothers and fathers of children with ASD. In addition, parent-reported coping strategies-, social supports, and sleep problems were compared between the two groups.

Method

Participants

The sample initially comprised 20 mother-father dyads of children with an independent diagnosis of an ASD. One dyad failed to return questionnaires within the allotted time and was excluded, leaving a final sample of 19 dyads. Participants were recruited through ASD support groups, schools, conferences, media, and online support groups and forums. The mothers in the present study were also included in Study 1.

Parents. The mean age of the mothers was 41.05 years (*SD* = 4.55, range: 32–48 years), and the mean age of the fathers was 42.16 years (*SD* = 5.46, range: 32–51 years). Fourteen mothers (74%) reported that they were not in employment, with three (16%) reporting that they were in part-time employment, and two (11%) reporting full-time employment. Three fathers (16%) reported that they were not in employment, while one was in part-time employment, and 15 (79%) were in full-time employment.

Children. Four (21%) of the children were girls, and 15 (79%) were boys. Children had a mean age of 88.11 months (*SD* = 40.27, range: 33–161 months).

Children received their ASD diagnoses when they were on average 44.11 months old (*SD* = 20.01, range: 24–96 months). Autism was reported as the primary diagnosis for 16 (84%) of the children, while one was reported as having Asperger's syndrome, and two (11%) were reported as having PDD-NOS. Children were reported as having an average of 2.58 (*SD* = 1.17) diagnoses, ranging from 1–5 diagnoses. Ten children (53%) were reported as having an ID, with five (26%) reported as having a mild ID, three (16%) reported as having a moderate ID, and two (11%) reported as having a severe ID.

Ethics

Ethical approval was the same as in Study 1.

Materials

All materials were the same as in Study 1.

Procedure

The procedure was the same as in Study 1.

Analysing Salivary Analytes

Salivary analyses were the same as in Study 1. Cortisol values were available for 11 mothers and 10 fathers, and sAA values for 11 mothers and 10 fathers after applying the exclusion criteria outlined in Study 1. However, only dyads that had valid cortisol or sAA values available for both members of the dyad were included in those statistical analyses. Seven and nine dyads were available for inclusion in cortisol and sAA statistical comparisons, respectively.

Data Analysis

As in Study 1, cortisol and sAA data were positively skewed, and were natural-log and square-root transformed, respectively (Adam & Kumari, 2009; Granger et al., 2007). Outliers identified on boxplots were then recoded to the next highest value in the distribution (Tabachnick & Fidell, 2001). Skewness statistics were less than 0.35 for all of the cortisol and sAA distributions after data transformation. For ease of interpretation, raw values are presented in tables and graphs.

For each stress measure, the father's score was subtracted from the mother's score for each dyad, providing a difference score for each. Dyad difference scores were then tested for significance of difference from zero, using one-sample *t*-tests. Alpha values were corrected using family-wise Bonferroni adjustment (see Table 13). Comparisons between mothers and fathers with respect to sleep problems (PSQI), quantity of unmet supports, health-relevant variables (exercise and cigarettes), and diary information (location, activities, and moods) showed that the two groups did not differ significantly on any of these variables, which therefore are not reported below. In order to examine relationships between parenting stress and partners' mental health (anxiety and depression), Pearson's *r* correlations were conducted between these variables for mothers and fathers.

Results

Descriptive Analysis

Mean group scores for mothers and fathers are reported in Table 12. Low levels of anxiety and depression were reported. Group anxiety and depression scores for fathers, and group depression scores for mothers, all fell within the normal range (0–7), and group anxiety scores for mothers fell within mild range (8–10; Zigmond & Snaith, 1983). However, 11 mothers (58%) had anxiety scores that fell within the moderate range (11–14). Group means indicated high levels of parenting stress. Overall parenting stress, difficult child, parental distress, and parent-child dysfunctional interaction scores all fell above the 85th percentile of the PSI for both mothers and fathers (with the exception of parental distress, which fell at the

Table 12

Descriptive Information for Parenting Responsibility, Parenting Stress, Anxiety, Depression, Cortisol, Alpha-Amylase, and Cardiovascular Measurements for Mothers (n = 19) *and Fathers* (n = 19)

	Mothers		Fathers	
	M *(SD)*	Range	M *(SD)*	Range
Parenting responsibility	44.56 (7.23)	25 – 57	29.72 (6.75)	22 – 51
Anxiety	10.21 (3.28)	5 – 16	6.37 (3.39)	0 – 14
Depression	7.26 (2.73)	2 – 11	5.00 (3.18)	1 – 14
Total parenting stress	107.84 (17.86)	85 – 146	95.21 (19.19)	60 – 134
Parent-child dysfunctional interaction	31.26 (7.29)	20 – 47	28.26 (6.50)	16 – 42
Difficult child	42.42 (6.12)	30 – 52	37.00 (8.94)	20 – 53
Parental distress	34.16 (8.59)	15 – 48	29.95 (7.86)	17 – 43
Defensive responding	20.42 (5.29)	8 – 28	18.00 (5.19)	10 – 27
Cortisol: Sample 1 (µg/dL)	0.50 (0.49) [a]	0.27 – 2.09	0.36 (0.18) [b]	0.04 – 0.81
Cortisol: Sample 2 (µg/dL)	0.52 (0.43) [a]	0.25 – 2.00	0.39 (0.17) [b]	0.06 – 0.62
Cortisol: Sample 3 (µg/dL)	0.45 (0.21) [a]	0.21 – 0.83	0.40 (0.22) [b]	0.13 – 0.87
Cortisol: Sample 4 (µg/dL)	0.40 (0.22) [a]	0.17 – 0.86	0.39 (0.17) [b]	0.18 – 0.72
Alpha-amylase (U/mL)	122.69 (82.33) [b]	34 – 308	169.46 (137.76) [c]	8 – 390
Mean 24-hour SBP (mmHg)	120.78 (11.45)	99 – 146	137.78 (15.57)	118 – 168
Mean SBP night-time reduction (%)	14.71 (5.93)	0 – 24	13.40 (7.35)	1 – 23.7
Mean 24-hour DBP (mmHg)	71.39 (8.24)	51 – 83	82.17 (11.49)	68 – 108
Mean DBP night-time reduction (%)	20.02 (6.38)	6 – 33	19.02 (9.08)	3 – 34
Mean 24-hour HR (bpm)	76.50 (9.12)	62 – 94	71.56 (7.74)	60 – 94
Mean 24-hour MAP (mmHg)	87.83 (9.12)	67 – 102	100.67 (12.36)	86 – 125
Mean 24-hour PP (mmHg)	49.33 (5.53)	42 – 65	55.89 (8.17)	46 – 78

Note. [a] n = 12. [b] n = 11. [c] n = 10.

70th percentile for fathers), indicating clinically significant levels of parenting stress (Abidin, 1995). Defensive responding, which is of concern when scores are below 10, did not appear to be a significant issue for mothers or fathers, with mean scores of 20.42 and 18.00, respectively.

Mean cortisol levels for both groups are illustrated in Figure 2, alongside average cortisol values reported by Wust et al. (2000). One-sample *t*-tests were conducted to compare cortisol values of mothers and fathers of children with ASD to Wust et al.'s (2000) normal cortisol values. Alpha values were corrected using family-wise Bonferroni adjustment ($\alpha = .0125$). For mothers, cortisol values immediately after waking did not significantly differ from the reported averages, *t* (15) = -0.24, *p* = .81. However, cortisol values 30 minutes, *t* (18) = -6.23, *p* < .001, and 45 minutes, *t* (18) = -6.16, *p* < .001, after waking were significantly lower than the reported averages. For fathers, all cortisol values were significantly lower than the reported averages: immediately after waking, *t* (17) = -11.78, *p* < .001; 30 minutes after waking, *t* (19) = -9.83, *p* < .001; and 45 minutes after waking, *t* (16) = -11.20, *p* < .001. Mean sAA levels for mothers (*M* = 116.92, *SD* = 81.00, range: 34–308 U/mL) and fathers (*M* = 169.46, *SD* = 137.76, range: 8–390 U/mL) fell above Salimetrics' expected adult mean of 92.40 U/mL (Salimetrics, 2012c). However, this difference did not reach statistical significance for mothers, *t* (12) = 1.05, *p* = .32, or fathers, *t* (10) = 1.77, *p* = .11.

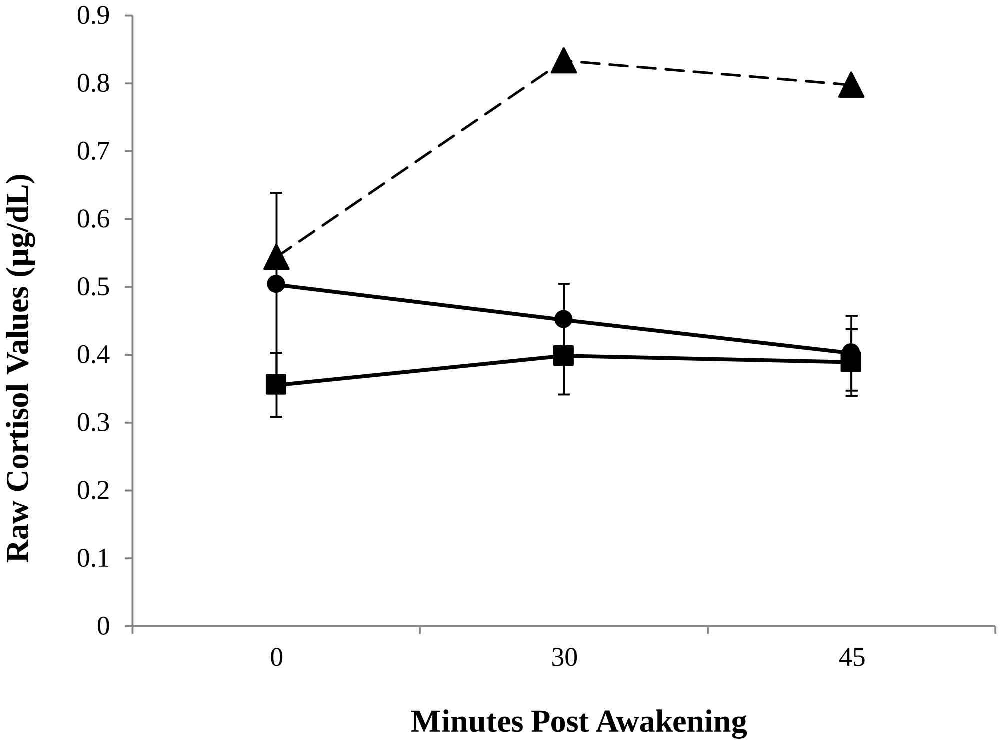

Figure 2. Post-awakening mean group cortisol values (µg/dL) of mothers (circles) and fathers (boxes) of children with ASD in the present study and as published by Wust et al. (2000; triangles and dashed line)

Note. Standard error bars are shown for cortisol values of mothers and fathers of children with ASD in the present study, but are not available for results published by Wust et al. (2000)

Mean 24-hour DBP for both groups, and mean 24-hour SBP for mothers fell within the normotensive range, defined by O'Brien et al. (2000) as a 24-hour SBP ≤ 135 mmHg and a 24-hour DBP ≤ 85 mmHg (see Table 12). Mean 24-hour SBP for fathers slightly exceeded the normotensive range, with mean 24-hour SBP for 11 fathers (58%) falling within the hypertensive range. DBP means were also more variable for fathers than mothers, with only one mother (5%), but seven fathers (37%), falling within the hypertensive DBP range.

Testing Dyad Difference Scores

As outlined above, dyad difference scores were calculated for each of the stress measures, and tested to see if they significantly differed from zero using one sample *t*-tests with family-wise Bonferroni adjustments. Results are reported in Table 13. As shown in Table 13, mothers reported higher levels of parenting responsibility than fathers. Similarly, mothers reported higher levels of anxiety, depression, overall parenting stress, and difficult child scores than fathers. However, there were no significant differences between mothers’ and fathers' parent-child dysfunctional interaction or parental distress scores. Similarly, there were no significant differences in AUC_G, $AUC_{I,}$ or sAA levels between mothers and fathers.

With respect to CV measures, there were no significant differences in SBP or DBP dipping (see Table 13). Standard deviations from awake and asleep SBP, DBP, and HR measures were used as estimates of BP and HR variability (Kikuya et al., 2000). Fathers had higher awake SBP, DBP, and HR variability, and higher asleep SBP variability, than mothers. Asleep DBP and HR variability did not significantly differ between mothers and fathers, although they were approaching significance, with higher variability for fathers than mothers.

As shown in Table 13, mothers reported significantly higher use of instrumental support, planning, and acceptance as coping strategies than fathers, while fathers reported significantly higher use of behavioural disengagement and humour than mothers. Mothers and fathers did not significantly differ on use of any other coping strategies. Fathers reported higher satisfaction with social supports than mothers. Although mothers and fathers did not significantly differ in terms of social support quantity, it was approaching significance, with mothers reporting a slightly higher quantity of social supports than fathers.

Table 13

Summary of One-Sample T-Tests for Dyad Difference Scores

Variable	Mean (SD)	*t*	*df*	Adjusted α [a]
Parenting responsibility	14.83 (13.58)	4.64*	17	.05
Anxiety	3.84 (3.86)	4.34*	18	.025
Depression	2.26 (2.74)	2.64*	19	.025
Overall parenting stress	12.63 (18.69)	2.95*	19	.013
Parent-child dysfunctional interaction	3.00 (8.03)	1.63	19	.013
Difficult child	5.42 (8.34)	2.83*	19	.013
Parental distress	4.21 (8.72)	2.11	19	.013
SBP night-time reduction (%)	1.31 (7.76)	0.71	18	.025
DBP night-time reduction (%)	1.00 (9.35)	0.45	18	.025
Awake SBP variability	-5.36 (7.46)	-3.14*	18	.008
Asleep SBP variability	-6.65 (7.63)	-3.70*	17	.008
Awake DBP variability	-4.97 (6.97)	-3.11*	19	.008
Asleep DBP variability	-4.76 (7.18)	-2.81[b]	18	.008
Awake HR variability	-5.76 (7.01)	-3.58*	19	.008
Asleep HR variability	-5.56 (8.39)	-2.81 [b]	18	.008
AUC_G	0.25 (0.94)	0.72	7	.025
AUC_I	-0.01 (0.81)	-0.02	7	.025
Alpha-amylase	-3.11 (8.06)	-1.16	9	.05
Active coping	0.90 (2.02)	1.93	19	.005
Denial	0.63 (1.46)	1.88	19	.005
Substance use	0.37 (1.86)	0.86	19	.005
Emotional support	0.58 (2.27)	1.11	19	.005
Instrumental support	2.90 (1.63)	7.74*	19	.005
Behavioural disengagement	-2.84 (1.92)	-6.44*	19	.005
Planning	2.53 (3.04)	3.62*	19	.005
Humour	-2.37 (2.17)	-4.77*	19	.005
Acceptance	3.11 (3.03)	4.46*	19	.005
Religion	0.32 (2.19)	0.63	19	.005
Self-blame	0.90 (2.16)	1.81	19	.005
Social support quantity	4.88 (8.85)	2.27 [b]	17	.025
Social support quality	-2.41 (4.05)	-2.46*	17	.025
Quantity of illnesses	1.05 (1.22)	3.75*	19	.013
Alcohol intake	-1.31 (1.94)	-3.00*	17	.013

Note. *Significant at the adjusted α value denoted. [a]Alpha (α) value adjusted using family-wise Bonferroni correction. [b]Approaching significance.

Comparisons were also made with a number of health-relevant variables, and significant differences are reported in Table 13. Mothers were found to have more illnesses than fathers, while fathers were found to drink more alcohol than mothers. Mothers and fathers did not differ on any other health-relevant variables.

Dipping Status

Because no significant differences were observed between the size of SBP or DBP dipping between mothers and fathers, dipping patterns were explored further, using the following criteria: dipping was defined as a ≥ 10% but < 20% fall in nocturnal BP, non-dipping was defined as a < 10% but > 0% fall, extreme dipping was defined as a nocturnal BP fall ≥ 20%, and reverse dipping was defined as a nocturnal BP > 0% (Xu, Zhang, & Tan, 2013). Pearson's chi-square test for independence indicated no significant association between gender and dipping status for SBP, χ^2 (2, *N* = 36) = 0.66, *p* = .72, or DBP, χ^2 (2, *N* = 36) = 0.50, *p* = .78.

Furthermore, parenting stress, anxiety, and depression were entered into separate 2 (gender) x 4 (dipping status) way analysis of variance (ANOVA) to determine if there was an interaction between dipping status and parent-reported stress. However, no significant effects were observed (*p* = *ns*). The majority of mothers and fathers were normal SBP dippers. Only three mothers (16%) and five fathers (26%) were found to be non-dippers for SBP, and only two mothers (11%) and three fathers (16%) found to be non-dippers for DBP. However, five mothers (26%) and four fathers (21%) were found to be extreme SBP dippers, and 10 mothers (53%) and eight fathers (42%) were found to be extreme DBP dippers. No reverse dipping was observed.

Partners' Mental Health

Pearson's *r* correlations were conducted between the parenting stress subscales, to determine if they were correlated with partner anxiety and depression scores. As shown in Table 14, parenting stress for mothers was not significantly correlated with partners' anxiety and depression scores. However, parenting stress for fathers was positively correlated with partners' anxiety and depression.

Table 14

Summary of Correlations between Parental Stress (PSI) and Partner's Anxiety and Depression (HADS), for Mothers (*n* = 19) and *Fathers* (*n* = 19)

Variable	Mothers		Fathers	
	Partner's Anxiety	Partner's Depression	Partner's Anxiety	Partner's Depression
Parenting stress	.06	.06	.62**	.63**
Parent-child dysfunctional interactions	-.18	-.18	.43	.49*
Difficult child scores	.15	.13	.54*	.50*
Parental distress	.17	.18	.53*	.55*

Note. **p* < .05. ** *p* < .01.

Child Variables

Two separate analyses were used to examine the effects on parental stress of the following child variables: behaviour problems, ADHD and related symptoms, ASD severity, adaptive behaviour, sleep problems, and unmet service needs. First, the child variables were converted into dichotomous variables using a median split. Each of the stress measures were then entered into a separate 2 (gender) x 2 (child variable) ANOVA. The alpha level was adjusted using family-wise Bonferroni correction (α = .002). Significant differences were not found at this level. Further analyses were conducted in case any information had been lost as a result of dichotomising the child variables. One-way between-subjects ANOVA analyses were conducted separately for each of the stress measures. Next, these analyses were repeated in an analysis of covariance (ANCOVA), with child variables entered individually as covariates in separate analyses. This enabled comparison of patterns between the ANOVA and ANCOVA as a means of gauging the influence of variables included in the ANCOVA analyses. Bonferroni correction again resulted in an adjusted alpha level of .002. No significant differences were found for the child variables, so these are not presented here.

Discussion

The present study compared parent-report and physiological measures of stress between mothers and fathers of children with ASD. Mothers reported significantly higher levels of parenting responsibility, anxiety, depression, parenting stress, difficult child scores, and parental distress than fathers. However, fathers had significantly higher BP and HR variability than mothers. Mothers and fathers did not differ significantly with respect to parent-child dysfunctional interactions, parental distress, SBP or DBP dipping, AUC_G, AUC_I, or sAA levels. Furthermore, no significant differences were found between mothers and fathers with respect to child variables, suggesting that they were similarly affected by those variables.

Perceived Parenting Stress, Anxiety, and Depression

Mothers reported significantly higher levels of parenting responsibility than fathers, which is consistent with previous findings that mothers are more often the primary caregiver (e.g., Heller et al., 1997; Konstantareas & Homatidis, 1992).

Higher parenting responsibility among mothers may have contributed to their reported higher levels of anxiety, depression, and parenting stress, as has been previously reported (e.g., Hastings, 2003; Moes, Koegel, Schreibman, & Loos, 1992). However, there were no significant differences between mothers' and fathers' reports of parent-child dysfunctional interactions or parental distress, suggesting that having a child with ASD may influence the parent-child relationship, irrespective of the parent's level of involvement.

Coping strategies and social support may also contribute to differences in levels of anxiety, depression, and parenting stress. Mothers reported higher use of instrumental support, planning, and acceptance than fathers. This may again reflect the greater level of parenting responsibility assumed by mothers, with mothers being more responsible than fathers for planning and seeking advice in relation to the child. Fathers, on the other hand, reported higher use of behavioural disengagement and humour than mothers, which may be linked with the lower levels of parenting responsibility, anxiety, depression, and parenting stress reported by fathers. It is possible that fathers who disengage or use humour to cope may also be more likely to report lower levels of anxiety, depression, or parenting stress, or that fathers who were less involved and experiencing less stress found it easier to disengage and use humour than mothers. Mothers tended to report higher levels of social support than fathers, although the difference fell short of statistical significance. Fathers, on the other hand, reported significantly higher satisfaction with social supports. These findings suggest that increased support may be beneficial in reducing anxiety, depression, and parenting stress, particularly for mothers.

Partners' Mental Health

Partners' mental health has previously been identified as a potential source of stress for parents of children with ASD. Hastings (2003) reported that fathers' mental health (i.e., anxiety and depression) was associated with mothers' stress, although mothers' mental health was not associated with fathers' stress. Hastings et al. (2005b) found that both mothers' and fathers' stress were predicted by their partners' depression. In the present study, mothers' stress was not significantly correlated with fathers' mental health (i.e., anxiety or depression); however, fathers' stress was significantly correlated with mothers' mental health. Mothers did report significantly higher levels of anxiety, depression, and parenting stress than fathers in the present study, whereas anxiety and depression levels did not significantly differ between groups in Hastings' (2003) study. Mothers only reported higher depression symptoms, but not anxiety or stress, in Hastings et al.'s (2005b) study. The mixed findings in relation to parental stress and partners' mental health may depend on the extent to which the partner is experiencing anxiety and depression. The present study does provide further evidence of a relationship between parental stress and partners' mental health. This warrants further investigation to determine the causal nature of this relationship, and to determine if partners' mental health is a source of stress for both mothers and fathers of children with ASD. In this case, optimal outcomes may be achieved from stress interventions that involve both mothers and fathers.

Cortisol

Previous research has indicated that parents of children with ASD may experience hypocortisolism (Seltzer et al., 2010). Ruiz-Robledillo et al. (2013) also reported that non-supported caregivers of individuals with ASD had lower cortisol awakening responses than supported caregivers and non-caregivers. In the present study, there were no significant differences between mothers and fathers with respect to AUC_G or AUC_I. Both groups of parents had lower than average levels of cortisol (Wust et al., 2000). These findings provide further evidence that parents of children with ASD experience blunted cortisol activity, and that risk of hypocortisolism is not limited to mothers of children with ASD. Conversely, sAA levels for both mothers and fathers were found to be somewhat higher than Salimetrics’ (2012c) reported averages, although this did not reach statistical significance. This is similar to previous reports of increased SNS activity and blunted cortisol responses among individuals with PTSD (Fries et al., 2005). Hypocortisolism is associated with an increased risk of health problems (Heim et al., 2000), and Ruiz-Robledillo et al. (2013) reported a link between lower cortisol awakening responses and higher somatic symptoms among caregivers. Fries et al. (2005) propose that hypocortisolism is a protective response that reduces the harmful effects of glucocorticoid responses to daily stressors, with the drawback of symptoms such as pain, fatigue, and high stress sensitivity. Preventing and managing chronic stress may be the most effective approach to dealing with hypocortisolism and preventing associated health problems among parents of children with ASD. For instance, interventions such as cognitive-behavioural therapy (CBT) or ACT may help parents to manage stress (e.g., Blackledge & Hayes, 2006; Gaab et al., 2003). Additionally, as Ruiz-Robledillo et al. (2013) found that cortisol levels were blunted only for parents of children who were not receiving support, these findings highlight the importance of providing necessary services for individuals with ASD, in addition to providing supports for their parents.

BP and HR Variability

High BP and low HR variability using standard deviations of readings obtained from ABP monitoring have been found to predict CV mortality (Kikuya et al., 2000; Pickering & James, 1994). Research has demonstrated that HR and BP variability are both independent risk factors for CV mortality (Kikuya et al., 2000). In the present study, fathers had significantly higher BP and HR variability than mothers. This suggests that fathers may be at higher risk of CV disease than mothers as a result of higher BP variability, but that mothers may be at higher CV risk than fathers as a result of lower HR variability. Previous research has reported that, although antihypertensive medication may be effective in lowering BP, it may not be effective in regulating BP variability (Garcia-Vera, Sanz, & Labrador, 2004). Garcia-Vera et al. (2004) found that stress management training could be effective in reducing BP variability. Such findings provide further support for the need for stress management programmes for parents of children with ASD.

With respect to night-time reduction in SBP and DBP, there were no significant differences between mothers and fathers in terms of the extent of dipping or type of dipping pattern. Although the majority of mothers and fathers were normal dippers, three mothers and five fathers were found to be non-dippers for SBP, and two mothers and three fathers were non-dippers for DBP. Five mothers and four fathers were found to be extreme SBP dippers, while ten mothers and eight fathers were found to be extreme DBP dippers. Extreme dipping has been found to be associated with increased risk of stroke and silent cerebrovascular disease (Hoshide et al., 2002; Kario & Shimada, 2004). Antihypertensive medication has also been reported to be less effective for extreme dippers than normal dippers (Hoshide et al., 2002), and treatment of hypertension in individuals with extreme dipping may exacerbate night-time hypotension, increasing risk of stroke at night (Kario & Pickering, 2000). Furthermore, higher BP variability and extreme dipping together may accelerate target-organ damage (Shimada & Kario, 1997). Given the associated health consequences, combining stress management training with routine health assessments, with a particular emphasis on ambulatory BP, may be beneficial in assessing, treating, and possibly preventing CV health problems for parents of children with ASD.

Limitations and Future Research

A number of issues potentially affect the generalisability of the present study. The sample size was relatively low, with only 19 dyads participating. Additionally, only a subset of 7 and 9 dyads were available for inclusion in the cortisol and sAA analyses, respectively, reducing statistical power. Furthermore, the present study did not use a control group of parents of typically developing children, possibly limiting interpretation of the results. However, to our knowledge, this is the first study to directly compare cortisol, alpha-amylase, and ambulatory blood pressure and heart rate between mothers and fathers of children with ASD. Indicators of chronic stress, including BP and HR variability, BP dipping, and hypocortisolism, would suggest the need for further investigation of parents of children with ASD, as well as parents of typically developing children, in order to better understand the extent of possible threats to health associated with chronic parenting challenge. Thus, Chapter 4 will extend the present study to investigate differences between parents of children with ASD and parents of typically developing children.

Chapter 4: Study 3

Comparison of Parent-Report and Physiological Measurements of Stress between Parents of Children with and without Autism Spectrum Disorders

As reported in Chapters 2 and 3, parents of children with ASD often experience high levels of stress, which in turn can affect parental health. However, all parenting is inherently stressful, because of normative events experienced by every parent (Glasberg et al., 2006). Thus, when researching stress in parents of children with ASD, it is important to include parents of typically developing children, in order to control for stress related to normative parenting. Previous research has consistently reported higher levels of parenting stress among parents of children with ASD than parents of typically developing children, and children with other disabilities (Hayes & Watson, 2013). Hayes and Watson (2013) reported a large effect size in a meta-analysis of studies comparing parenting stress in parents of children with and without ASD, suggesting that assessment and intervention are warranted for stress among parents of children with ASD. Because parents of children with ASD have been found to experience heightened levels of stress, and chronic stress can have adverse effects on a range of psychological and physical outcomes (Loft et al., 2007), it is important to consider the physiological effects of chronic stress among parents of children with ASD compared to parents of typically developing children.

Parent-Reported Health

Studies that have compared health between parents of children with ASD and parents of typically developing children have usually employed parental self-reports of health and wellbeing. Parents of children with ASD have generally been found to report more illnesses or poorer health than parents of typically developing children. For example, Allik et al. (2006) found that mothers of children with Asperger’s syndrome or high-functioning autism reported poorer physical health than mothers of typically developing children. Similarly, Eisenhower et al. (2005) found that mothers of children with developmental delays at age 3 reported poorer concurrent and later physical health than mothers of typically developing children. Mothers of children with delayed development and behaviour problems were found to be at highest risk of poor physical health. Similarly, Smith, Seltzer, and Greenberg (2012) found that mothers of children with ASD reported a higher proportion of days with headaches, backache, muscle soreness, fatigue, and hot flashes than mothers of children without ASD. Parents of children with ASD have also been found to report poorer health than parents of children with other disabilities. For instance, Mugno et al. (2007) found that mothers of children with ASD reported poorer physical health, psychological state, and quality of life than parents of typically developing children and children with cerebral palsy or ID. However, while parents of children with ASD have been found to report poorer health than other parents, very little research into physiological indices has been conducted to support the parent-report literature, and to identify the mechanisms by which parents may be at risk of poorer health.

Salivary Biomarkers of Stress

As discussed in the previous chapters, only a small body of literature has researched salivary biomarkers of stress among parents of children with ASD. To our knowledge, levels of sAA in parents of children with ASD have not been investigated to date. Of the studies that have measured cortisol among parents of children with ASD, only a subset included a control group of parents of typically developing children. Marinovic-Curin et al. (2008) measured cortisol in nine families of children with ASD and seven families of typically developing children, and found that cortisol levels did not significantly differ between the two groups. Conversely, Seltzer et al. (2010) found that mothers of adolescents and adults with ASD had significantly lower daily cortisol levels, while Seltzer et al. (2009) found that parents of individuals with disabilities had less pronounced cortisol decline than a control group. Similarly, Ruiz-Robledillo et al. (2013) found that non-supported caregivers had more gastrointestinal symptoms and a blunted cortisol response compared to supported caregivers and non-caregivers. Thus, further research is necessary to determine if parents of children with ASD are at higher risk than parents of typically developing children of experiencing hypocortisolism which, as discussed in previous chapters, can be associated with increased risk of stress-related diseases and decreased immunity (Heim et al., 2000).

Ambulatory Blood Pressure

Similarly, very little research has investigated differences in ambulatory CV activity between parents of children with ASD and typically developing children. To date, only one study has investigated ambulatory BP in parents of children with ASD. Gallagher and Whiteley (2012) compared ambulatory BP between parents of children with developmental disabilities (including children with ASD), and parents of typically developing children. They found that parents of children with DD had higher daily BP than control parents. This was an important finding, given that hypertension is a known risk factor for future development of illnesses such as stroke and coronary heart disease (Sapolsky, 2004). However, given that this is the only study to date that has compared ambulatory BP among parents of children with and without ASD, additional research is needed to determine if Gallagher and Whiteley’s (2012) findings can be replicated, and to further understand the effects of parenting a child with ASD on CV activity.

Aims of the Present Study

Further research is needed to identify differences in physiological activity between parents of children with ASD and parents of typically developing children. The present study aimed to compare parent-report, salivary biomarkers of stress, and ambulatory hemodynamic activity between parents of children with ASD and a control group of parents of typically developing children, matched by parent and child age and gender. In particular, the study aimed to identify if parents of children with ASD had higher parent-reported stress, anxiety, depression, CV activity, and sAA levels, and blunted cortisol activity, compared to parents of typically developing children.

Method

Participants

The sample comprised two groups. The ASD group included the 19 mother-father dyads of children and adolescents with a diagnosis of an ASD from Study 2 (see Chapter 3 for participant details). Participants for the ASD group were recruited through ASD support groups, schools, conferences, media, and online support groups and forums. The control group included 19 mother-father dyads of typically developing children or adolescents matched to the dyads in the ASD group by child and parent gender and age. Participants for the control group were recruited from mainstream schools, parent groups, and personal contacts. Participants did not receive any payment for taking part in the study.

Inclusion criteria. Inclusion criteria for the control group were that the typically developing child was the same gender and aged within 18 months of the child with ASD. The matched children differed in age by an average of 7.21 months (*SD* = 5.27, range: 1–17 months). Additionally, parents in the control group were matched by gender, and were matched within 10 years of age to the parents of children with ASD. The matched parents differed in age by an average of 3.31 years (*SD* = 1.35, range: 0–6 years).

Control group. The mean age of the mothers was 36.94 years (*SD* = 4.53, range: 27–46 years), and the mean age of the fathers was 38.83 years (*SD* = 5.66, range: 28–50 years). Five (26%) of the mothers reported that they were not in employment, with nine (47%) reporting that they were in part-time employment, and five (26%) reporting that they were in full-time employment. One father reported being unemployed, with three (16%) reporting that they were in part-time employment, and 15 (79%) reporting that they were in full-time employment.

Four of the children (22%) in the control group were girls, and 15 (79%) were boys. Children had a mean age of 81.5 months (*SD* = 39.49 range: 25–158 months). Children in the control group had no diagnoses of any disability or serious illness. See Table 15 for mean scores of children from the ASD and control groups on psychosocial variables.

Ethics

Ethical approval was the same as in Study 1.

Materials

The materials were the same as in Study 1.

Procedure

The procedure was the same as in Study 1.

Analysing Salivary Analytes

Salivary analyses were the same as in Study 1. After applying the exclusion criteria outlined in Study 1, sufficient data were available to include 12 mothers and 11 fathers from the ASD group, and 13 mothers and 11 fathers from the control group, in the cortisol (area under the curve) analyses. Sufficient data were available to conduct sAA analyses for 11 mothers and 10 fathers in the ASD group, and 11 mothers and fathers in the control group.

Data Analysis

As in Study 1, cortisol and sAA data were positively skewed, and were natural-log and square-root transformed, respectively (Adam & Kumari, 2009; Granger et al., 2007). Outliers identified on boxplots were then recoded to the next highest value in the distribution (Tabachnick & Fidell, 2001). Skewness statistics were less than 0.35 for all of the cortisol and sAA distributions after data transformation. For ease of interpretation, raw scores are presented in figures and tables.

Two-way ANOVA or ANCOVA analyses were conducted to evaluate the effects of group and gender on the following variables: HADS and PSI subscales, AUC_G, $AUC_{I,}$ raw cortisol values, sAA, mean CV measures, CV variability, SSQ, Brief COPE and PSQI subscales, and demographic and cuff inflation diary information. Alpha values were corrected using family-wise Bonferroni adjustment. Bonferroni-adjusted alpha values are presented in all ANOVA or ANCOVA tables. To determine if an ANOVA or ANCOVA would be used, Pearson's *r* correlations were conducted to determine if potential covariates were likely to influence the outcome variable. Potential covariates that were not correlated with the outcome variable were excluded. Potential covariates that *were* correlated with the outcome variable were then screened to determine if they met the ANCOVA assumptions of linearity, homogeneity of regression slopes (i.e., by customising the ANCOVA model to look at the independent variable x covariate interaction), and independence of covariates (i.e., Pearson's *r* correlations were conducted between covariates to ensure they were not highly correlated with each other; Field, 2005). If the ANCOVA assumptions were not met, ANOVA analyses were conducted instead of ANCOVA. For variables where ANCOVA analyses were conducted (i.e., HADS anxiety and depression, sAA, mean asleep SBP, mean 24-hour HR, and mean awake, asleep, and 24-hour DBP and MAP), the covariates that were included have been identified below the corresponding tables.

For nominal variables, Pearson's chi-square test for independence was conducted to test for an association between the participant group and the nominal variable (i.e., employment status, cortisol responding vs. non-responding, SBP and DBP dipping status, and medication usage).

Results

Descriptive Statistics

Mean scores for child variables from both groups are presented in Table 15. Higher scores were reported for children from the ASD group than children from the control group for all variables except the Vineland, with higher adaptive behaviour reported for children in the control group.

Mean scores for parent variables are presented in Tables 16–28. Mean anxiety and depression scores fell within the normal range (0–7) for the control group (Zigmond & Snaith, 1983). Mean depression scores for the ASD group also fell within the normal range, with mean anxiety scores falling within the mild range (8–10). However, anxiety scores for 13 parents (34%) from the ASD group and one parent from the control group fell within the moderate range (11–14), with anxiety scores for one parent from the ASD group, but no parents from the control group, falling within the severe range (15–21). Only two parents (5%) from the ASD group, but no parents from the control group, had depression scores within the moderate range, with no scores for either group falling within the severe range. High levels of parenting stress were reported by parents in the ASD group, with scores on all subscales of the PSI falling at or above the 85th percentile, indicating clinically significant levels of parenting stress (Abidin, 1995). Low levels of parenting stress

Table 15

Descriptive Information for Child Variables in the ASD (*n* = 38) *and Control* (*n* = 38) *Groups*

Variable	ASD Group		Control Group	
	M *(SD)*	Range	M *(SD)*	Range
GARS: Stereotyped behaviour	10.42 (2.55)	7 – 15	2.26 (1.63)	1 – 5
GARS: Communication	10.37 (3.39)	3 – 16	2.11 (1.82)	1 – 6
GARS: Social interaction	10.74 (1.70)	7 – 13	1.79 (1.27)	1 – 5
GARS: Autism Index	103.32 (10.52)	83 – 126	46.68 (12.41)	14 – 68
Vineland: Communication domain	64.37 (14.85)	40 – 91	119.89 (15.97)	89 – 149
Vineland: Daily living domain	65.32 (12.85)	43 – 93	113.37 (16.46)	95 – 155
Vineland: Socialisation domain	62.74 (12.48)	38 – 85	119.37 (18.94)	96 – 160
Vineland: Motor domain	73.82 (15.20)	56 – 102	107.09 (11.68)	84 – 124
Vineland: Composite score	63.11 (11.86)	38 – 88	119.84 (17.65)	100 – 160
BPI: SIB frequency	7.26 (5.34)	0 – 19	0.79 (1.93)	0 – 7
BPI: SIB severity	4.74 (3.38)	0 – 12	0.58 (1.30)	0 – 4
BPI: Stereotypy frequency	27.68 (19.40)	2 – 68	1.37 (2.83)	0 – 10
BPI: Stereotypy severity	16.05 (10.37)	2 – 42	0.84 (1.68)	0 – 5
BPI: Aggression frequency	5.74 (5.15)	0 – 18	1.11 (2.45)	0 – 9
BPI: Aggression severity	3.95 (3.67)	0 – 15	0.74 (1.48)	0 – 4
Conners: Oppositional	55.28 (9.52)	41 – 74	46.25 (7.03)	38 – 62
Conners: Inattention	66.11 (11.72)	45 – 86	43.94 (3.53)	41 – 54
Conners: Hyperactivity	72.50 (12.56)	53 – 90	45.69 (5.59)	42 – 64
Conners: ADHD index	69.67 (10.25)	50 – 84	43.31 (4.99)	40 – 59
CSHQ: Bedtime resistance	9.11 (3.40)	6 – 15	7.16 (2.52)	5 – 14
CSHQ: Sleep onset delay	2.05 (0.85)	1 – 3	1.05 (0.23)	1 – 2
CSHQ: Sleep duration	4.21 (1.62)	3 – 7	3.37 (0.90)	3 – 6
CSHQ: Parasomnias	9.79 (2.57)	7 – 17	8.32 (1.77)	7 – 13
CSHQ: Total	49.53 (11.01)	35 – 76	39.05 (6.47)	33 – 58
Unmet service needs	3.72 (1.49)	1 – 6	0	0

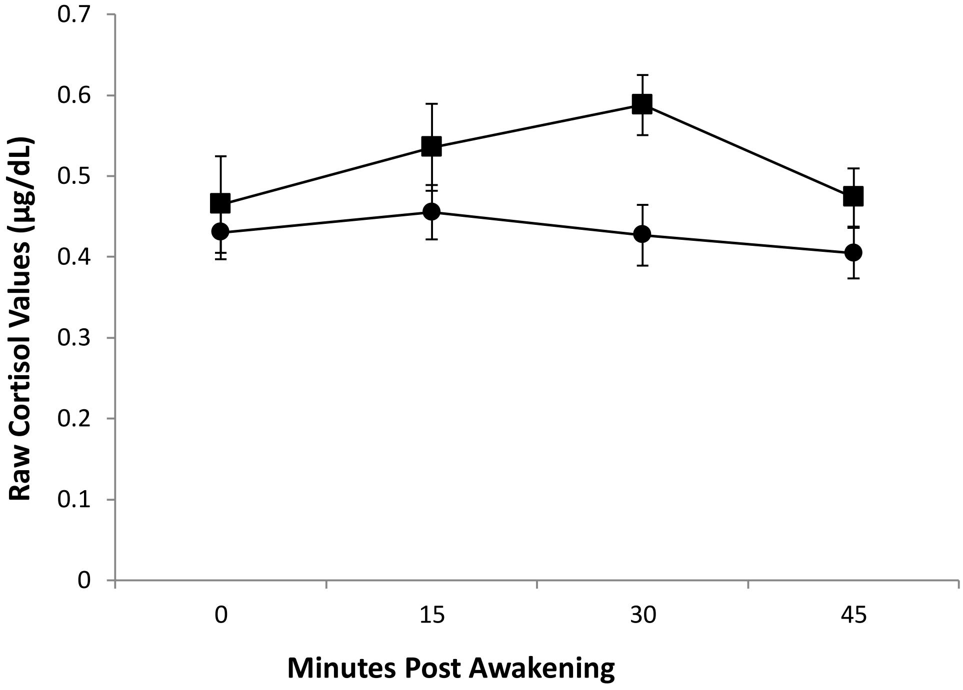

Figure 3. Post-awakening mean group cortisol values (µg/dL) for parents in ASD (circles) and control (boxes) groups, with standard error bars

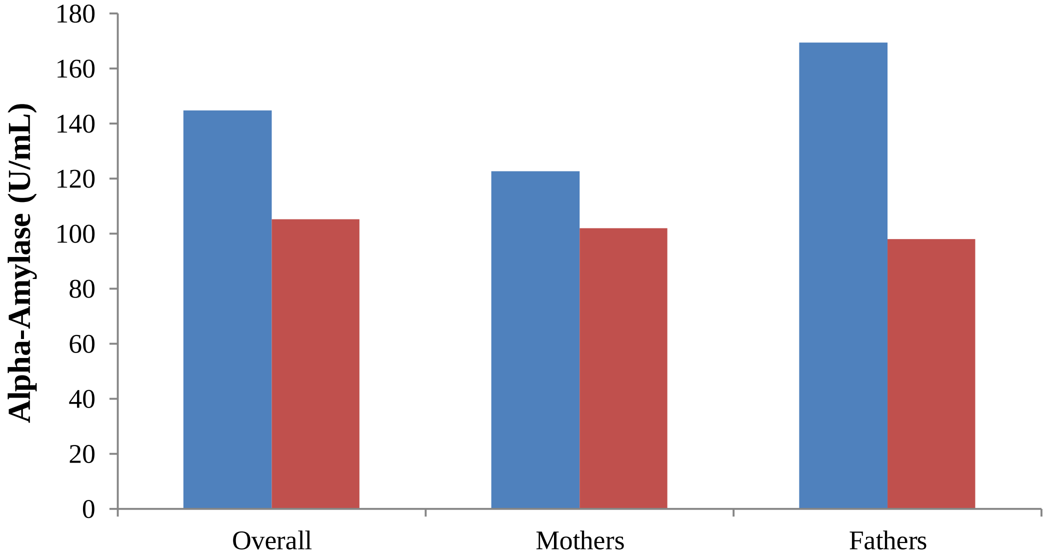

Figure 4. Mean group alpha-amylase values (U/mL) for parents in ASD (blue) and control (red) groups

were reported by parents in the control group, with mean group scores on all subscales of the PSI falling below the 35th percentile. Defensive responding, which is of concern when scores are below 10, did not appear to be an issue for either group.

Mean cortisol and sAA levels for both groups are illustrated in Figures 3 and 4, respectively. Mean group CV measures are presented in Tables 22 – 26. Mean 24-hour SBP and DBP for both groups fell within the normotensive range, defined as a 24-hour SBP $\leq$ 135 mmHg and a 24-hour DBP $\leq$ 85 mmHg (O'Brien et al., 2000).

Parenting Responsibility, Anxiety, Depression, and Parenting Stress

ANOVA results revealed a significant main effect for gender with respect to parenting responsibility, such that mothers reported higher levels of parenting responsibility than fathers (see Table 16). However, there was no significant main effect for group, and no significant interaction between group x gender. For anxiety, depression, and the parenting stress subscales, there were significant main effects for group, such that the ASD gropu reported significantly higher levels of anxiety, depression, overall parenting stress, parental distress, difficult child scores, and parent-child dysfunction interactions than the control group (see Table 16). No significant main effects for gender and no significant group x gender interaction effects were observed for anxiety, depression, or parenting stress.

Cortisol and Alpha-Amylase

There were no significant main effects for gender nor group, and no significant interaction effects for group x gender, for AUC_G, $AUC_{I,}$ or sAA, such that similar cortisol and sAA levels were observed across groups and gender (see Table 17). However, ANOVA analyses of individual cortisol measures revealed a significant main effect for group for the third cortisol measure. Parents in the ASD group had significantly lower cortisol levels on the third measure than parents in the control

Table 16

ANOVA/ANCOVA Results for Parenting Responsibility, Anxiety, Depression, and Parenting Stress Subscales

Variable		ASD Group		Control Group			ANOVA/ANCOVA Results		
	Gender	M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Parenting responsibility	Mothers	44.56 (7.23)	25 – 57	44.63 (4.43)	35 – 51	Group	0.43 (1, 70)	.516 ^{a}	.006
	Fathers	29.72 (6.75)	22 – 51	27.84 (5.06)	20 – 38	Gender	130.71 (1, 70)	.000 ^{a} *	.651
	Total	37.14 (10.20)	22 – 57	36.24 (9.71)	20 – 51	Group x Gender	0.50 (1, 70)	.482 ^{a}	.007
HADS: Anxiety ^{d}	Mothers	10.21 (3.28)	5 – 16	5.74 (3.05)	1 – 11	Group	32.51 (1, 69)	.000 ^{b} *	.320
	Fathers	6.37 (3.39)	0 – 14	3.63 (2. 34)	0 – 9	Gender	3.98 (1, 69)	.050 ^{b}	.055
	Total	8.29 (3.82)	0 – 16	4.68 (2.89)	0 – 11	Group x Gender	1.65 (1, 69)	.204 ^{b}	.023
HADS: Depression ^{d}	Mothers	10.21 (3.28)	2 – 11	2.68 (1.73)	0 – 6	Group	51.27 (1, 69)	.000 ^{b} *	.426
	Fathers	5.00 (3.18)	1 – 14	2.11 (1.94)	0 – 6	Gender	1.14 (1, 69)	.290 ^{b}	.016
	Total	6.13 (3.14)	1 – 14	2.39 (1.84)	0 – 6	Group x Gender	2.67 (1, 69)	.107 ^{b}	.037
PSI: Total	Mothers	107.84 (17.86)	85 – 146	58.79 (10.88)	39 – 74	Group	138.93 (1, 72)	.000 ^{c} *	.659
	Fathers	95.21 (19.19)	60 – 134	59.21 (13.56)	37 – 83	Gender	2.86 (1, 72)	.095 ^{c}	.038
	Total	101.53 (19.37)	60 – 146	59.00 (12.13)	37 – 83	Group x Gender	3.27 (1, 72)	.075 ^{c}	.043
PSI: Parent-child dysfunctional interactions	Mothers	31.26 (7.29)	20 – 47	15.47 (3.79)	12 – 24	Group	101.25 (1, 72)	.000 ^{c} *	.584
	Fathers	28.26 (6.50)	16 – 42	17.53 (4.72)	12 – 25	Gender	0.13 (1, 72)	.720 ^{c}	.002
	Total	29.76 (6.98)	16 – 47	16.50 (4.35)	12 – 25	Group x Gender	3.67 (1, 72)	.059 ^{c}	.049

Note. * Significant at Bonferroni-adjusted α value. ^{a} α value = .05. ^{b} Bonferroni-adjusted α value = .025. ^{c} Bonferroni-adjusted α value = .01. ^{d} ANCOVA; covariate = PRS score.

Table 16, Continued

ANOVA/ANCOVA Results for Parenting Responsibility, Anxiety, Depression, and Parenting Stress Subscales

Variable		ASD Group		Control Group			ANOVA/ANCOVA Results		
	Gender	M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	Ƞ^{2}
PSI: Difficult child	Mothers	42.42 (6.12)	30 – 52	21.79 (6.12)	13 – 32	Group	139.20 (1, 72)	.000 [a] *	.659
	Fathers	37.00 (8.94)	20 – 53	20.79 (5.51)	13 – 33	Gender	4.23 (1, 72)	.043 [a]	.055
	Total	39.71 (8.04)	20 – 53	21.29 (5.77)	13 – 33	Group x Gender	2.00 (1, 72)	.161 [a]	.027
PSI: Parental distress	Mothers	34.16 (8.60)	15 – 48	21.53 (5.94)	12 – 35	Group	44.65 (1, 72)	.000 [a] *	.383
	Fathers	29.95 (7.86)	17 – 43	20.89 (5.39)	12 – 30	Gender	2.23 (1, 72)	.140 [a]	.303
	Total	32.05 (8.40)	15 – 48	21.21 (5.60)	12 – 35	Group x Gender	1.22 (1, 72)	.274 [a]	.017
PSI: Defensive responding	Mothers	20.42 (5.30)	8 – 28	13.05 (3.47)	7 – 20	Group	38.94 (1, 68)	.000 [a] *	.364
	Fathers	18.00 (5.19)	10 – 27	12.11 (3.25)	7 – 20	Gender	3.14 (1, 68)	.081 [a]	.044
	Total	19.21 (5.31)	8 – 28	12.58 (3.35)	7 – 20	Group x Gender	0.60 (1, 68)	.441 [a]	.099

Note. * Significant at Bonferroni-adjusted α value. [a] Bonferroni-adjusted α value = .01.

Table 17

ANOVA/ANCOVA Results for Cortisol and Alpha-Amylase Measurements

Variable	Gender	ASD Group		Control Group			ANOVA/ANCOVA Results		
		M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
AUC_G	Mothers	-1.11 (0.50)	-1.71 – 0.04	-0.86 (0.46)	-1.58 – 0.06	Group	4.34 (1, 51)	.042 [a]	.078
	Fathers	-1.28 (0.43)	-2.06 – -0.74	-1.04 (0.44)	-1.63 – -0.45	Gender	4.31 (1, 51)	.043 [a]	.078
	Total	-1.19 (0.46)	-2.06 – 0.04	-0.93 (0.45)	-1.63 – 0.06	Group x Gender	0.74 (1, 51)	.394 [a]	.014
AUC_I	Mothers	0.11 (0.49)	-0.75 – 0.79	0.07 (0.46)	-0.44 – 1.26	Group	0.52 (1, 51)	.474 [a]	.010
	Fathers	0.12 (0.74)	-0.64 – 1.36	0.36 (0.44)	-0.29 – 1.09	Gender	0.21 (1, 51)	.650 [a]	.004
	Total	0.11 (0.61)	-0.75 – 1.36	0.20 (0.47)	-0.44 – 1.26	Group x Gender	1.60 (1, 51)	.211 [a]	.030
sAA [c]	Mothers	122.69 (82.33)	33.70 – 307.70	102.11 (75.78)	23.30 – 243.80	Group	0.27 (1, 38)	.608 [b]	.007
	Fathers	169.46 (137.76)	8.20 – 390.20	98.10 (93.66)	16.10 – 261.80	Gender	0.00 (1, 38)	.962 [b]	.000
	Total	144.96 (111.81)	8.20 – 390.20	100.20 (82.60)	16.10 – 261.80	Group x Gender	0.56 (1, 38)	.457 [b]	.015

Note. [a] Bonferroni-adjusted α value = .025. [b] α value = .05. [c] ANCOVA; covariate = participant age.

Table 18

ANOVA Results for Individual Cortisol Measurements

Variable		ASD Group		Control Group			ANOVA Results		
	Gender	M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Cortisol 1	Mothers	0.50 (0.49)	0.27 – 2.09	0.53 (0.19)	0.25 – 0.86	Group	1.83 (1, 48)	.183 [a]	.037
Immediately after waking	Fathers	0.36 (0.18)	0.04 – 0.81	0.37 (0.17)	0.21 – 0.71	Gender	5.14 (1, 48)	.028 [a]	.097
	Total	0.43 (0.36)	0.04 – 2.09	0.47 (0.20)	0.21 – 0.86	Group x Gender	0.20 (1, 48)	.656 [a]	.004
Cortisol 2	Mothers	0.52 (0.43)	0.25 – 2.00	0.53 (0.22)	0.19 – 1.13	Group	3.96 (1, 56)	.051 [a]	.066
15 minutes after waking	Fathers	0.39 (0.17)	0.06 – 0.62	0.54 (0.21)	0.26 – 0.90	Gender	0.63 (1, 56)	.430 [a]	.011
	Total	0.46 (0.33)	0.06 – 2.00	0.53 (0.21)	0.19 – 1.13	Group x Gender	1.27 (1, 56)	.264 [a]	.022
Cortisol 3	Mothers	0.45 (0.21)	0.23 – 0.83	0.63 (0.25)	0.31 – 1.17	Group	11.66 (1, 56)	.001 [a] *	.172
30 minutes after waking	Fathers	0.40 (0.22)	0.13 – 0.87	0.57 (0.22)	0.25 – 1.02	Gender	1.63 (1, 56)	.207 [a]	.028
	Total	0.42 (0.21)	0.13 – 0.87	0.60 (0.23)	0.25 – 1.17	Group x Gender	0.07 (1, 56)	.796 [a]	.001
Cortisol 4	Mothers	0.40 (0.22)	0.17 – 0.86	0.52 (0.24)	0.13 – 1.06	Group	3.43 (1, 55)	.069 [a]	.059
45 minutes after waking	Fathers	0.39 (0.17)	0.18 – 0.72	0.44 (0.13)	0.20 – 0.66	Gender	0.13 (1, 55)	.717 [a]	.002
	Total	0.40 (19)	0.17 – 0.86	0.48 (0.20)	0.13 – 1.06	Group x Gender	0.15 (1, 55)	.703 [a]	.003

Note. * Significant at Bonferroni-adjusted α value. [a] Bonferroni-adjusted α value = .013.

group (see Table 18). No other significant effects were observed for the individual measures.

Cortisol values were further analysed to determine if the groups differed in the number of cortisol responders and non-responders. Responders were defined as participants who had an increase of salivary cortisol levels of at least 2.4 nmol/L above individual baseline (Wust et al., 2000). Non-responding was observed in 63% of parents in the ASD group, and 54% of parents in the control group. Chi-square analysis revealed that there was not a significant association between parenting group and cortisol responding, χ^2 (1, N = 51) = 0.41, p = .52.

CV Measurements

Significant main effects for gender were found for mean awake and 24-hour SBP and DBP, such that fathers were found to have higher SBP and DBP than mothers (see Table 19 and Table 20). Conversely, a significant main effect for gender was found for 24-hour HR, such that mothers were found to have significantly higher HR than fathers (see Table 21). No significant main effects for group, and no significant interaction effects for group x gender, were observed for these measures. Similarly, no significant main effects for group nor gender, and no significant interaction effects for group x gender, were observed for the other mean CV measures (see Table 22 and Table 23). Chi-square analyses were conducted to investigate associations between group and type of SBP and DBP dipping. There was no significant association between group and dipping status for SBP, χ^2 (2, N = 70) = 0.23, p = .89, or DBP, χ^2 (2, N = 68) = 1.42, p = .49.

As in Study 2, standard deviations from awake and asleep SBP, DBP, and HR measures were used as estimates of BP and HR variability (Kikuya et al., 2000). There were significant main effects for gender for awake SBP and awake HR

Table 19

ANOVA/ANCOVA Results for Mean Systolic Blood Pressure Measurements

Variable		ASD Group		Control Group			ANOVA/ANCOVA Results		
	Gender	M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Mean awake SBP	Mothers	124.74 (11.56)	103 – 152	131.47 (15.19)	110 – 165	Group	0.02 (1, 76)	.888 [a]	.000
	Fathers	141.68 (15.37)	119 – 175	136.42 (11.95)	116 – 162	Gender	13.80 (1, 76)	.000 [a] *	.154
	Total	134.10 (12.92)	114 – 164	133.58 (14.01)	110 – 165	Group x Gender	3.78 (1, 76)	.055 [a]	.047
Mean asleep SBP [b]	Mothers	106.22 (12.37)	87 – 128	114.56 (16.67)	92 – 156	Group	0.06 (1, 69)	.814 [a]	.001
	Fathers	122.56 (14.96)	97 – 150	114.06 (17.42)	91 – 170	Gender	5.55 (1, 69)	.021 [a]	.074
	Total	116.11 (13.43)	92 – 144	112.13 (14.26)	91 – 156	Group x Gender	4.14 (1, 69)	.046 [a]	.057
Mean 24-hour SBP	Mothers	120.78 (11.45)	99 – 146	129.06 (15.01)	107 – 162	Group	0.10 (1, 72)	.752 [a]	.001
	Fathers	137.78 (15.57)	118 – 168	132.28 (12.18)	112 – 152	Gender	11.44 (1, 72)	.001 [a] *	.137
	Total	129.28 (15.99)	99 – 168	130.67 (13.57)	107 – 162	Group x Gender	4.62 (1, 72)	.035 [a]	.060
Mean SBP dip	Mothers	14.71 (5.93)	0 – 24	13.55 (6.87)	2 – 29	Group	0.39 (1, 72)	.537 [a]	.005
	Fathers	13.40 (7.35)	1 – 24	16.66 (11.45)	-16 – 32	Gender	0.12 (1, 72)	.727 [a]	.002
	Total	14.05 (6.61)	0 – 24	15.10 (9.44)	-16 – 32	Group x Gender	1.19 (1, 72)	.278 [a]	.016

Note. * Significant at Bonferroni-adjusted α value. [a] Bonferroni-adjusted α value = .013. [b] ANCOVA; covariate = exercise duration.

Table 20

ANOVA/ANCOVA Results for Mean Diastolic Blood Pressure Measurements

Variable		ASD Group		Control Group			ANOVA/ANCOVA Results		
	Gender	M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Mean awake DBP [b]	Mothers	74.95 (8.65)	53 – 86	76.47 (9.78)	59 – 97	Group	0.11 (1, 62)	.742 [a]	.002
	Fathers	85.11 (10.95)	71 – 111	81.26 (7.59)	71 – 95	Gender	10.45 (1, 62)	.002 [a] *	.144
	Total	80.03 (11.01)	53 – 111	78.87 (8.97)	59 – 97	Group x Gender	1.53 (1, 62)	.221 [a]	.024
Mean asleep DBP [c]	Mothers	59.44 (7.91)	47 – 75	59.72 (8.01)	48 – 77	Group	0.33 (1, 55)	.567 [a]	.006
	Fathers	69.11 (12.03)	55 – 92	61.22 (8.84)	50 – 79	Gender	4.54 (1, 55)	.038 [a]	.076
	Total	64.28 (11.17)	47 – 92	60.47 (8.34)	48 – 79	Group x Gender	1.55 (1, 55)	.218 [a]	.027
Mean 24-hour DBP [d]	Mothers	71.39 (8.24)	51 – 81	73.78 (8.35)	57 – 88	Group	0.02 (1, 58)	.885 [a]	.000
	Fathers	82.17 (11.49)	68 – 108	77.17 (7.65)	63 – 91	Gender	9.19 (1, 58)	.004 [a] *	.137
	Total	76.78 (11.27)	51 – 108	75.47 (8.08)	57 – 91	Group x Gender	2.89 (1, 58)	.095 [a]	.047
Mean DBP dip	Mothers	20.02 (6.38)	6 – 27	22.26 (7.91)	9 – 37	Group	3.78 (1, 72)	.056 [a]	.050
	Fathers	19.02 (9.08)	3 – 34	24.19 (9.95)	3 – 41	Gender	0.00 (1, 72)	.966 [a]	.000
	Total	19.52 (7.75)	3 – 34	23.23 (8.91)	3 – 41	Group x Gender	0.52 (1, 72)	.473 [a]	.007

Note. * Significant at Bonferroni-adjusted α value. [a] Bonferroni-adjusted α value = .013. [b] ANCOVA; covariate = BMI. [c] ANCOVA; covariates = BMI and exercise duration. [d] ANCOVA; covariates = BMI and participant age.

Table 21

ANOVA/ANCOVA Results for Mean Heart Rate Measurements

Variable		ASD Group		Control Group			ANOVA/ANCOVA Results		
	Gender	M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Mean awake HR	Mothers	78.16 (9.18)	62 – 97	72.26 (6.24)	63 – 84	Group	3.30 (1, 76)	.073 [a]	.042
	Fathers	73.47 (8.07)	62 – 90	72.37 (6.79)	56 – 83	Gender	1.07 (1, 76)	.305 [a]	.014
	Total	75.82 (8.85)	62 – 97	72.32 (6.43)	56 – 84	Group x Gender	1.29 (1, 76)	.259 [a]	.017
Mean asleep HR	Mothers	68.83 (9.13)	54 – 84	62.50 (8.11)	53 – 87	Group	1.84 (1, 72)	.179 [a]	.025
	Fathers	62.00 (7.92)	51 – 75	61.89 (7.65)	52 – 79	Gender	2.78 (1, 72)	.100 [a]	.037
	Total	65.42 (9.11)	51 – 84	62.19 (7.78)	52 – 87	Group x Gender	2.13 (1, 72)	.149 [a]	.029
Mean 24-hour HR [b]	Mothers	76.50 (8.81)	62 – 94	69.94 (5.99)	61 – 84	Group	0.50 (1, 59)	.485 [a]	.008
	Fathers	71.56 (7.74)	60 – 86	70.89 (5.80)	61 – 82	Gender	8.45 (1, 59)	.005 [a] *	.125
	Total	74.03 (8.55)	60 – 94	70.42 (5.83)	61 – 84	Group x Gender	1.96 (1, 59)	.166 [a]	.032

Note. * Significant at Bonferroni-adjusted α value. [a] Bonferroni-adjusted α value = .017. [b] ANCOVA; covariate = BMI.

Table 22

ANOVA/ANCOVA Results for Mean Arterial Pressure Measurements

Variable	Gender	ASD Group		Control Group		ANOVA/ANCOVA Results			
		M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Mean awake MAP [b]	Mothers	91.58 (9.27)	70 – 108	94.79 (11.09)	76 – 119	Group	0.68 (1, 61)	.411 [a]	.011
	Fathers	103.95 (11.90)	87 – 128	99.68 (8.59)	86 – 115	Gender	4.95 (1, 61)	.030 [a]	.075
	Total	97.76 (12.24)	70 – 128	97.24 (10.10)	76 – 119	Group x Gender	1.89 (1, 61)	.175 [a]	.030
Mean asleep MAP [c]	Mothers	75.06 (9.16)	61 – 93	78.06 (10.15)	64 – 98	Group	0.05 (1, 55)	.819 [a]	.001
	Fathers	86.78 (12.65)	69 – 111	78.72 (11.07)	64 – 109	Gender	5.38 (1, 55)	.024 [a]	.089
	Total	80.92 (12.40)	61 – 111	78.39 (10.47)	64 – 109	Group x Gender	2.08 (1,55)	.155 [a]	.036
Mean 24-hour MAP [b]	Mothers	87.83 (9.12)	67 – 102	92.00 (10.01)	74 – 111	Group	0.22 (1, 57)	.642 [a]	.004
	Fathers	100.67 (12.36)	86 – 125	95.44 (8.92)	80 – 110	Gender	4.61 (1, 57)	.036 [a]	.075
	Total	94.25 (12.53)	67 – 125	93.72 (9.51)	74 – 111	Group x Gender	3.00 (1, 57)	.089 [a]	.050

Note. [a] Bonferroni-adjusted α value = .017. [b] ANCOVA; covariates = BMI and parenting responsibility. [c] ANCOVA; covariates = BMI, participant age, and exercise duration.

Table 23

ANOVA Results for Mean Pulse Pressure Measurements

Variable		ASD Group		Control Group			ANOVA Results		
	Gender	M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Mean awake PP	Mothers	49.74 (5.69)	42 – 66	54.95 (8.59)	44 – 72	Group	0.90 (1, 76)	.345 [a]	.012
	Fathers	56.58 (8.40)	47 – 79	55.11 (6.90)	42 – 70	Gender	5.46 (1, 76)	.022 [a]	.067
	Total	53.16 (7.88)	42 – 79	55.03 (7.69)	42 – 72	Group x Gender	3.55 (1, 76)	.063 [a]	.045
Mean asleep PP	Mothers	46.67 (7.01)	37 – 65	54.72 (11.69)	38 – 89	Group	2.51 (1, 72)	.117 [a]	.034
	Fathers	53.33 (7.51)	42 – 70	52.83 (11.38)	40 – 91	Gender	1.32 (1, 72)	.254 [a]	.018
	Total	50.00 (7.92)	37 – 70	53.78 (11.41)	38 – 91	Group x Gender	3.34 (1, 72)	.072 [a]	.044
Mean 24-hour PP	Mothers	49.33 (5.53)	42 – 65	55.33 (9.41)	43 – 76	Group	1.80 (1, 72)	.184 [a]	.024
	Fathers	55.89 (8.17)	46 – 78	55.17 (6.70)	44 – 69	Gender	4.18 (1, 72)	.045 [a]	.055
	Total	52.61 (7.64)	42 – 78	55.25 (8.05)	43 – 76	Group x Gender	3.24 (1, 72)	.076 [a]	.043

Note. [a] Bonferroni-adjusted α value = .017.

Table 24

ANOVA Results for Cardiovascular Variability Measurements

Variable		ASD Group		Control Group			ANOVA Results		
	Gender	M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Awake SBP variability	Mothers	23.62 (6.46)	17 – 41	26.78 (6.24)	18 – 44	Group	0.20 (1, 71)	.656 [a]	.003
	Fathers	29.19 (5.46)	21 – 44	27.18 (5.15)	20 – 38	Gender	5.37 (1, 71)	.023 [a] *	.070
	Total	26.40 (6.08)	17 – 44	26.99 (5.63)	18 – 44	Group x Gender	4.01 (1, 71)	.049 [a]	.053
Asleep SBP variability	Mothers	22.48 (4.36)	15 – 30	27.51 (6.91)	20 – 48	Group	0.30 (1, 68)	.587 [a]	.004
	Fathers	29.13 (8.89)	14 – 48	25.88 (6.59)	16 – 45	Gender	2.41 (1, 68)	.126 [a]	.034
	Total	25.81 (7.68)	14 – 48	26.69 (6.71)	16 – 48	Group x Gender	6.52 (1, 68)	.013 [a] *	.087
Awake DBP variability	Mothers	22.88 (4.06)	17 – 33	26.72 (7.27)	17 – 48	Group	1.76 (1, 72)	.189 [a]	.024
	Fathers	27.95 (5.07)	19 – 39	27.35 (5.13)	20 – 37	Gender	4.91 (1, 72)	.030 [a]	.064
	Total	25.36 (5.18)	17 – 39	27.04 (6.21)	17 – 48	Group x Gender	2.95 (1, 72)	.090 [a]	.039
Asleep DBP variability	Mothers	19.09 (4.24)	10 – 29	23.17 (7.03)	12 – 39	Group	0.37 (1, 68)	.547 [a]	.005
	Fathers	23.84 (6.15)	7 – 36	21.37 (4.69)	13 – 33	Gender	1.24 (1, 68)	.270 [a]	.018
	Total	21.47 (5.74)	7 – 36	22.27 (5.96)	12 – 39	Group x Gender	6.08 (1, 68)	.016 [a] *	.082

Note. * Significant at Bonferroni-adjusted α value. [a] Bonferroni-adjusted α value = .025.

Table 24, Continued

ANOVA Results for CV Variability Measurements

Variable	Gender	ASD Group		Control Group		ANOVA Results			
		M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Awake HR variability	Mothers	22.98 (3.71)	17 – 32	25.62 (6.45)	18 – 41	Group	0.58 (1, 72)	.451 [a]	.008
	Fathers	28.74 (6.17)	21 – 44	28.10 (6.34)	19 – 44	Gender	9.67 (1, 72)	.003 [a] *	.118
	Total	25.86 (5.81)	17 – 44	26.86 (6.43)	18 – 44	Group x Gender	1.53 (1, 72)	.220 [a]	.021
Asleep HR variability	Mothers	19.28 (4.18)	10 – 27	22.90 (7.86)	12 – 42	Group	0.89 (1, 68)	.350 [a]	.013
	Fathers	24.84 (6.78)	12 – 40	24.35 (8.62)	12 – 42	Gender	4.45 (1, 68)	.039 [a]	.061
	Total	22.06 (6.23)	10 – 40	23.63 (8.16)	12 – 42	Group x Gender	1.52 (1, 68)	.222 [a]	.022

Note. * Significant at Bonferroni-adjusted α value. [a] Bonferroni-adjusted α value = .025.

variability, such that fathers had higher variability than mothers (see Table 24). For asleep SBP and DBP variability, there were significant group x gender interaction effects, such that fathers of children with ASD had the highest variability. There were no other significant effects for SBP, DBP, or HR variability.

In order to control for the use of antihypertensive medication, all analyses including CV measures were repeated, excluding any participant taking antihypertensive medication. The outcomes of the analyses did not differ when these participants were excluded, so the participants were retained in the CV analyses.

Supports and Coping Strategies

For instrumental support, planning, acceptance, and self-blame, there were significant main effects for group, such that parents in the ASD group reported higher use of those coping strategies than the control group (see Table 25). There were no significant main effects for gender, and no significant interaction effects for group x gender, for these coping strategies. For unmet supports, social support quality, and the remaining coping strategies, there were no significant main effects for group nor gender, and no significant interaction effects for group x gender. However, there was a significant main effect for gender for social support quantity, such that fathers reported a significantly lower number of social supports than mothers. There was not a significant main effect for group for social support quantity; however, it was approaching significance, with parents in the ASD group reporting a lower quantity of social supports than parents in the control group. There was no significant group x gender interaction effect for social support quantity.

Sleep

For PSQI global, subjective sleep quality, sleep duration, and sleep disturbances, there were significant main effects for group, such that the ASD group reported

Table 25

ANOVA Results for Unmet Supports, Social Support, and Coping Strategies

Variable		ASD Group		Control Group		ANOVA Results			
	Gender	M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Unmet supports	Mothers	3.46 (1.45)	2 – 7	3.00 (1.00)	2 – 4	Group	1.48 (1, 31)	.232^{a}	.046
	Fathers	3.31 (1.25)	2 – 6	250 (1.05)	1 – 4	Gender	0.39 (1, 31)	.535^{a}	.013
	Total	3.39 (1.33)	2 – 7	2.67 (1.00)	1 – 4	Group x Gender	0.11 (1, 31)	.742^{a}	.004
SSQ: Social support quantity	Mothers	16.47 (9.53)	6 – 36	21.94 (9.45)	5 – 42	Group	5.08 (1, 66)	.028^{b}	.071
	Fathers	11.53 (6.76)	0 – 28	15.00 (6.54)	4 – 25	Gender	8.99 (1, 66)	.004^{b}*	.120
	Total	14.14 (8.59)	0 – 36	18.68 (8.82)	4 – 42	Group x Gender	0.25 (1, 66)	.616^{b}	.004
SSQ: Social support quality	Mothers	29.58 (4.46)	19 – 36	32.67 (3.53)	24 – 36	Group	0.32 (1, 66)	.571^{b}	.005
	Fathers	32.59 (3.10)	27 – 36	30.81 (7.31)	6 – 36	Gender	0.25 (1, 66)	.618^{b}	.004
	Total	31.00 (4.12)	19 – 36	31.79 (5.62)	6 – 36	Group x Gender	4.45 (1, 66)	.039^{b}	.063
COPE: Active coping	Mothers	5.84 (1.34)	4 – 8	4.68 (2.00)	2 – 8	Group	4.63 (1, 71)	.035^{c}	.061
	Fathers	4.95 (1.75)	2 – 8	4.22 (2.37)	2 – 8	Gender	2.40 (1, 71)	.125^{c}	.033
	Total	5.40 (1.60)	2 – 8	4.46 (2.17)	2 – 8	Group x Gender	0.25 (1, 71)	.622^{c}	.003
COPE: Denial	Mothers	2.79 (1.55)	2 – 8	2.26 (0.81)	2 – 5	Group	2.86 (1, 71)	.095^{c}	.039
	Fathers	2.32 (0.95)	2 – 6	2.06 (0.24)	2 – 3	Gender	0.33 (1, 71)	.147^{c}	.029
	Total	2.55 (1.29)	2 – 8	2.16 (0.60)	2 – 5	Group x Gender	0.33 (1, 71)	.569^{c}	.005

Note. * Significant at Bonferroni-adjusted α value. ^{a} α value = .05. ^{b} Bonferroni-adjusted α value = .025. ^{c} Bonferroni-adjusted α value = .004.

Table 25, Continued

ANOVA Results for Unmet Supports, Social Support, and Coping Strategies

Variable		ASD Group		Control Group			ANOVA Results		
	Gender	M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
COPE: Substance use	Mothers	2.68 (1.49)	2 – 8	2.00 (0.00)	2 – 2	Group	5.02 (1, 71)	.028 ^{a}	.066
	Fathers	2.32 (1.00)	2 – 6	2.06 (0.24)	2 – 3	Gender	0.55 (1, 71)	.461 ^{a}	.008
	Total	2.50 (1.27)	2 – 8	2.03 (0.16)	2 – 3	Group x Gender	1.01 (1, 71)	.318 ^{a}	.014
COPE: Emotional support	Mothers	4.84 (1.46)	2 – 8	4.16 (1.64)	2 – 7	Group	4.33 (1, 71)	.041 ^{a}	.057
	Fathers	4.05 (1.35)	2 – 7	3.28 (1.60)	2 – 8	Gender	5.67 (1, 71)	.020 ^{a}	.074
	Total	4.45 (1.45)	2 – 8	3.73 (1.66)	2 – 8	Group x Gender	0.02 (1, 71)	.898 ^{a}	.000
COPE: Instrumental support	Mothers	5.42 (1.68)	2 – 8	3.84 (1.50)	2 – 7	Group	15.58 (1, 71)	.000 ^{a} *	.180
	Fathers	4.26 (1.56)	2 – 7	3.06 (1.35)	2 – 6	Gender	7.59 (1, 71)	.007 ^{a}	.097
	Total	4.84 (1.70)	2 – 8	3.46 (1.46)	2 – 7	Group x Gender	0.28 (1, 71)	.601 ^{a}	.004
COPE: Behavioural disengagement	Mothers	2.42 (1.30)	2 – 7	2.16 (0.50)	2 – 4	Group	1.39 (1, 71)	.242 ^{a}	.019
	Fathers	2.53 (1.22)	2 – 7	2.22 (0.94)	2 – 6	Gender	0.12 (1, 71)	.726 ^{a}	.002
	Total	2.47 (1.25)	2 – 7	2.19 (0.74)	2 – 6	Group x Gender	0.01 (1, 71)	.932 ^{a}	.000
COPE: Planning	Mothers	6.26 (1.70)	2 – 8	4.53 (1.87)	2 – 8	Group	22.35 (1, 71)	.000 ^{a} *	.239
	Fathers	5.26 (1.52)	2 – 8	3.33 (1.61)	2 – 8	Gender	7.99 (1, 71)	.006 ^{a}	.101
	Total	5.76 (1.67)	2 – 8	3.95 (1.83)	2 – 8	Group x Gender	0.06 (1, 71)	.804 ^{a}	.001

Note. * Significant at Bonferroni-adjusted α value. ^{a} Bonferroni-adjusted α value = .004.

Table 25, Continued

ANOVA Results for Unmet Supports, Social Support, and Coping Strategies

Variable	Gender	ASD Group		Control Group		ANOVA Results			
		M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
COPE: Humour	Mothers	3.79 (1.32)	2 – 6	2.79 (0.98)	2 – 4	Group	7.14 (1, 71)	.009 [a]	.091
	Fathers	3.42 (1.64)	2 – 8	2.83 (1.10)	2 – 5	Gender	0.30 (1, 71)	.587 [a]	.004
	Total	3.61 (1.48)	2 – 8	2.81 (1.02)	2 – 5	Group x Gender	0.48 (1, 71)	.490 [a]	.007
COPE: Acceptance	Mothers	6.42 (1.71)	2 – 8	4.05 (1.93)	2 – 7	Group	30.49 (1, 71)	.000 [a] *	.300
	Fathers	6.16 (1.50)	3 – 8	3.94 (2.01)	2 – 8	Gender	0.20 (1, 71)	.656 [a]	.003
	Total	6.29 (1.59)	2 – 8	4.00 (1.94)	2 – 8	Group x Gender	0.04 (1, 71)	.852 [a]	.000
COPE: Religion	Mothers	3.79 (2.07)	2 – 8	2.95 (1.31)	2 – 6	Group	5.12 (1, 71)	.027 [a]	.067
	Fathers	3.32 (1.97)	2 – 8	2.44 (0.86)	2 – 5	Gender	1.66 (1, 71)	.201 [a]	.023
	Total	3.55 (2.01)	2 – 8	2.70 (1.13)	2 – 6	Group x Gender	0.00 (1, 71)	.969 [a]	.000
COPE: Self-blame	Mothers	4.37 (1.98)	2 – 8	3.16 (1.21)	2 – 6	Group	12.82 (1, 71)	.001 [a] *	.153
	Fathers	3.47 (1.22)	2 – 6	2.44 (0.62)	2 – 4	Gender	6.61 (1, 71)	.012 [a]	.085
	Total	3.92 (1.68)	2 – 8	2.81 (1.02)	2 – 6	Group x Gender	0.08 (1, 71)	.773 [a]	.001

Note. * Significant at Bonferroni-adjusted α value. [a] Bonferroni-adjusted α value = .004.

Table 26

ANOVA Results for Parental Sleep Dysfunction

Variable	ASD Group		Control Group			ANOVA Results		
	M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
PSQI: Global	6.84 (3.17)	2 – 12	4.32 (2.60)	0 – 9	Group	12.28 (1, 71)	.001 [a] *	.147
	6.16 (2.83)	1 – 12	3.89 (3.22)	1 – 12	Gender	0.66 (1, 71)	.420 [a]	.009
	6.50 (2.98)	1 – 12	4.10 (2.89)	0 – 12	Group x Gender	0.04 (1, 71)	.851 [a]	.000
PSQI: Subjective sleep quality	1.16 (0.50)	0 – 2	0.58 (0.61)	0 – 2	Group	11.90 (1, 71)	.001 [a] *	.114
	1.11 (0.66)	0 – 2	0.67 (0.77)	0 – 2	Gender	0.01 (1, 71)	.906 [a]	.000
	1.13 (0.58)	0 – 2	0.62 (0.68)	0 – 2	Group x Gender	0.23 (1, 71)	.636 [a]	.003
PSQI: Sleep latency	1.26 (1.10)	0 – 3	0.84 (0.96)	0 – 3	Group	3.88 (1, 71)	.053 [a]	.052
	1.21 (1.13)	0 – 3	0.72 (0.75)	0 – 2	Gender	0.14 (1, 71)	.710 [a]	.002
	1.24 (1.10)	0 – 3	0.78 (0.85)	0 – 3	Group x Gender	0.02 (1, 71)	.885 [a]	.000
PSQI: Sleep duration	0.95 (0.71)	0 – 2	0.58 (0.61)	0 – 2	Group	8.68 (1, 71)	.004 [a] *	.109
	1.32 (0.82)	0 – 3	0.67 (0.84)	0 – 3	Gender	1.74 (1, 71)	.191 [a]	.024
	1.13 (0.78)	0 – 3	0.62 (0.72)	0 – 3	Group x Gender	0.66 (1, 71)	.419 [a]	.009

Note. * Significant at Bonferroni-adjusted α value. [a] Bonferroni-adjusted α value = .007.

Table 26, Continued

ANOVA Results for Parental Sleep Dysfunction

Variable	Gender	ASD Group		Control Group			ANOVA Results		
		M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
PSQI: Habitual sleep efficiency	Mothers	0.47 (0.84)	0 – 2	0.63 (1.01)	0 – 3	Group	0.24 (1, 71)	.625 [a]	.003
	Fathers	0.37 (0.50)	0 – 1	0.39 (0.70)	0 – 2	Gender	0.92 (1, 71)	.341 [a]	.013
	Total	0.42 (0.68)	0 – 2	0.51 (0.87)	0 – 3	Group x Gender	0.14 (1, 71)	.706 [a]	.002
PSQI: Sleep disturbances	Mothers	1.53 (0.61)	1 – 3	1.05 (0.52)	0 – 2	Group	8.23 (1, 71)	.005 [a] *	.104
	Fathers	1.11 (0.46)	0 – 2	0.89 (0.47)	0 – 2	Gender	5.91 (1, 71)	.018 [a]	.077
	Total	1.32 (0.57)	0 – 3	0.97 (0.50)	0 – 2	Group x Gender	1.14 (1, 71)	.288 [a]	.016
PSQI: Sleeping medication	Mothers	0.26 (0.81)	0 – 3	0.11 (0.46)	0 – 2	Group	0.03 (1, 71)	.873 [a]	.000
	Fathers	0.05 (0.23)	0 – 1	0.17 (0.71)	0 – 3	Gender	0.30 (1, 71)	.588 [a]	.004
	Total	0.16 (0.59)	0 – 3	0.14 (0.59)	0 – 3	Group x Gender	0.99 (1, 71)	.324 [a]	.014

Note. * Significant at Bonferroni-adjusted α value. [a] Bonferroni-adjusted α value = .007.

higher levels of sleep dysfunction than the control group across all these subdomains of the PSQI (see Table 26). There were no significant main effects for gender and no significant interaction effects for group x gender for these subdomains. Furthermore, there were no significant main effects for group nor gender, and no significant interaction effects for group x gender for sleep latency, habitual sleep efficiency, or needing to use sleeping medication.

Demographic and Health-Relevant Variables

Significant main effects for group and gender were observed for quantity of illnesses, such that mothers reported a higher number of illnesses than fathers, and participants in the ASD group reported a higher number of illnesses than participants in the control group (see Table 27). A significant interaction effect for group x gender was also observed, with mothers of children with ASD reporting the highest number of illnesses. With respect to alcohol consumption, a significant main effect was found for gender, such that fathers reported a higher level of alcohol consumption than mothers. There was no significant main effect for group, and no significant group x gender interaction effect, for alcohol consumption. No significant main effects for group nor gender, and no significant interaction effects for group x gender, were observed for quantity of cigarettes smoked, quantity of caffeine consumption, or exercise duration.

Chi-square analysis revealed a significant association between group and employment status, χ^2 (1, *N* = 72) = 6.18, *p* = .01, with significantly more participants in the ASD group (*n* = 15) than in the control group (*n* = 6) unemployed. With respect to medication usage, chi-square analysis revealed that there was no association between group and use of antidepressants, χ^2 (1, *N* = 76) = 0.56, *p* = .46. Chi-square analysis could not be conducted for use of antihypertensive medication,

Table 27

ANOVA Results for Health-Relevant Variables

Variable		ASD Group		Control Group			ANOVA Results		
	Gender	M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Quantity of illnesses	Mothers	1.47 (1.61)	0 – 6	0.11 (0.32)	0 – 1	Group	17.17 (1, 72)	.000 [a] *	.193
	Fathers	0.42 (0.77)	0 – 3	0.05 (0.23)	0 – 1	Gender	6.95 (1, 72)	.010 [a] *	.088
	Total	0.95 (1.35)	0 – 6	0.08 (0.27)	0 – 1	Group x Gender	5.69 (1, 72)	.020 [a] *	.073
Quantity of cigarettes smoked (weekly)	Mothers	0.00 (0.00)	0 – 0	14.84 (36.51)	0 – 140	Group	2.09 (1, 72)	.153 [b]	.028
	Fathers	14.74 (32.55)	0 – 100	21.32 (42.16)	0 – 140	Gender	2.05 (1, 72)	.157 [b]	.028
	Total	7.37 (23.90)	0 – 100	18.08 (39.04)	0 – 140	Group x Gender	0.31 (1, 72)	.579 [b]	.004
Quantity of alcohol consumed (weekly)	Mothers	2.71 (1.51)	0 – 7	2.62 (1.63)	0 – 5	Group	0.79 (1, 67)	.378 [b]	.012
	Fathers	4.15 (2.87)	0 – 10	5.28 (3.35)	0 – 12	Gender	12.25 (1, 67)	.001 [b] *	.155
	Total	3.39 (2.34)	0 – 10	3.99 (2.95)	0 – 12	Group x Gender	1.09 (1, 67)	.300 [b]	.016
Quantity of caffeine consumed (daily)	Mothers	3.34 (2.09)	0 – 8	4.72 (5.33)	0 – 20	Group	0.02 (1, 71)	.879 [b]	.000
	Fathers	3.76 (3.40)	0 – 14	2.63 (2.48)	0 – 8	Gender	1.05 (1, 71)	.308 [b]	.015
	Total	3.55 (2.79)	0 – 14	3.65 (4.20)	0 – 20	Group x Gender	2.38 (1, 71)	.127 [b]	.032
Exercise duration (daily, in minutes)	Mothers	26.84 (29.82)	0 – 100	23.33 (44.56)	0 – 180	Group	1.80 (1, 69)	.184 [a]	.025
	Fathers	33.95 (43.48)	0 – 120	14.41 (23.44)	0 – 60	Gender	0.01 (1, 69)	.916 [a]	.000
	Total	30.39 (36.95)	0 – 120	19.00 (35.66)	0 – 180	Group x Gender	0.87 (1, 69)	.354 [a]	.012

Note. * Significant at Bonferroni-adjusted α value. [a] α value = .05. [b] Bonferroni-adjusted α value = .017.

as the cell count was lower than five. However, seven participants in the ASD group, compared to zero participants in the control group, reported taking antihypertensive medication.

Cuff Inflation Diary

With respect to information collected from the cuff inflation diary, a significant main effect for caregiving was found, such that participants from the ASD group reported being engaged in caregiving more frequently than participants from the control group (see Table 28). However, there was no significant main effect for gender, and no significant interaction effect for group x gender. No significant main effects for group nor gender, and no significant interaction effects for group x gender, were observed for engaging in work activities or social interactions, location, or mood ratings on the day of the study.

Table 28

ANOVA Results for Cuff Inflation Diary Information

Variable		ASD Group		Control Group			ANOVA Results		
	Gender	M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Activities: Caregiving	Mothers	13.16 (9.84)	2 – 39	8.26 (8.81)	0 – 36	Group	10.25 (1, 75)	.002 ^{a} *	.120
	Fathers	12.26 (9.53)	0 – 31	2.44 (4.82)	0 – 19	Gender	1.31 (1, 75)	.257 ^{a}	.017
	Total	12.58 (9.47)	0 – 39	6.18 (8.44)	0 – 36	Group x Gender	1.14 (1, 75)	.289 ^{a}	.015
Activities: Work	Mothers	6.95 (7.50)	0 – 20	7.05 (5.43)	0 – 15	Group	1.65 (1, 75)	.203 ^{a}	.021
	Fathers	4.74 (5.77)	0 – 21	7.89 (8.98)	0 – 28	Gender	0.55 (1, 75)	.462 ^{a}	.007
	Total	5.55 (6.65)	0 – 21	7.62 (7.51)	0 – 28	Group x Gender	0.32 (1, 75)	.572 ^{a}	.004
Activities: Social interaction	Mothers	19.47 (8.26)	6 – 39	15.95 (7.96)	2 – 27	Group	1.90 (1, 75)	.172 ^{a}	.025
	Fathers	20.79 (8.38)	5 – 32	17.72 (11.95)	0 – 33	Gender	0.32 (1, 75)	.575 ^{a}	.004
	Total	19.33 (8.82)	5 – 39	16.36 (9.93)	0 – 33	Group x Gender	0.03 (1, 75)	.869 ^{a}	.000

Note. * Significant at Bonferroni-adjusted α value. ^{a} Bonferroni-adjusted α value = .017.

Table 28, Continued

ANOVA Results for Cuff Inflation Diary Information

Variable	Gender	ASD Group		Control Group			ANOVA Results		
		M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Location: Home	Mothers	27.74 (10.46)	7 – 43	25.95 (6.13)	15 – 38	Group	5.48 (1, 75)	.022 [a]	.068
	Fathers	27.32 (8.34)	11 – 46	16.83 (12.52)	1 – 39	Gender	2.45 (1, 75)	.122 [a]	.032
	Total	26.80 (9.90)	7 – 46	21.62 (10.64)	1 – 39	Group x Gender	3.35 (1, 75)	.071 [a]	.043
Location: Work	Mothers	1.00 (4.36)	0 – 19	0.11 (0.32)	0 – 1	Group	3.48 (1, 68)	.066 [a]	.049
	Fathers	2.42 (5.76)	0 – 21	7.00 (9.19)	0 – 28	Gender	4.86 (1, 68)	.031 [a]	.067
	Total	1.17 (4.29)	0 – 21	3.46 (7.22)	0 – 28	Group x Gender	3.18 (1, 68)	.079 [a]	.045
Location: Car [b]	Mothers	1.79 (2.12)	0 – 6	2.21 (1.90)	0 – 5	Group	0.00 (1, 75)	.984 [a]	.000
	Fathers	2.79 (2.44)	0 – 7	2.50 (2.66)	0 – 10	Gender	0.99 (1, 75)	.323 [a]	.013
	Total	2.28 (2.30)	0 – 7	2.28 (2.24)	0 – 10	Group x Gender	0.22 (1, 75)	.642 [a]	.003
Location: Public place	Mothers	3.58 (2.78)	0 – 10	2.95 (2.39)	0 – 7	Group	0.26 (1, 75)	.610 [a]	.003
	Fathers	2.11 (2.49)	0 – 9	3.33 (3.05)	0 – 9	Gender	0.68 (1, 75)	.413 [a]	.009
	Total	2.73 (2.69)	0 – 10	3.03 (2.68)	0 – 9	Group x Gender	2.55 (1, 75)	.115 [a]	.033

Note. [a] Bonferroni-adjusted α value = .013. [b] ANCOVA; covariate = PRS score.

Table 28, Continued

ANOVA Results for Cuff Inflation Diary Information

Variable	Gender	ASD Group		Control Group			ANOVA Results		
		M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Mood: Sad	Mothers	40.32 (13.46)	9 – 66	35.68 (9.60)	19 – 62	Group	4.79 (1, 69)	.032 [a]	.065
	Fathers	37.26 (8.37)	15 – 49	29.47 (13.54)	1 – 45	Gender	0.02 (1, 69)	.881 [a]	.000
	Total	38.72 (11.41)	9 – 66	32.58 (11.99)	1 – 62	Group x Gender	0.32 (1, 69)	.572 [a]	.005
Mood: Active	Mothers	76.32 (26.92)	17 – 117	75.84 (24.80)	41 – 124	Group	0.00 (1, 76)	.972 [a]	.000
	Fathers	73.16 (21.40)	14 – 105	68.05 (36.93)	4 – 136	Gender	0.65 (1, 76)	.424 [a]	.008
	Total	72.53 (25.71)	14 – 117	72.30 (30.75)	4 – 136	Group x Gender	0.39 (1, 76)	.535 [a]	.005
Mood: Interested	Mothers	84.42 (34.38)	15 – 157	80.37 (25.00)	45 – 117	Group	0.51 (1, 75)	.479 [a]	.007
	Fathers	81.53 (22.83)	22 – 113	71.00 (35.07)	1 – 131	Gender	0.93 (1, 75)	.339 [a]	.012
	Total	80.93 (29.60)	15 – 157	76.28 (30.04)	1 – 131	Group x Gender	0.52 (1, 75)	.475 [a]	.007
Mood: Stressed	Mothers	46.89 (18.58)	10 – 88	39.58 (10.07)	20 – 59	Group	4.84 (1, 76)	.031 [a]	.060
	Fathers	44.00 (14.08)	18 – 78	33.37 (16.16)	2 – 54	Gender	1.34 (1, 76)	.251 [a]	.017
	Total	45.00 (16.17)	10 – 88	37.53 (14.09)	2 – 59	Group x Gender	0.39 (1, 76)	.533 [a]	.005

Note. [a] Bonferroni-adjusted α value = .006.

Table 28, Continued

ANOVA Results for Cuff Inflation Diary Information

Variable	Gender	ASD Group		Control Group			ANOVA Results		
		M *(SD)*	Range	M *(SD)*	Range		*F* (*df*)	*p*	η^2
Mood: Upset	Mothers	38.90 (13.48)	9 – 75	35.53 (8.17)	19 – 50	Group	2.34 (1, 76)	.130 [a]	.003
	Fathers	36.90 (8.70)	12 – 49	29.90 (13.85)	1 – 47	Gender	1.11 (1, 76)	.296 [a]	.014
	Total	37.58 (11.34)	9 – 75	33.58 (11.98)	1 – 56	Group x Gender	0.59 (1, 76)	.446 [a]	.008
Mood: Excited	Mothers	43.47 (17.13)	9 – 82	45.63 (12.69)	28 – 69	Group	0.03 (1, 76)	.873 [a]	.000
	Fathers	52.21 (22.68)	13 – 95	32.78 (26.94)	1 – 92	Gender	0.51 (1, 76)	.477 [a]	.007
	Total	47.38 (20.03)	9 – 95	46.63 (21.91)	1 – 92	Group x Gender	1.64 (1, 76)	.205 [a]	.021
Mood: Frustrated	Mothers	42.32 (18.86)	8 – 97	38.79 (12.11)	19 – 65	Group	1.40 (1, 75)	.241 [a]	.018
	Fathers	41.32 (12.69)	13 – 74	32.78 (13.93)	1 – 52	Gender	0.51 (1, 75)	.476 [a]	.007
	Total	41.33 (15.71)	8 – 97	37.33 (14.49)	1 – 73	Group x Gender	0.45 (1, 75)	.504 [a]	.006
Mood: Alert	Mothers	99.95 (39.63)	13 – 157	97.79 (33.93)	37 – 160	Group	0.64 (1, 76)	.426 [a]	.008
	Fathers	102.68 (33.82)	18 – 170	83.68 (43.76)	2 – 152	Gender	0.55 (1, 76)	.461 [a]	.007
	Total	98.35 (37.84)	13 – 170	91.48 (39.01)	2 – 160	Group x Gender	1.37 (1, 76)	.245 [a]	.018

Note. [a] Bonferroni-adjusted α value = .006.

Discussion

The present study compared parent-report and physiological measures of stress between mothers and fathers of children with and without ASD, with both groups of parents matched by parent and child age and gender. Mothers reported higher parenting responsibility than fathers across both parenting groups. Parents of children with ASD reported significantly higher anxiety, depression, and parenting stress than parents of typically developing children. Parents of children with ASD also had lower levels of cortisol on the third morning cortisol measure. However, the groups did not significantly differ with respect to sAA levels or mean BP or HR.

Parenting Responsibility and Parent-Reported Stress Measures

Mothers within both parenting groups reported significantly higher levels of parenting responsibility than fathers, but there was no significant difference in levels of parenting responsibility between the two groups. This is consistent with existing research on paternal involvement, with fathers previously reported to spend about one-fourth as much time as mothers directly engaged with their children, and about one-third as much time being accessible to their children (Lamb & Tamis-Lemonda, 2004). This indicates that mothers are still the primary caregivers in many families, and suggests that this gender difference in parental involvement may occur whether or not the child has a disability.

Regardless of parental gender, parents of children with ASD reported significantly higher levels of anxiety, depression, overall parenting stress, parental distress, difficult child scores, and parent-child dysfunctional interactions than parents of typically developing children. These findings support previous research that has reported higher psychological distress among parents of children with ASD than parents of typically developing children (e.g., Duarte et al., 2005; Mugno et al., 2007; Rao & Biedel, 2009). The fact that heightened anxiety, depression, and parenting stress were reported by both mothers and fathers of children with ASD highlights the importance of providing supports and stress interventions to parents of children with ASD. Furthermore, because chronic stress, anxiety, and depression can contribute to the development of health problems, these findings lend further support to the importance of investigating physiological measures of stress among parents of children with ASD.

Salivary Biomarkers of Stress

As discussed in Chapters 2 and 3, mothers and fathers of children with ASD have often been found to experience hypocortisolism. In the present study, AUC_G, $AUC_{I,}$ and sAA levels did not significantly differ between parents of children with ASD and parents of typically developing children, nor did the type of morning cortisol response (i.e., cortisol responding or non-responding). However, analysis of absolute cortisol values using ANOVA analyses with Bonferroni adjustment revealed that parents of children with ASD had significantly lower levels of cortisol on the third awakening response (i.e., 30 minutes after waking) than parents of typically developing children. Cortisol levels, which typically reach their peak 30 minutes after waking, were at their highest for parents of typically developing children for this measure, but had actually declined from baseline among parents of children with ASD. This provides further evidence of dysregulated cortisol awakening responses among parents of children with ASD. Given the known links between hypocortisolism and stress-related diseases (Heim et al., 2000), these findings further support the need for health assessments and stress interventions for such parents.

CV Activity

Gallagher and Whiteley (2012) reported that caregivers of children with DD, including ASD, had higher mean SBP than caregivers of typically developing children. However, in the present study, CV activity did not significantly differ between parents of children with ASD and parents of typically developing children. Fathers were found to have higher mean awake SBP, 24-hour SBP, and awake DBP than mothers across both groups of parents, while mothers were found to have higher 24-hour HR. As men are known to experience higher BP than women, and women are known to experience higher HR than men (Fuster & Kelly, 2010), these findings suggest that gender is the strongest predictor of CV activity, as opposed to having a child with ASD or level of self-reported stress. Furthermore, the type of night-time reduction in SBP and DBP (i.e., dipping status) did not significantly differ between the two groups. These findings are inconsistent with those of Gallagher and Whiteley (2012). However, in the present study, seven parents of children with ASD, in comparison to zero parents of typically developing children, were taking antihypertensive medication. This indicates that parents of children with ASD were more likely to have hypertension than parents of typically developing children, although differences in CV activity did not reach statistical significance, even after controlling for use of antihypertensive medication. To our knowledge, Gallagher and Whiteley's study is the only other study that has investigated ambulatory BP among parents of children with ASD. The conflicting findings between their study and the present research indicates that further investigation of ambulatory BP and HR among parents of children with ASD is necessary to elucidate the effects, if any, of caring for a child with ASD on CV health.

In addition to mean CV activity, the present study compared CV variability between parents of children with ASD and parents of typically developing children. To our knowledge, no such comparison has been made previously. As discussed in Chapter 3, high BP and *low* HR variability using standard deviations of readings obtained from ABP monitoring have been reported to independently predict CV mortality (Kikuya et al., 2000; Pickering & James, 1994). Similar to Study 2, the present study found that fathers had higher awake SBP and HR variability than mothers. This was found across both parenting groups, suggesting that gender may be the strongest predictor of CV variability. However, asleep SBP and DBP variability were found to have significant interaction effects, with fathers of children with ASD and mothers of typically developing children having the highest sleeping BP variability. Thus, while it would appear that in general, gender was the strongest predictor of CV variability, parenting group may also be a predictor of night-time CV variability. However, given that the interaction effect was only observed with sleeping BP, differences in the effects of the ABP monitor on sleeping patterns on the night of the study could have influenced sleeping BP variability. Further research is necessary to determine why mothers and fathers of children with ASD had lower and higher sleeping BP variability, respectively, than mothers and fathers of typically developing children.

Supports and Coping Strategies

Lower levels of social support have been reported to predict stress, depression, and anxiety among parents of children with ASD (Boyd, 2002). This is consistent with the present study, in which parents of children with ASD reported lower levels of social support but higher stress, anxiety, and depression than parents of typically developing children. Fathers reported significantly less social supports than mothers, while parents of children with ASD reported less social supports than parents of typically developing children, although this fell short of reaching statistical significance. The two groups did not significantly differ in the quality of social supports reported, or the quantity of unmet supports. These findings suggest that fathers have less access to social supports than mothers, regardless of parenting group. Furthermore, although it failed to reach statistical significance, it would also appear that parents of children with ASD have somewhat less access to social supports than parents of typically developing children. Given the previous reported links between low levels of social support and higher levels of stress, depression, and anxiety, it is possible that parents of children with ASD could benefit from greater access to social supports.

Coping strategies also have the potential to alleviate or exacerbate stress levels among parents of children with ASD (e.g., Hastings et al., 2005a; Dunn, Burbine, Bowers, & Tantleff-Dunn, 2001). As outlined by Hastings et al. (2005a), parents of children with ASD may use different coping strategies than parents of typically developing children or children with other disabilities, given that they experience different stressors, and experience those stressors within different environments. In the present study, although the two groups did not differ regarding their use of most coping strategies, parents of children with ASD reported higher use of instrumental support, planning, acceptance, and self-blame than parents of typically developing children. Mothers and fathers of children with ASD reported similar use of these coping strategies. While planning, acceptance, and instrumental support are problem-focused strategies, and are therefore likely to be associated with more positive outcomes for parents, the use of self-blame was found to be a strong predictor of anxiety, depression, and distress among mothers in Chapter 2. This suggests that parents of children with ASD may benefit from interventions such as ACT in order to manage their emotions and reduce the negative outcomes found to be associated with the use of self-blame in Chapter 2.

Sleep

As reported in Chapters 2 and 3, children with ASD are known to have high levels of sleeping problems and consequently, parents of children with ASD often experience poor sleep quantity and quality. In the present study, parents of children with ASD reported significantly higher levels of overall sleep dysfunction, and dysfunction in the areas of sleep quality, duration, and disturbances than parents of typically developing children, although the two groups did not significantly differ with respect to sleep latency, habitual sleep efficiency, or use of sleeping medication. The present study confirms that parents of children with ASD are at higher risk of poor sleep than parents of typically developing children. Sleep dysfunction can strongly impair human functioning, and has been reported to be associated with health, depression, tension, and anger (Pilcher, Ginter, & Sadowsky, 1997; Pilcher & Huffcutt, 1996). Thus, the present study provides further support for the importance of providing supports and respite services for parents of children with ASD who are experiencing high levels of sleep dysfunction. Furthermore, as discussed in Chapter 2, sleep assessments and interventions for children with ASD could have health benefits for parents, in addition to benefiting the child.

Demographic and Health-Relevant Variables

Some parents may be unable to work full-time when they have a child with ASD due to the commitment required in caring for the child. For instance, Dillenburger et al. (2010) reported that only 56% of the parents surveyed in their study were currently in employment. Similarly, in the present study, a significantly higher number of parents of children with ASD were unemployed, compared to parents of typically developing children. This has the potential to contribute to financial concerns for parents of children with ASD, in addition to limiting social support or respite from caregiving for parents who are unable to engage in employment.

Stress has been linked to increased health-risk behaviours (i.e., behaviours that can increase a person's risk of health problems), such as smoking or alcohol consumption (Morrison & Bennett, 2009). However, in the present study, there were no significant differences between the two parenting groups in relation to smoking, caffeine intake, or exercise duration. This suggests that parents of children with ASD were not more likely to engage in health-risk behaviours. Fathers reported significantly higher alcohol consumption than mothers, regardless of parenting group. The two parenting groups also did not differ in relation to the use of over-the-counter or prescription medication, with the exception of antihypertensive medication, as discussed previously. However, mothers of children with ASD reported the highest quantity of illnesses, suggesting that parenting a child with ASD is associated with a higher number of health problems for mothers in particular. These findings suggest that parents of children with ASD are not more likely to engage in health-risk behaviours, but that mothers of children with ASD are at risk of experiencing a higher number of illnesses. This further supports the need for health screening and supports for parents of children with ASD.

Daily Activities and Mood Ratings

Previous research by Seltzer et al. (2009) found that parents of children with disabilities did not differ from parents of typically developing children in their patterns of time use, or time spent engaging in caregiving. In the present study, although parents of children with ASD did not significantly differ in most areas of time use (i.e., location, work activities, or social interactions), they *did* report engaging in significantly more caregiving interactions on the day of the study than parents of typically developing children. This is in contrast to Konstantareas and Homatadis (1992), who found that parents of children with ASD reported *less* involvement with their child than parents of typically developing children. Furthermore, in contrast to Seltzer, Abbeduto, Krauss, Greenberg, and Swe (2004), who found that parents of children with disabilities reported more negative and less positive affect on a daily basis, parents of children with ASD in the present study did not significantly differ from parents of typically developing children in their mood ratings on the day of the study. Spending more time engaged in caregiving has been hypothesised to be associated with increased parental stress due to the higher demands for care and supervision that children with ASD have compared to typically developing children (Seltzer et al., 2009). However, this did not appear to be the case in the present study, with parents of children with ASD reporting higher levels of caregiving, but similar ratings of negative moods such as stress, than parents of typically developing children. Thus, the findings of the present study indicate that parents may not necessarily experience more negative affect as a result of increased time spent directly caring for their child with ASD.

Limitations and Future Research Directions

In the present study, extraneous life-events or stressors, such as bereavement or a recent job loss, were not controlled for in either group. Furthermore, although the participants in both groups were matched by child age and gender, it was not possible to match participants on all characteristics, such as family size or participant age. Consequently, factors that were not measured could have contributed to differences or similarities observed between the two groups. Additionally, as in

Chapters 2 and 3, cortisol and sAA data were only available for a subset of the sample, possibly reducing statistical power for these measures. However, this is the first study to directly compare parent-reported stress, salivary biomarkers, and 24-hour ambulatory BP between parents of children with ASD and typically developing children, and provides important insight into stress and health among parents of children with ASD. Further research incorporating similar physiological measures could provide further insight into the effects of parenting a child with ASD on parental health and wellbeing.

Chapter 5

General Discussion

Summary of Studies

The present research investigated levels of stress among parents of children with ASD, with particular emphasis on salivary biomarkers of stress and ambulatory CV activity. Three inter-related studies were conducted to help advance understanding of the impact parenting a child with ASD has on parent-reported wellbeing and physiological activity. In all three studies, participants completed a set of questionnaires regarding child (e.g., behaviour problems) and parent (e.g., coping strategies used) variables. Participants collected saliva samples which were analysed for cortisol and sAA levels. An ABP monitor was worn for a 24-hour period, which recorded SBP, DBP, HR, MAP, and PP at 20 minute and 45 minutes intervals during the day and night, respectively. Additionally, a diary was completed to assess locations, activities, and moods on the day of the study. Study 1 aimed to identify predictors of parent-reported stress, anxiety, and depression, cortisol, sAA, and CV activity among mothers of children with ASD. The study investigated factors that could exacerbate (e.g., child behaviour problems) or moderate (e.g., social support) parenting stress. Next, Study 2 examined if there were differences in the effects of parenting a child with ASD based on parental gender or level of parenting responsibility. The study employed dyadic comparisons to ensure parents were matched on major child characteristics. Finally, in Study 3, in order to control for stress related to normative parenting, parent-report and physiological measures of stress were compared between mother-father dyads of children with ASD and a matched control group of mother-fathers dyads of typically developing children. The measurement of a number of physiological parameters of stress provided a stronger investigation of stress in parents of children with ASD than is typically conducted, as the majority of past studies have focused exclusively on parental reports of stress and health.

Study 1

Study 1 identified predictors of stress measures in 74 mothers of children with ASD using hierarchical multiple regression models. According to Zigmond and Snaith's (1983) criteria, group anxiety scores fell within the mild (8–10) to moderate (11–14) range, while group depression scores fell within the normal (0–7) to mild range. Thus, mothers of children with ASD, as a group, did not appear to have clinically-significant levels of anxiety or depression. However, high levels of parenting stress were reported across all subdomains of the PSI. Group means for all PSI subscales fell above the 85th percentile, indicating clinically-significant levels of parenting stress. With respect to physiological measures, group mean values for CV activity fell within the normotensive range, while sAA levels did not significantly differ from expected averages reported by Salimetrics (2012c). However, cortisol values were found to significantly differ from average values reported by Wust et al. (2000). Mothers of children with ASD were found to have significantly lower cortisol levels immediately, 30, and 45 minutes after waking in comparison to Wust et al.'s (2000) values, providing evidence that mothers of children with ASD may be at risk of experiencing hypocortisolism.

Issues related to unmet service needs and sleep, in particular, were found to predict a range of negative maternal outcomes. Mothers reporting more unmet service needs of their child predicted higher maternal depression, distress, and CV activity (i.e., awake and asleep SBP, asleep and 24-hour MAP, and asleep PP). Overall maternal sleep dysfunction predicted maternal anxiety and awake and 24-hour MAP, while higher reports of child sleep problems were found to predict higher maternal anxiety, 24-hour SBP, awake HR, and awake and asleep PP. These findings indicate that a higher quantity of unmet service needs and sleep problems were associated with more negative maternal outcomes. With respect to coping strategies, only maladaptive coping strategies were found to be significant predictors of maternal outcomes. Use of self-blame was found to predict maternal anxiety, depression, and distress, while use of behavioural disengagement predicted 24-hour SBP. However, a higher quantity of social support predicted lower levels of depression. Variables related to the child's diagnosis and behaviour were also predictive of maternal outcomes. Later detection of a problem by the parents predicted higher asleep DBP and MAP, and a smaller night-time drop in DBP. Oppositional behaviour and socialisation deficits (one of the core deficits in ASD) predicted maternal stress and parent-child dysfunction interactions, while oppositional behaviour also predicted difficult child scores. However, higher adaptive behaviour, on the other hand, predicted *lower* parent-child dysfunctional interactions and difficult child scores, while higher fine motor skills predicted lower AUC_I.

This study was only the second to investigate ambulatory BP and HR in mothers of children with ASD. It is also the first study to combine the measurement of ambulatory blood pressure, heart rate, and salivary biomarkers among mothers of children with ASD, providing useful information about the physiological effects of chronic stress on such caregivers. However, because Study 1 focused exclusively on mothers of children with ASD, it provided no insight into the different effects, if any, for mothers and fathers caring for a child with ASD.

Study 2

Having identified significant predictors of stress for mothers of children with ASD, Study 2 extended the same research methodology to fathers of children with ASD. In doing so, we aimed to test for differences between maternal and paternal outcomes as a result of parenting a child with ASD. The study included 19 mother-father dyads, which ensured that mothers and fathers were matched on major child characteristics, such as child age or gender. The use of mother-father dyads also provided an opportunity to assess the different effects on mothers and fathers of parenting the same child within the same environment. Similarly to Study 1, low levels of anxiety and depression were reported by both mothers and fathers, while both groups reported high levels of parenting stress within the clinically-significant range. Mean sAA levels were not significantly different between both groups. For mothers, cortisol levels immediately after waking did not statistically differ from Wust et al.'s (2000) reported averages; however, the 30 minute and 45 minute cortisol values, along with all three cortisol values for fathers, were significantly lower than average. This is a further indication that mothers *and* fathers of children with ASD may experience hypocortisolism. Group mean CV measures fell within the normotensive range for both mothers and fathers.

For each variable, the father's score was subtracted from the mother's score for each dyad, providing a difference score for each. Dyad difference scores were then tested for significance of difference from zero using one-sample *t*-tests with family-wise Bonferroni adjustment. Dyad comparisons revealed that mothers reported significantly higher levels of parenting responsibility, anxiety, depression, parenting stress, and difficult child scores than fathers. The two groups did not significantly differ with respect to parent-child dysfunctional interactions, difficult child scores,

AUC_G, AUC_I, or sAA, nor did they differ with respect to the size or the type of SBP or DBP dipping. However, fathers had significantly higher awake and asleep SBP variability and awake DBP variability than mothers, while mothers had significantly lower asleep HR variability than fathers. With respect to coping strategies employed, fathers reported significantly higher use of behavioural disengagement and humour than mothers, while mothers reported significantly higher use of instrumental support, planning, and acceptance than fathers. Mothers also reported having access to a higher quantity of social supports, but lower satisfaction with social supports, than fathers. Finally, mothers reported having significantly more illnesses than fathers, while fathers reported drinking alcohol more frequently than mothers. Correlation analyses also revealed that fathers' stress was significantly correlated with mothers' anxiety and depression, though mothers' stress was not significantly correlated with fathers' anxiety or depression.

The findings of Study 2 may have been affected by the small sample size and potential sampling bias (e.g., fathers may have been more involved in caregiving in families in which the father agreed to participate in the research, in comparison to families who did not participate). Nonetheless, this is the first study to compare both salivary biomarkers and ambulatory hemodynamic activity between mother-father dyads parenting children with ASD, and provides an important comparison of maternal and paternal stress and wellbeing. Given that all parents can experience stress as a result of their parenting roles, it is important to control for stress related to normative parenting.

Study 3

In order to control for stress related to normative parenting, Study 3 compared the parent-report and physiological measures of stress of the mother-father dyads of children with ASD from Study 2 to those of a matched control group of mother-father dyads with typically developing children, using the same methodological design as Studies 1 and 2. The issues regarding the small sample size and potential sampling bias that affected Study 2 were also present in Study 3, because the same parents of children with ASD were included in both studies. However, the use of mother-father dyads ensured that the mothers and fathers of typically developing children were matched on major child characteristics, while the two parenting groups were also matched by parent and child age and gender.

Mothers in both parenting groups reported significantly higher levels of parenting responsibility than fathers, suggesting that mothers are more frequently the primary caregivers, regardless of whether the child has an ASD. As expected, parents of children with ASD reported significantly higher levels of anxiety, depression, parenting stress, parental distress, parent-child dysfunctional interactions, and difficult child scores than parents of typically developing children. There were no significant differences between the two groups with respect to AUC_G, AUC_I, the number of cortisol responders, or sAA levels. However, parents of children with ASD had significantly lower raw cortisol levels than parents of typically developing children 30 minutes after waking at a time when cortisol levels should peak. This indicates some cortisol dysregulation in comparison to parents of typically developing children. Some surprising results emerged in relation to CV activity, with no significant differences between the two parenting groups' mean CV activity, CV variability, or the size or type of SBP and DBP dip. However, some gender differences emerged, with fathers having higher BP than mothers, and mothers having higher HR than fathers. Men are known to have higher BP and lower HR than women, so this finding suggests that stress related to parenting a child with

ASD did not significantly contribute to gender-specific differences in mean BP and HR. Similarly, fathers had higher awake SBP and *higher* HR variability than mothers, with higher BP variability and lower HR variability linked to end-organ damage and increased risk of CV mortality (e.g., Frattolla et al., 1993). However, sleeping BP variability was found to have a significant interaction effect, with fathers of children with ASD and mothers of typically developing children found to have the highest sleeping BP variability. This suggests that the risk of CV disease for mothers and fathers may depend on the relative risk imposed by the impact of CV variability in the respective groups.

In addition to comparing parent-report and physiological measures of stress between the two groups, Study 3 also compared other variables such as coping strategies and social supports. Parents of children with ASD reported significantly higher use of instrumental support, planning, acceptance, and self-blame than parents of typically developing children, though they did not differ on use of other coping strategies. Although the two groups did not significantly differ in the quantity of unmet supports or the quality of social supports reported, fathers reported a significantly lower quantity of social supports than mothers. Parents of children with ASD also reported a lower quantity of social supports than parents of typically developing children, though this fell short of reaching statistical significance. As expected, parents of children with ASD also reported higher levels of sleep dysfunction and dysfunction in the areas of subjective sleep quality, sleep duration, and sleep disturbances than parents of typically developing children. Parents of children with ASD were also more likely to be unemployed, and a higher number of parents of children with ASD were taking antihypertensive medication. However, the two groups did not differ with respect to any other medication usage or health-risk behaviours such as smoking. Fathers across both groups reported higher alcohol intake than mothers, while mothers reported a higher number of illnesses than fathers, and parents of children with ASD reported more illnesses than parents of typically developing children. Finally, parents of children with ASD reported spending significantly more time engaging in caregiving activities on the day of the study than parents of typically developing children, although they did not differ in other aspects of time use, location, or mood on the day of the study.

Implications of the Research Findings

The current thesis demonstrated that mothers (Chapters 2 and 3) and fathers (Chapter 3) of children with ASD often experience clinically significant levels of parenting stress. They were also found to experience higher levels of stress than parents of typically developing children (Chapter 4). Although previous research has also reported high levels of stress among parents of children with ASD (e.g., Duarte et al., 2005; Eisenhower et al., 2005; Hayes & Watson, 2013), the use of physiological measures in the present thesis furthers understanding of the effects on parents of caring for a child with ASD. There are several important issues arising from the research that, if addressed, may help to support parents of children with ASD more effectively.

Health Screening: Prevention and Treatment of Health Problems

The research findings in the present thesis emphasise the importance of considering the effects that caring for a child with ASD can have on a parent’s psychological and physical wellbeing. In Chapters 2 and 3, parents of children with ASD reported high levels of parenting stress, and they were found to experience higher levels of anxiety, depression, and parenting stress than parents of typically developing children in Chapter 4. Chronic stress is well documented to contribute to the development of anxiety, depression, and physical health problems such as coronary heart disease (e.g., Lovallo, 2005; Reid et al., 2001; Sapolsky, 2004). Therefore, it is unsurprising that in Chapter 4, parents of children with ASD were found to report a higher number of illnesses than parents of typically developing children.

The results relating to physiological measurements in the present thesis revealed a number of potential risk factors to health, which may shed further light on the link between chronic stress and health for these parents. The findings in relation to physiological markers were more limited than would have been expected, which may have been at least partly due to reduced statistical power in these analyses as a result of the smaller sample size. However, the research did identify a number of areas of potential concern among parents of children with ASD. Importantly, evidence of blunted cortisol awakening responses, or hypocortisolism, among parents of children with ASD was found in all three studies. Although one of the first studies investigating cortisol among parents of children with ASD found no difference with levels among parents of typically developing children (Marinovic-Curin et al., 2008), there is a growing body of literature demonstrating that parents of children with ASD are at risk of experiencing hypocortisolism (Dykens & Lambert, 2013; Lovell et al., 2012; Lovell et al., 2013; Ruiz-Robledillo et al., 2013; Seltzer et al., 2009; Seltzer et al., 2010; Wong et al., 2012). Previous studies have predominantly focused on parents of older children, with the present study having a younger sample in all studies ($M < 8.5$ years) than reported in previous literature. Thus, research has now reported hypocortisolism in parents of younger (the present study) and older (e.g., Seltzer et al., 2010; Wong et al., 2012) individuals with ASD, as well as with mothers and fathers (e.g., Lovell et al., 2012; Ruiz-Robledillo et al., 2013). This indicates that parents do not need to be parenting a child with ASD for decades in order to be at risk of experiencing hypocortisolism.

The potential for parents of children with ASD to develop hypocortisolism is a serious risk factor for the development of health problems, given that low levels of cortisol have also been associated with lower health ratings (e.g., Dykens & Lambert, 2013), and an increased risk of health problems (Heim et al., 2000). Furthermore, parents of children with ASD in the present study had somewhat higher than average levels of sAA, which is similar to increased SNS activity and blunted cortisol responses that have been reported among individuals with PTSD (Fries et al., 2005). However, as the difference in sAA levels did not reach statistical significance, further investigation is warranted with respect to sAA levels among parents of children with ASD. Fries et al. (2005) have proposed that hypocortisolism is a protective response which has the benefit of reducing the harmful effects of glucocorticoid responses to daily stressors, with the downside being an increase in symptoms such as pain, fatigue, and high stress sensitivity. Consequently, prevention of hypocortisolism through stress interventions may be the most effective approach in order to preclude the development of associated health problems.

In addition to dysregulation of HPA-axis activity, abnormal CV activity was also found among parents of children with ASD in the present study. Only one study (Gallagher & Whiteley, 2012) has previously researched ambulatory BP in parents of children with ASD. Gallagher and Whiteley’s (2012) finding that parents of children with DD (including ASD) had higher ambulatory BP than parents of children with ASD was not supported in the present thesis, with gender found to be a more important predictor of CV activity in Chapter 4. Furthermore, BP and HR for the ASD groups fell within the normotensive range, which was not expected. However, more subtle indicators of abnormal CV activity were observed. Extreme SBP or DBP dipping was found in a high number of mothers (*n* = 15) and fathers (*n* = 12) in Chapter 3. Extreme dipping is associated with an increased risk of silent cerebrovascular disease and stroke (Hoshide et al., 2002). Higher BP variability and extreme dipping together may accelerate target-organ damage (Shimada & Kario, 1997), which could result in an increased risk of health problems. Although gender appeared to be the strongest predictor of CV variability, an interaction effect was also observed in Chapter 4, with fathers of children with ASD and mothers of typically developing children found to have the highest night-time BP variability. It is unclear why fathers of children with ASD and mothers of typically developing children had the highest night-time BP variability, which necessitates further research on CV activity in both parenting groups.

The physiological outcomes identified in this thesis could contribute to the development of health problems for parents of children with ASD. Treatment of such health problems may be difficult given that, for example, antihypertensive medication has been reported to be less effective for extreme BP dippers than normal dippers (Hoshide et al., 2002). Furthermore, cortisol deficiency is usually treated, if warranted, with corticosteroids, which can have serious side-effects including dependency and the suppression of the adrenal response (Henzen et al., 2000). Thus, given the difficulties with treating stress-related health problems, prevention should be the primary goal for parents of children with ASD.

The growing body of literature demonstrating risk of hypocortisolism among parents of children with ASD, along with the present findings of CV abnormalities, necessitates that the health of these parents is made a priority following a diagnosis of ASD. In addition to providing support around the difficult diagnostic process, the findings in the present thesis suggest that all parents should be offered access to stress interventions to help them cope with stress arising from parenting a child with ASD. Stress interventions, which will be discussed in further detail below, can take different forms, but have been found to improve a range of psychological and physical outcomes. For example, a meta-analysis of mindfulness-based stress reduction (MBSR) found that MBSR was associated with a range of improvements, such as anxiety, coping styles, medical symptoms, and quality-of-life estimates across diverse populations (Grossman, Niemann, Schmidt, & Walach, 2004). In addition to stress interventions, routine health screening, with a particular emphasis on adrenal and ambulatory hemodynamic activity, should be provided to parents on a regular basis. Although there are costs associated with implementing health screening programmes, they are becoming increasingly popular around the world as a form of preventative medicine (Morrison & Bennett, 2009). Health screening programmes could facilitate the early identification and treatment of dysregulated cortisol and CV activity, which could prevent the development of related illnesses or health problems among parents of children with ASD.

Maternal and Paternal Needs

In order to ensure the most effective delivery of stress interventions, it is important to take into consideration the different needs of mothers and fathers, which were discussed in Chapter 3. Although the sample size in Study 2 was somewhat small, and future replication with a larger sample size would be of benefit in identifying different maternal and paternal needs, some important differences emerged between mothers and fathers. Mothers reported higher levels of parenting responsibility, anxiety, depression, and parenting stress than fathers, which suggests that they may be in greater need of support than fathers. Indeed, although mothers reported having more access to social supports than fathers, they actually reported *lower* satisfaction with social supports. This appeared to be a gender-specific difference, as fathers of typically developing children also reported lower levels of social support than mothers of typically developing children in Chapter 4. This is consistent with previous reports that men have smaller social support networks than women (e.g., Antonucci & Akiyama, 1987), and suggests that men may require less access to social support than women. Consequently, mothers in particular may benefit from greater access to high-quality social supports.

Different coping strategies were also reported to be used more frequently by mothers (i.e., instrumental support, planning, and acceptance) and fathers (i.e., behavioural disengagement and humour), indicating that mothers and fathers are likely to approach and cope with stressors in different ways. Thus, it follows that they may require different approaches in managing their stress levels, which underscores the importance of individualising supports and services. In Chapter 3, it was also reported that fathers' stress was correlated with mothers' anxiety and depression. Correlations between maternal and paternal outcomes have also been reported by Hastings (2003) and Hastings et al. (2005b). Therefore, although mothers and fathers may experience different levels of stress, anxiety, and depression, and may cope with them in different ways, it is important that both parents are included in interventions, because parental outcomes may be linked.

Stress Management Interventions

The present research identified clinically significant levels of stress among parents of children with ASD, and higher levels of anxiety and depression than parents of typically developing children. Furthermore, in Chapter 2, use of self-blame and behavioural disengagement significantly predicted anxiety, depression, parental distress, and 24-hour SBP among mothers of children with ASD. Parents of children with ASD also reported higher use of self-blame than parents of typically developing children in Study 3, though they also reported higher use of acceptance, planning, and instrumental support. Again, replication of these findings with a larger sample size would be beneficial, due to the relatively small sample sizes in Study 2 and Study 3. However, these findings indicate that parents of children with ASD may benefit from stress management interventions. As mentioned above, there are many different forms of stress interventions, or stress management training. One intervention, MBSR, uses mindful meditation to reduce suffering associated with a range of disorders, and has been found to be effective across a range of clinical populations (e.g., individuals with clinical anxiety or depression), in addition to stressed nonclinical groups (Grossman et al., 2004). In a review of stress interventions for parents of children with ID, Hastings and Beck (2004) found that the strongest evidence base was for cognitive behavioural group interventions.

CBT focuses on overt behaviour change, as well as teaching people to assess and change distortions in their thinking, and to identify and establish more stable cognitive schema and structures (Jacobson et al., 1996). A similar approach, ACT, may also be effective for parents of children with ASD. ACT is an intervention that attempts to build psychological flexibility, or a person’s ability to connect fully and consciously with the present moment, and to change or persist with behaviour that is in line with their identified values (Hayes, Strosahl, & Wilson, 1999). ACT helps people to react more mindfully to negative thoughts and emotions, so that they no longer seem to be barriers (Ciarrochi & Blackledge, 2006). Blackledge and Hayes (2006) found that an ACT intervention was effective in reducing psychological avoidance and improving psychological outcomes for parents of children with ASD.

It is possible that interventions such as CBT or ACT could benefit parents of children with ASD in managing maladaptive coping styles such as self-blame, and could reduce stress, anxiety, depression, and associated physiological dysregulation.

Behavioural Skills Training

In addition to emotional supports such as MBSR, CBT, or ACT, parents of children with ASD may also benefit from parent training. The present thesis identified a number of behavioural excesses and deficits among children with ASD that predicted negative outcomes among parents. In Chapter 2, oppositional behaviour (excesses) and socialisation deficits were found to predict maternal stress and parent-child dysfunction interactions, while oppositional behaviour also predicted difficult child scores. Adaptive behaviour, on the other hand, was found to moderate maternal outcomes. Higher overall adaptive behaviour predicted lower parent-child dysfunction and difficult child scores, while higher fine motor skills predicted lower AUC_I. Additionally, child sleep problems, which will be discussed in further detail below, were found to predict a number of negative outcomes for mothers in Chapter 2, and parents of children with ASD reported significantly higher parental sleep dysfunction than parents of typically developing children in Chapter 4. These findings suggest that improvements in these areas could be associated with positive outcomes for the parent, as well as the child.

Research has consistently shown that interventions based on the principles of ABA are associated with positive outcomes for individuals with ASD (e.g., Eikeseth, Smith, Jahr, & Eldevik, 2002; Eikeseth, Smith, Jahr, & Eldevik, 2007; Eldevik et al., 2009; Howard, Sparkman, Cohen, Green, & Stanislaw, 2005; U.S. Department of Health and Human Services, 1999). Behavioural interventions have been effective in improving social skills (e.g., Nikopoulos & Keenan, 2007; Rogers, 2000), oppositional behaviour or noncompliance (e.g., Bullock & Normand, 2006; Schilling & Schwartz, 2004), adaptive behaviour (e.g., Baker-Ericzen, Stahmer, & Burns, 2007; Matson et al., 2012), and sleep (e.g., Mindell, Kuhn, Lewin, Meltzer, & Sadeh, 2006; Richdale, 1999). Furthermore, parent training has been successful in teaching parents of children with ASD to implement behavioural interventions with their children (e.g., Brookman-Frazee, Stahmer, Baker-Ericzen, & Tsai, 2006; Matson, Mahan, & Matson, 2009; McConachie & Diggle, 2007). In a review of parental needs, Dillenburger et al. (2010) also found that 100% of parents rated parent training in ABA as a future need. Therefore, given the negative outcomes associated with certain child behavioural deficits and excesses in the present research, using behavioural training to equip parents with skills to remediate and manage these behaviours could be effective in improving parental wellbeing. In addition to providing such parent training for parents of older children, a move towards making parent training the norm within all early intervention programmes could result in better outcomes for children with ASD (Matson et al., 2009) and, based on the findings in the present research, could also have the potential to improve parental wellbeing.

However, the findings in the present thesis also illustrate the need to consider the effects that stress could have on child outcomes and the effectiveness of parent training. High parental stress can decrease the effectiveness of early intervention for children with ASD (Osborne, McHugh, Saunders, & Reed, 2007; Robbins, Dunlap, & Plienis, 1991), can decrease parental engagement with interventions (Dale, Jahoda, & Knott, 2006), and can negatively affect parenting behaviours and child behaviour problems (Osborne et al., 2007; Rodgers, 1998). In fact, dropout rates from parent training can be high (Matson et al., 2009). Behavioural interventions can be difficult to implement, and previous research has found that early intervention programmes can be associated with either increases (Koegel, 2000) or decreases (Birnbrauer & Leach, 1993) in parental stress. Parent training could be fraught with difficulties for parents. For instance, parents may feel that it reflects negatively on their parenting skills to attend parent training, or may find it upsetting to have to teach skills to their child that other children may acquire incidentally, such as socialisation skills. Therefore, combining stress interventions (e.g., ACT) to help parents emotionally, and behavioural skills training and supports to help parents to manage their child’s behavioural deficits or excesses, is likely to have the most effective outcomes for parents.

Respite Services and Sleep Interventions

Previous research has documented that sleep problems are common in children with ASD (e.g., Humphreys et al., 2013; Norton & Drew, 1994). The present thesis found further evidence that sleep is a significant issue for parents of children with ASD. In Chapter 2, different aspects of child sleep problems and parent-reported sleep dysfunction were found to predict maternal anxiety and CV activity. Furthermore, parents of children with ASD reported significantly higher sleep dysfunction than parents of typically developing children in Chapter 4. Sleep duration has been found to strongly amend human functioning (Pilcher & Huffcutt, 1996) and may have a direct association with mortality (Kripke, Simons, Garfinkel, & Hammond, 1979). However, although research has demonstrated that sleep problems are prevalent among children with ASD, parents do not appear to be receiving adequate supports in this area, given the findings that sleep problems are significantly affecting parental wellbeing. This indicates two areas of need for parents. First, children with ASD should be routinely screened for sleep problems, given the documented prevalence of sleep problems among this population. Screening for sleep problems would enable treatment to be implemented at an early stage, before sleep problems become well-established for the child and parents. Both behavioural interventions and interventions related to the circadian regulation of the sleep-wake cycle (e.g., melatonin) have documented success in improving child sleep habits (Malow et al., 2012; Richdale, 1999). The findings in the present thesis suggest that providing sleep interventions, and supporting parents with the difficulties associated with managing child sleeping problems, could have positive implications for parental wellbeing.

In addition, respite services could be invaluable for such parents. Respite has previously been identified as a necessity for parents of children with ASD (Norton & Drew, 1994). In a survey of parental needs, Dillenburger et al. (2010) found that while 93% of parents rated respite as a future need, only 38% of parents were in receipt of home or respite support. Similarly, in a survey of unmet needs, parents rated doing things they enjoy (91%), a break from caring for their child (87%), respite care (55%), and the child’s sleep pattern (52%) as areas of need that were not being met (Bromley, Hare, Davison, & Emerson, 2004). A number of factors make it difficult for parents to receive informal respite. In Chapter 4, parents of children with ASD reported engaging in more caregiving interactions on the day of the study than parents of typically developing children. Additionally, more parents of children with ASD reported being unemployed than parents of typically developing children, although due to the small sample size in Study 3, these findings may not generalise to the general population of parents of children with ASD. For parents who are unemployed and caring for their child full-time, parental burden and responsibility are likely to be heightened. Parents who are unemployed do not experience the respite associated with employment, and thus may have less of a reprieve from the demands of caregiving (Gray & Holden, 1992). Furthermore, children with ASD had higher behavioural problems than typically developing children in Chapter 4, and behavioural problems associated with ASD may make it more difficult for parents to go on family outings, or to use the services of babysitters (Norton & Drew, 1994). These factors, combined with the high levels of sleep problems experienced by parents of children with ASD in the present research, underline the importance of formal respite services for parents. Previous research has reported an association between higher levels of parenting stress and use of respite care and services (Chan & Sigafoos, 2000; Thomas, Ellis, McLaurin, Daniels, & Morrissey, 2007). Thus, given the heightened stress levels reported by parents of children with ASD in the present study, it is likely that many parents would be willing to use such supports if they were made available to them.

Unmet Service Needs

Availability of services for individuals with ASD was identified as a significant source of stress for parents of children with ASD in the present thesis. In previous studies, parents of children with ASD have reported that their children require services that they are not receiving (e.g., Kohler, 1999). A higher quantity of unmet service needs has been found to be associated with higher levels of maternal burden in mothers of adults with ID (Smith, 1997), and was found to affect relationships between mothers and adolescents with ASD after they finished school (Taylor & Seltzer, 2010). To our knowledge, the present study is the first to identify an association between the quantity of unmet service needs for individuals with ASD and negative physiological outcomes for parents. In Chapter 2, a higher quantity of unmet service needs was predictive of higher maternal depression, distress, and CV activity (i.e., awake and asleep SBP, asleep and 24-hour MAP, and asleep PP). In addition to any potential anger or guilt that parents may experience if their children's needs are not being met, this could also lead to financial burden for parents. In Chapter 4, parents of children with ASD were found to be less likely to be in employment than parents of typically developing children, which indicates that some parents of children with ASD may already be at a financial disadvantage. Furthermore, the cost of raising a child with ASD has been reported to be approximately three times higher than the cost of raising a typically developing child, and this financial burden is increased by the discrepancy between the costs of raising a child with a disability and the services that are received (Dillenburger et al., 2010). Bromley et al. (2004) found that parents rated money (71%) and advice on service availability (79%) as important service needs. Thus, unmet service needs add burden to parents and, as reported in the present thesis, this may have physical implications for parents, such as increased risk of developing hypertension. In a time of global recession in which cutbacks are being made to services (e.g., Autism Ireland, 2013; Baker, 2013; Carbery, 2013; Carty & Stack, 2013), these findings emphasise the importance of considering any long-term effects of such cutbacks to the wellbeing of the parents as well as outcomes for individuals with ASD.

Implications for Future Research

Hypocortisolism

As discussed previously, there is a growing body of literature demonstrating that parents of children with ASD are at risk of hypocortisolism. In the present thesis, the findings in relation to cortisol were not as marked as they have been in the majority of previous research. This may have been due to the small sample size available for the cortisol analyses. However, some evidence of hypocortisolism was observed among mothers and fathers of children with ASD (Chapters 2 and 3). Parents of children with ASD were also found to have lower levels of cortisol than parents of typically developing children, though the differences were not as substantial as expected (Chapter 4). The present research had a relatively young sample of children and adolescents (*M* < 8.5 years in all three studies), while hypocortisolism has also been reported among parents of older adolescents and adults with ASD (Seltzer et al., 2009; Seltzer et al., 2010; Wong et al., 2012). Thus, hypocortisolism among parents of children with ASD appears to be a relatively consistent finding within the literature. However, the mechanisms by which parents are at risk of hypocortisolism have not been investigated. Literature on hypocortisolism suggests that it serves a protective function among individuals who experience chronic stress, lowering chronic HPA-axis activity with the side effect of symptoms such as fatigue and stress sensitivity (Fries et al., 2005). However, genetic and developmental factors have also been hypothesised to contribute to hypocortisolism in some individuals (Heim et al., 2000). Cortisol dysregulation has also been reported in a number of studies of stress in individuals with ASD (e.g., Corbett, Mendoza, Wegelin, Carmean, & Levine, 2008; Corbett, Schupp, Levine, & Mendoza, 2009; Devlin, Healy, Leader, & Hughes, 2011; Marinovic-Curin et al., 2008). Consequently, longitudinal research involving parents of children with ASD and caregivers who are not biological parents would provide interesting insight into the nature of hypocortisolism among caregivers of children with ASD. This would help to shed light on whether hypocortisolism develops in parents of children with ASD as a result of chronic stress, or is already present in parents of children with ASD due to genetic factors. Furthermore, as individuals with PTSD have been reported to experience high levels of sAA in addition to hypocortisolism (Heim et al., 2000), future research should investigate sAA levels in a larger sample of parents of children with ASD to enhance understanding of their SNS activity.

Ambulatory Hemodynamic Activity

Chronic stress is known to be a risk factor for the development of hypertension (e.g., Lovallo, 2005; Morrison & Bennett, 2009; Sapolsky, 2004), which in turn is a risk factor for the development of CV disease (WHF, 2012). However, although parents of children with ASD are known to experience high levels of stress, there is a dearth of research on CV activity in this population to determine the effects, if any, of stress related to parenting a child with ASD on hemodynamic activity. To our knowledge, Gallagher and Whiteley's (2012) study is the only investigation of CV activity among parents of children with ASD to date. They reported that parents of children with DD, including ASD, had higher levels of BP than parents of typically developing children. However, the findings in the present thesis were not consistent with those of Gallagher and Whiteley. In Study 3, there were no differences between the two groups with respect to mean CV activity or variability. The small sample size and potential sampling bias may have been a factor in this finding. There may not have been sufficient statistical power to detect differences between the two groups. However, the sample size in Study 3 ($N = 76$) was comparable to the sample size used in Gallagher and Whiteley's study ($N = 65$). Additionally, in Chapter 3, a high proportion of parents of children with ASD were found to be extreme dippers, but none of the parents in Gallagher and Whiteley's study were identified as extreme dippers, even though the same dipping criteria were used in both studies. Differences may at least partially have been due to the fact that Gallagher and Whiteley's study included parents of children with a range of developmental disabilities, and not exclusively ASD. However, some similarities also emerged. In Chapter 4, parents of children with ASD and typically developing children did not differ with respect to the type of night-time BP dipping, which is consistent with Gallagher and Whiteley's findings. The fact that there is very little research in this area, and the conflicting findings between Gallagher and Whiteley's findings and those of the present thesis, indicate a necessity for further research into the effects of chronic stress on CV activity among parents of children with ASD. Furthermore, given the limited findings in relation to ambulatory CV activity in the present study, it may also be useful for future research to compare cardiovascular reactivity (CVR) to psychological stressors between parents of children with ASD and parents of typically developing children in a controlled laboratory setting.

Disparity between Parent Reports and Physiological Measurements

In the present research, there was a disparity between parental reports and physiological parameters of stress. Because parents of children with ASD reported clinically significant levels of parenting stress, it was expected that there would be evidence of higher BP and HR among parents of children with ASD than parents of typically developing children, which was not the case. Additionally, sAA levels were not significantly higher among parents of children with ASD than among parents of typically developing children. Although there was some evidence of blunted cortisol activity, the difference in cortisol activity between parents of children with ASD and parents of typically developing children was also not as marked as was expected. Thus, although studies incorporating parental reports may be useful in identifying perceived stress and mental health among parents of children with ASD, this research highlights the importance of also including physiological measurements in research investigating stress and health among this population.

Further research would be useful in elucidating the reasons for the disparity between parent-report and physiological measurements in the present study. It is currently unclear if this disparity is a result of differences in perceived stress and activation of the stress-response, limited statistical power due to the relatively small sample size in the present study, or if the present research failed to investigate important physiological markers in parents of children with ASD. Past research has reported poorer antibody responses to vaccination among parents of children with developmental disabilities (e.g., Gallagher et al., 2009). Research by Gallagher et al. (2008) also reported that caregivers (of individuals who were sick or had a disability) had lower secretory immunoglobulin A (SIgA) levels (i.e., an antibody involved in immunity) than non-caregivers. Gallagher et al. (2008) also reported a correlation between reported levels of caregiving burden and levels of SIgA. Additionally, C-reactive protein (CRP), which is a measure of inflammation, is known to increase almost 1000-fold during a stress-response, and has been found to be a stronger predictor of CV events than cholesterol (Ridker, 2003). Given that parents of children with ASD have been found to report poorer health than parents of typically developing children (including in the present study), future research investigating these physiological parameters of stress in addition to the measurements used in the present study, using a larger sample size, may improve understanding of the link between parenting a child with ASD, chronic stress, and health.

Long-Term Effects of Unmet Service Needs

As discussed above, a higher number of service needs being reported by parents of children with ASD was predictive of higher depression, distress, and mean CV activity in Chapter 2. In addition to the present research, previous studies have found associations between higher unmet needs and poorer parental outcomes (e.g., Smith, 1997; Taylor & Seltzer, 2010). However, to our knowledge, there is no longitudinal research to identify the physiological effects on parents of their child's service needs not being met. The present findings indicate that there is a relationship between health measures and the disparity between access to, and need of, services. Longitudinal investigation of the relationship between service needs and access and parental outcomes, particularly with respect to physiological measures, could further knowledge of both the psychological and physiological implications for parents of their child's service needs not being met.

Identifying Effective Screening and Intervention Packages

The findings of the present thesis suggest that a multifaceted approach to stress among parents of children with ASD is warranted. As discussed above, it is likely that a combination of an appropriate stress intervention such as ACT, along with behavioural skills training and supports, would produce optimal outcomes among parents of children with ASD. However, although there is literature demonstrating positive parental outcomes from parent training (e.g., Keen, Couzens, Musprat, & Rodger, 2010) and stress interventions such as ACT (Blackledge & Hayes, 2006), no research has been conducted to identify the most effective overall treatment package for parents of children with ASD. Hastings and Beck (2004) pointed out that stress interventions are likely to improve the efficacy of parent training, but there is insufficient research on outcomes from a two-fold intervention approach for parents of children with ASD. Although such research could be difficult, given that individual needs may vary between parents, component analyses or group comparisons could provide useful information regarding optimal treatment choices for preventing, managing, and reducing stress among parents of children with ASD.

Limitations

Although this study furthers understanding of the effects that caring for a child with ASD can have on parents, there are a number of factors that potentially affect the generalisability of the research.

Small Sample Size

Relatively small sample sizes in the present thesis may have affected statistical power in some of the analyses. Cortisol and sAA data were only available for a subset of the sample in Chapters 2–4, reducing the sample size and statistical power for the salivary analyses. However, obtaining a large sample can be difficult when measuring salivary biomarkers, due to the prohibitive costs of the equipment for collecting and analysing the saliva samples (Adam & Kumari, 2009). Nonetheless, it is still important to be mindful of the reduced sample size for these analyses. Similar research on a larger scale could provide useful information about HPA-axis and SNS activity among parents of children with ASD.

Additionally, there was a relatively small sample size in Chapter 2 for conducting multiple regression analyses. Statistical power could have been increased if there was a larger sample size, or more stringent criteria for selecting variables than the criterion that was used (i.e., $n \geq 20 + 5m$, where m refers to the number of predictor variables; Khamis & Kepler, 2010). However, variables were only included if they were deemed relevant, and less than seven variables were used where possible (e.g., for mean SBP and DBP night-time reduction). Furthermore, due to the difficulty in recruiting mother-father dyads, the sample size in Chapter 3 was somewhat small. However, because fathers are often omitted from parental stress research, it was considered important to include fathers in the present research.

Replication of the present research on a larger scale could prove useful in furthering understanding of the physiological effects of stress on parents of children with ASD.

Reliance on Parent-Reported Compliance

In the present research, it was necessary to rely on parental reports of compliance with saliva collection protocols. Furthermore, sleep was not directly assessed while parents were wearing the ABP monitor. As a result, it is possible that salivary and CV data may have been influenced by noncompliance with data collection guidelines, or by sleep disruption as a result of wearing the ABP monitor. To encourage compliance with saliva collection, efforts were made to make saliva collection easier, in line with previous recommendations (Adam & Kumari, 2009). Written instructions were provided and verbally explained to participants, while saliva collection tubes were provided in a user-friendly saliva collection kit, which was colour coded and labelled. Similarly, the samples were collected from participants to eliminate any difficulty with returning the completed samples. Equipment such as MEMS® TrackCaps would have provided a more direct measure of saliva sample timing compliance, while equipment such as an Actigraph would have been useful to objectively assess waking (Adam & Kumari, 2009). Unfortunately, it was not possible to include such equipment in the present research for financial reasons. However, future research investigating physiological measures of stress in parents of children with ASD should incorporate such equipment if possible.

Sampling Bias

Given the nature of the present research, it is possible that sampling bias may affect the generalisability of the research findings for a number of reasons. First, it is possible that parents who were more highly stressed were more amenable to participating in stress research. Thus, the sample may have been more highly stressed than the general population of parents of children with ASD. Secondly, it is equally possible that the parents of children with ASD included in the present study were *less* stressed than the general population, as many were recruited through ASD support groups. Thus, those parents may have been better at seeking supports and may have had lower levels of stress than other parents of children with ASD as a result. Furthermore, more highly stressed parents may have avoiding participating because they were required to carry out the study over a 24-hour period. Finally, fathers were more difficult to recruit to the present research than mothers. It is a possibility that the fathers who chose to participate were not representative of other fathers of children with ASD. As such, comparisons between mother-father dyads where the fathers were willing to participate may have revealed different results than would have been obtained in families where the fathers are unwilling to participate in research, which could reflect less paternal involvement within the family.

Efforts were made to overcome these issues. Advertising and recruitment materials were distributed through many different mediums, including schools, support groups, and local media, and were aimed at all parents of children with ASD, regardless of the levels of stress they were experiencing. Furthermore, as discussed above, attempts were also made to reduce effort and burden on participants. For example, materials were clearly labelled in a folder, and the researcher travelled to the participants to attach and remove the monitor. However, future research should consider additional methods of reducing effort for parents, in order to encourage their participation in stress research. Finally, the inclusion of a matched control group of mother-father dyads of typically developing children in Chapter 4 provides a certain level of control for sampling bias in relation to fathers. This enabled comparisons between similar parental dyads that had children with or without ASD. However, the potential sampling bias issues should still be considered when interpreting the research results.

Extraneous Variables

In the present research, numerous variables that had the potential to affect parental stress and wellbeing were measured, such as child behaviour problems, sleeping problems, coping strategies, and so on. However, the parent-report measure of stress used was the Parenting Stress Index, which focuses exclusively on stress related to the parenting role (Abidin, 1995). Other life stressors, such as bereavement, job loss, or family or relationship issues, were not measured during the study. As a result, it is possible that extraneous variables could have influenced the research findings by either alleviating or exacerbating parental stress levels among parents of children with or without ASD. The inclusion of a control group in Chapter 4 somewhat controlled for the influence of extraneous variables, as there was equal likelihood that extraneous variables could have influenced parents in either parenting group. Therefore, the present body of research provides valuable information about the experiences of parents of children with ASD but it is possible that factors that were not measured could have influenced the results.

Conclusions

The overarching aim of this research was to identify factors that predicted stress among parents of children with ASD, and to determine the effects, if any, of stress related to parenting a child with ASD on physiological outcomes. Overall, this research has established that (i) parents of children with ASD experience higher levels of stress, anxiety, and depression than parents of typically developing children, (ii) mothers of children with ASD reported significantly higher levels of parenting responsibility, anxiety, depression, and parenting stress than fathers of children with ASD, although they did not differ with respect to mean BP, HR, cortisol, or sAA levels, (iii) parents of children with ASD are at risk of experiencing hypocortisolism, (iv) parents of children with ASD may be at risk of some abnormal CV activity, though not to the extent that would have been expected, and (v) there are several key areas in which intervention may be effective for parents of children with ASD.

The research findings indicate that further supports are needed for parents of children with ASD. A comprehensive approach is needed in order to prevent chronic stress and stress-related health problems for parents. A combination of a stress intervention (such as CBT or ACT) and behavioural skills training and supports is recommended to ensure all parental needs are met. Furthermore, combining these interventions with routine health screening with an emphasis on HPA-axis and ambulatory hemodynamic activity could prevent the development of stress-related health problems, ensuring optimal outcomes for parent of children with ASD. Additionally, respite services and interventions and supports in the areas of sleeping problems, along with ensuring that the service needs of individuals with ASD are met, could have important implications for parental health and wellbeing. Finally, the research findings indicate the importance of providing interventions for both mothers and fathers. Although fathers may experience lower levels of stress than mothers, the relationship between paternal stress and maternal anxiety and depression show that parental outcomes may be interlinked. Thus, better outcomes may be achieved if interventions are made available to both parents.

Areas of future research were identified and discussed and future work is, of course, essential in furthering understanding of the relationship between chronic stress related to parenting a child with ASD and physiological outcomes for parents. The present thesis adds substantially to the existing literature, and further underscores the need for relevant support services being made available for parents of children with ASD and all who face chronic parenting challenges.

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Appendix A

Personal Information and Child Questionnaires

OÉ Gaillimh
NUI Galway

I.D. ( )( )( )
Date ( )( )( )( )( )( )

Questionnaires

The following set of questionnaires will ask you questions about your wellbeing, details about your family and characteristics of your child. You will be given 1-2 months, during which time you may complete the questionnaire in whichever way suits you best. You may complete all questionnaires in one sitting or you may complete in sections, taking breaks as required. Questionnaires regarding your child are presented first, followed by questionnaires about yourself.

If you are uncomfortable with any of the questions presented, you may choose to skip the question.

Child and Family Information Questionnaire

This questionnaire will ask you questions about child and family. Please try to be as honest as possible when answering these questions, and please be assured that the answers you give will be kept confidential. If you are uncomfortable answering any question, you may skip it.

Child Details

Please answer these questions in relation to your child who is the focus of this study

1) What is your child's age? Years _______ Months _________

2) What is your child's sex?

☐ Male
☐ Female

3) Where does your child live?

☐ Home
☐ Residential setting
☐ Supported living
☐ Lives independently
☐ Other (*please specify*): _______________

4) Has your child been diagnosed with an Autism Spectrum Disorder (ASD)?

☐ Yes
☐ No *(please skip to Q.5)*

(i) Which type of ASD has your child been diagnosed with?

☐ Autistic Disorder (Autism)
☐ Asperger's Disorder
☐ Pervasive Developmental Disorder-Not Otherwise Specified

(ii) What age was your child when you first noticed a problem? ____________

(iii) What age was your child when he/she was diagnosed with an ASD? ________

5) Does your child have a diagnosis of an intellectual disability?

☐ Yes
☐ No *(please skip to Q.6)*

If you answered yes, please indicate what severity:

☐ Mild
☐ Moderate
☐ Severe
☐ Profound
☐ Other (*please specify*): ________________

6) Does your child have a physical disability?

☐ Yes
☐ No *(please skip to Q.7)*

If you answered yes, please specify what type: ________________________________

7) Does your child have gastrointestinal (i.e., stomach) problems?

☐ Yes, severe
☐ Yes, moderate
☐ Yes, mild
☐ No gastrointestinal problems

8) Has your child been diagnosed with an Attention Deficit Disorder?

☐ No *(please skip to Q.9)*
☐ Diagnosed with Attention Deficit Disorder (ADD)
☐ Diagnosed with Attention Deficit Hyperactivity Disorder (ADHD)

If your child has been diagnosed with an Attention Deficit Disorder, please indicate the severity:

☐ Mild
☐ Moderate
☐ Severe

9) Does your child have epilepsy?

☐ Yes
☐ No

10) Does your child have any other disability, disorder or illness (e.g., epilepsy, dyspraxia, anxiety disorder, etc.)?

☐ Yes
☐ No

If you answered yes, please specify what type: ________________________________

11 (a) Is your child currently in education?

☐ Yes
☐ No *(Please move on to Q.12)*

(b) Type of education: If your child is **currently** *attending any type of educational placement listed below, please indicate the* **number of hours** *a week your child is currently receiving.* **<u>Please note all that apply</u>.**

	Number of hours per week
Mainstream preschool	
Mainstream primary school	
Mainstream secondary school	
Special Needs School	
ASD Unit (in a Primary school)	

	Number of hours per week
ASD Unit (in a Secondary school)	
ABA (Applied Behaviour Analysis) preschool	
Applied Behaviour Analysis (ABA) school	
Supported by a Special Needs Assistant	
Home tutoring (ABA-based)	
Home tutoring (**not** ABA-based*). Please specify type of tutoring*: ___________________ ___________________	
Other type of educational placement (*please specify*): ____ ___________________	

12) Please read the interventions listed below and select any that apply to your child. Please tick the appropriate box if your child is **currently receiving** *or is not currently receiving but has* **received in the past***.*

	Currently receiving	**Received in the past**
Applied Behaviour Analysis (ABA)		
Speech & Language Therapy		
Occupational Therapy		
Sensory Integration Therapy		
Auditory Integration Therapy		
Play Therapy		
Music Therapy		
Animal Therapy		
Floortime		
Son-Rise Programme		
TEACCH		
Gluten-free, Casein-free (GFCF) Diet		
DAN! Protocol		
Relationship Development Intervention (RDI)		
Tomatis Method		
Neurofeedback		

	Currently receiving	**Received in the past**
Hyperbaric Oxygen Treatment		
Chelation Therapy		
Anti-Psychotic Medication		
Other *(please specify):* ___________________		
Other *(please specify):* ___________________		
Other *(please specify):* ___________________		

13) (a) Please read the services listed below and specify if your child **needs** *or* **does not need** *each service listed.*

(b) If your child **<u>has access</u>** *to the service, please indicate the number of* **<u>monthly hours</u>** *of access your child has to the service.*

	Part A		**Part B**
	Needs service	**Does not need service**	**Hours of access to service**
Occupational Therapy			
Physical Therapy			
Speech & Language Therapy			
Behavioural Therapy (ABA)			
Special Needs Assistant			
Recreational or Social Activities			
Transportation Services			
After School Support			
Respite Services			
Income Support (e.g., teaching budget skills)			
Vocational/Employment Services			
Psychological/Psychiatric Services			
Crisis Intervention Services			
Other *(please specify):*			

Family Details

Please answer the following questions in relation to all children in your family.

14) (a) How many children do you have? __________

(If you only have one child, you do not need to complete 14b)

(b) Please list the age and sex of all your children below, starting with your oldest child. Please include your child who is the focus of this study. When responding to 'diagnosis', please tick yes beside each child who has received any diagnosis of a physical, intellectual, or developmental disability or disorder, or any serious illness. If you tick yes, please specify what type of disability or illness the child has.

☐ **Child 1:** *Age* _____ *Sex:* Male ☐ Female ☐ *Lives at home?* Yes ☐ No ☐

Diagnosis: No ☐ Yes ☐ *Please specify:* ________________________

☐ **Child 2:** *Age* _____ *Sex:* Male ☐ Female ☐ *Lives at home?* Yes ☐ No ☐

Diagnosis: No ☐ Yes ☐ *Please specify:* ________________________

☐ **Child 3:** *Age* _____ *Sex:* Male ☐ Female ☐ *Lives at home?* Yes ☐ No ☐

Diagnosis: No ☐ Yes ☐ *Please specify:* ________________________

☐ **Child 4:** *Age* _____ *Sex:* Male ☐ Female ☐ *Lives at home?* Yes ☐ No ☐

Diagnosis: No ☐ Yes ☐ *Please specify:* ________________________

☐ **Child 5:** *Age* _____ *Sex:* Male ☐ Female ☐ *Lives at home?* Yes ☐ No ☐

Diagnosis: No ☐ Yes ☐ *Please specify:* ________________________

☐ **Child 6:** *Age* _____ *Sex:* Male ☐ Female ☐ *Lives at home?* Yes ☐ No ☐

Diagnosis: No ☐ Yes ☐ *Please specify:* ________________________

If you have more than 6 children, please provide details for any additional children here: ____

____

____

____

Personal and Health Information Questionnaire

This questionnaire will ask you general questions about yourself and some questions about your health. Please try to be as honest as possible when answering these questions, and please be assured that the answers you give will be kept confidential. If you are uncomfortable answering any question, you may leave it blank.

Your Personal Details

1) Your Age: _________

2) *Your sex*:

☐ Male
☐ Female

3(a) Are you currently in paid employment?

☐ No
☐ Yes, part-time
☐ Yes, full-time

4) What is your relationship to your child?

☐ Biological parent
☐ Step-parent
☐ Adoptive parent
☐ Foster parent
☐ Other *(please specify):* _________________

5) (a) Please read the supports listed below and specify if you **need** *or* **do not need** *each support listed.*
(b) If you have access to the support, please indicate the number of **weekly hours** *of access to the support that you have.*

	Part A		**Part B**
	Need	**Do not need**	**Hours of access to support**
Housekeeping support from a paid housekeeper			
Housekeeping support from a partner, family or friends			
Caregiving support from a paid child-minder			
Caregiving support from a partner, family or friends			
Caregiving support from a service or organisation (unpaid)			
Caregiving support from a service or organisation (paid)			
Emotional support			

Health Details

We need to obtain some health and lifestyle information to ensure that the blood pressure data and saliva samples are analysed correctly. Only information that may impact on blood pressure, salivary biomarkers, and stress will be requested. All information is confidential.

12 (a) What is your weight? _________

(b) What is your height? _________

(c) Do you have any oral diseases or dental problems?

☐ *Yes (please specify):* ____
☐ *No*

13 (a) Which of the following describes you best:

☐ Smoker
☐ Non-smoker
☐ Ex-smoker

(b) If you are a smoker, what number of cigarettes do you smoke in a week? _________

14 (a) Which of the following best describes your typical daily activity levels?

☐ Mainly sitting
☐ Occasionally sitting but mainly standing
☐ Mainly standing or being active
☐ Mainly engaging in strenuous activity or heavy labour

*(b) How much time (in minutes) do you spend exercising on average each week?*_______

(c) Please circle the number that best describes your fitness level. 1 = very unfit, 6 = very fit

1 2 3 4 5 6

15 (a) How often do you drink alcohol?

☐ Never
☐ Occasionally
☐ Fortnightly
☐ Weekly
☐ More than once a week
☐ Daily

(b) If you drink alcohol, how many units on average do you consume in one sitting? _______

16) How often do you drink caffeine?

☐ Several times a day
☐ Once a day
☐ Several times a week
☐ Once a week
☐ Less than once a week
☐ Never

17) Please indicate if you *<u>currently</u>* *or in the* *<u>past</u>* *have had any of the following conditions, or if there is a* *<u>family history</u>* *of the condition. Please tick all responses that apply.*

	Currently have	**Had in the past**	**There is a family history**
High blood pressure			
Low blood pressure			
Heart attack			
Other cardiovascular disease			
Psychiatric disorder			
Eating disorder			
Depression			
Alcohol dependency			
Drug dependency			
Cancer			
Stroke			
Endocrine disorder			
Stomach ulcer			
Irritable bowel syndrome			
Insomnia			
Anxiety or panic attacks			
Circulatory problems			
Diabetes			
Fatigue			
Chronic Fatigue Syndrome			
Chronic headaches or migraines			
Fibromyalgia			
Other *(please specify):* _________ __________________			
Other *(please specify):* _________ __________________			

18) Are you currently taking any of the following prescription medications? (Please tick any that apply).

☐ Oral contraceptive pill
☐ Antihypertensive medication
☐ Steroids
☐ Antibiotics
☐ Antidepressants
☐ Other *(Please specify):* ____
☐ None

19) How often do you take over-the-counter medication? (e.g., Panadol)

- ☐ Never
- ☐ Occasionally
- ☐ Fortnightly
- ☐ Weekly
- ☐ More than once a week
- ☐ Daily

20) Have you recently lost a lot of weight?

- ☐ No
- ☐ Yes *(Please specify): How much? ____ Over what time period? ____*

21) Are you currently attending psychotherapy or any other form of counselling?

- ☐ Yes
- ☐ No

22) To be answered by women only: Please indicate if any of the following apply to you:

- ☐ Currently pregnant *(Please indicate what trimester):*1st ____ 2nd ____ 3rd ___
- ☐ Currently experiencing perimenopause (period of change leading into menopause)
- ☐ Postmenopausal *(Please indicate number of years since you reached menopause): ____________________*

Appendix B
Saliva Collection Guidelines

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NUI Galway

<u>How to provide the samples:</u>

1) Please do not drink alcohol for 24 hours before collecting saliva.
2) Please wait 48 hours after dental work before collecting saliva.
3) Try not to eat a major meal, drink (except water), smoke, or exercise for 1 hour before each collection. You may drink water at any time.
4) Please avoid dairy, sugary foods and caffeine for 20 minutes before collection.
5) Please do not brush your teeth for 45 minutes before collection.
6) Please rinse your mouth with water 10-15 minutes before collecting the afternoon sample.
7) Please be honest when reporting the time of the saliva collections and how much you have smoked, exercised, eaten and drank when asked in the diary.

<u>The samples:</u>

1) ***"CAR 1"***:	Please provide this sample **<u>immediately</u>** after waking. (It will help to leave the SOS tubes next to your bed.)
2) *"**CAR 2**"*:	Please provide this sample 15 minutes after waking.
3) *"**CAR 3**"*:	Please provide this sample 30 minutes after waking.
4) *"**CAR 4**"*:	Please provide this sample 45 minutes after waking.
5) *"**12pm**"*:	Please provide this sample at 12pm (i.e., 12 noon). Please rinse your mouth with water 10-15 minutes beforehand).

<u>How to collect the samples:</u>

1) See guidelines above for things to avoid prior to collecting the sample.
2) For sample 5, please rinse your mouth with water 10-15 minutes before collecting the sample (e.g., rinse your mouth with water at 11.45 and collect the sample at 12:00)
3) Please ensure your hands are clean when you are collecting a sample and handle the wadding as little as possible.
4) Take the cap off the appropriate tube and remove the white wadding.
5) Place the wadding under your tongue for <u>two minutes</u>. The wadding should not be sticking out from under your tongue. <u>Please do not chew the wadding</u>.
6) After 2 minutes, place the wadding back into the tube and replace the cap.
7) Place the tube into the ziplock bag provided, and put into your freezer as soon as possible after collecting each sample.
   <u>NB</u>: If you do not have access to a freezer straight after collection, samples should be placed into a refrigerator no later than 30 minutes after collection. Samples should be moved to a freezer no later than 4 hours after collection. All samples should remain frozen until they are collected.
8) Please complete the appropriate diary entry after each saliva collection, and please be as honest as possible. If, for example, you have eaten or brushed your teeth before collecting a sample, please report this on the diary.

*If you have any questions, please contact Ciara on 087********

Appendix C

Daily Diary

I.D. ( ) ( ) ( )
Date ( ) ( ) ( ) ( ) ( ) ( )

Daily Diary

This diary will help us to pinpoint specific stressful events in your daily life. It will also help us to identify other factors that may influence your blood pressure and stress hormones throughout the day (e.g., exercise). Please complete an entry each time the cuff inflates on the blood pressure monitor while you are awake, if possible.

Morning diary:	Please complete on the morning of data collection after you have collected your morning saliva samples.
Saliva diary:	Please complete at 12pm after you collect your saliva sample.
Cuff inflation diary:	Please complete each time the cuff inflates **DURING WAKING HOURS.**
Summary diary:	This diary will be provided when your blood pressure monitor is being removed. Please answer based on the previous 24 hours.

Please try to be as accurate as possible. Thank you for your time.

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Morning Diary

Please complete this diary entry when you collect the morning saliva samples.

Exactly 15 mins between each

Tube	Time Taken	Tube label
1 (Upon waking)		
2 (+ 15 min)		
3 (+30 min)		
4 (+45 min)		

1) What time did you wake up? ____________

2) How many hours did you sleep last night? _________________

3) How much stress do you think you are going to experience today?

- ☐ None at all
- ☐ A little amount
- ☐ A medium amount
- ☐ A lot

4) Did you brush your teeth between waking and collecting the final morning saliva sample?

- ☐ Yes
- ☐ No

5) Please tick if you did any of the following between waking and collecting the final sample:

- ☐ Drank alcohol
- ☐ Drank caffeine
- ☐ Drank other flavoured drink
- ☐ Drank water
- ☐ Smoked or taken any nicotine substance such as Nicorette
- ☐ Ate a meal *(If you tick, please answer Q. 6)*
- ☐ Exercised *(If you tick, please answer Q. 7)*

6) If you ate a meal in the past hour, please indicate what you ate: _____________

____

7) If you exercised, please indicate:

(a) the duration: __________________

(b) the intensity:

- ☐ Light (not sweating, able to talk)
- ☐ Moderate (sweating after 10 minutes, able to talk)
- ☐ High (sweating after less than 5 minutes, only able to talk briefly)

Please Turn Over

8) In the past 24 hours, have you taken any of the following medications?

- ☐ Over-the-counter medication (e.g., Panadol)
- ☐ Steroids
- ☐ Antibiotics
- ☐ Oral contraceptive pill
- ☐ Antidepressants
- ☐ Antihypertensive (i.e., high blood pressure) medication
- ☐ Other (*please specify):*

  ____

*9) For **each of the following,** please rate how you currently feel.*

1 = not at all, 4 = very much.

(i) Sad:	1	2	3	4
(ii) Active:	1	2	3	4
(iii) Interested:	1	2	3	4
(iv) Stressed:	1	2	3	4
(v) Upset:	1	2	3	4
(vi) Excited:	1	2	3	4
(vii) Frustrated:	1	2	3	4
(viii) Alert:	1	2	3	4

Behaviour Incidents Checklist

Please answer based on your child's behaviour over the past 24 hours.

1) Did your child hurt himself or herself?

- ☐ Yes
- ☐ No

2) Was your child destructive or hurtful to others?

- ☐ Yes
- ☐ No

3) Was your child destructive to property?

- ☐ Yes
- ☐ No

4)Did your child show any unusual or repetitive behaviour?

- ☐ Yes
- ☐ No

Please Turn Over

5) Did your child show any socially inappropriate behaviour?

- ☐ Yes
- ☐ No

6) Was your child withdrawn or inattentive today?

- ☐ Yes
- ☐ No

7) Was your child uncooperative?

- ☐ Yes
- ☐ No

Thank you for your time.

OÉ Gaillimh
NUI Galway

Saliva Collection Diary Entry

<u>12pm</u> Saliva Sample

1) Time sample taken: ______________

2) When did you last brush your teeth?

- ☐ More than 45 minutes ago
- ☐ Less than 45 minutes ago

3) If you have done any of the following in the past hour, please tick:

- ☐ Drank alcohol
- ☐ Drank caffeine
- ☐ Drank other flavoured drink
- ☐ Drank water
- ☐ Smoked or taken any nicotine substance such as Nicorette
- ☐ Ate a meal *(If you tick, please answer Q. 4)*
- ☐ Exercised *(If you tick, please answer Q. 5)*

4) If you ate a meal in the past hour, please indicate what you ate: ______________
____

5) If you exercised in the past hour, please indicate:
(a) the duration: __________________
(b) the intensity:

- ☐ Light (not sweating, able to talk)
- ☐ Moderate (sweating after 10 minutes, able to talk)
- ☐ High (sweating after less than 5 minutes, only able to talk briefly)

This diary entry is now complete. Thank you.

OÉ Gaillimh
NUI Galway

Cuff Inflation Diary Entry

Please complete an entry each time the cuff inflates during waking hours.

1) Time of cuff inflation: _______________

2) Location:

- ☐ Home
- ☐ Work
- ☐ Car
- ☐ Public place
- ☐ Other *(Please specify):* ______________________

3) If you have been doing any of the following since the last cuff inflation, please tick:

- ☐ Engaging in a conversation or social interaction
- ☐ Engaging in caregiving activities
- ☐ Engaging in work activities

*4) For **each of the following,** please rate how you feel.* 1 = not at all, 4 = very much.

(i) Sad:	1	2	3	4
(ii) Active:	1	2	3	4
(iii) Interested:	1	2	3	4
(iv) Stressed:	1	2	3	4
(v) Upset:	1	2	3	4
(vi) Excited:	1	2	3	4
(vii) Frustrated:	1	2	3	4
(viii) Alert:	1	2	3	4

OÉ Gaillimh
NUI Galway

Summary Morning Diary Entry

Please complete this diary entry when the blood pressure monitor is removed, based on the previous 24 hours

1) What time did you go to sleep last night? ____________

2) What time did you wake up this morning? ________________

3) How many hours did you sleep last night? _________________

4) Please indicate how much time you slept yesterday (during the day): ________

5) Would you consider yesterday to be a normal day in terms of activities and responsibilities (e.g., work, caregiving) that you typically have to manage from day-to-day? __________

6) Please indicate how many caffeine drinks you had: _____________

7) Please indicate how many cigarettes you smoked: ______________

8) Please indicate how many times you used other nicotine substances (e.g., Nicorette): ______________

9) Please indicate how many units of alcohol you drank: ___________

10) Please indicate if you took any of the following medications since waking yesterday. Please tick any that apply.

- ☐ Over-the-counter medication (e.g., Panadol)
- ☐ Steroids
- ☐ Antibiotics
- ☐ Oral contraceptive pill
- ☐ Antidepressants
- ☐ Antihypertensive (i.e., high blood pressure) medication
- ☐ Other *(please specify):* __________________________

11 (a) Did you exercise since waking yesterday:

- ☐ Yes
- ☐ No

(b) If you answered yes, please indicate:

(i) the duration: ________________________

(ii) the intensity:

- ☐ Light (not sweating, able to talk)
- ☐ Moderate (sweating after 10 minutes, able to talk)
- ☐ High (sweating after less than 5 minutes, only able to talk briefly)

Please Turn Over

12) Overall, how stressful was your day since you woke yesterday. Please rate by circling the appropriate number. 1 = not at all stressful, 4 = very stressful.

1 2 3 4

13) Please indicate if you had any of the following support since waking yesterday:

- ☐ Housekeeping assistance from a paid housekeeper
- ☐ Housekeeping assistance from family, friends or partner
- ☐ Caregiving support from a paid child minder
- ☐ Caregiving support from your partner
- ☐ Caregiving support from family or friends
- ☐ Caregiving support or tuition from an unpaid organisation or service (e.g., afterschool or respite services)
- ☐ Caregiving support or tuition from a paid organisation or service (e.g., a paid tutor)
- ☐ Emotional support (e.g., from a friend or spouse)
- ☐ Other *(please specify):* ______________________________

Behaviour Incidents Checklist

Please answer based on your child's behaviour over the past 24 hours.

1) Did your child hurt himself or herself?

- ☐ Yes
- ☐ No

2) Was your child destructive or hurtful to others?

- ☐ Yes
- ☐ No

3) Was your child destructive to property?

- ☐ Yes
- ☐ No

4)Did your child show any unusual or repetitive behaviour?

- ☐ Yes
- ☐ No

5)Did your child show any socially offensive behaviour?

- ☐ Yes
- ☐ No

6) Was your child withdrawn or inattentive?

- ☐ Yes
- ☐ No

7) Was your child uncooperative?

- ☐ Yes
- ☐ No

Appendix D

Consent Form

OÉ Gaillimh
NUI Galway

Study Number:
Participant Identification Number:

Title of project: Psychophysiological Measures of Stress in Caregivers of Children with Autism Spectrum Disorders

Researcher: Ciara Foody

Please initial box

1. I confirm that I have read the attached information sheet for the above study and have had the opportunity to ask questions. ☐
2. I am satisfied that I understand the information provided and have had enough time to consider the information. ☐
3. I am satisfied that I understand the 'confidentiality' information provided. ☐
4. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my legal rights being affected. ☐
5. I agree to take part in the above study. ☐

Name of Participant	Date	Signature
____________________	_______________	______________________
Name of Person Taking Consent (if different from researcher)	Date	Signature
____________________	_______________	______________________
Researcher	Date	Signature
____________________	_______________	______________________

Appendix E

Summary of Significant Correlations from Study 1 (Chapter 2)

Table E1

Summary of Pearson's R Correlations between Perceived Anxiety, Depression, and Parenting Stress, and Child Variables

Variable	HADS-A	HADS-D	PSI-Total	PSI-PCDI	PSI-DC	PSI-PD
Number of children with a diagnosis		.244*				
Interventions tried		-.275*				
Conners:						
Oppositional	.222*	.289**	.576**	.419**	.567**	.393**
Inattention		.246*	.533**	.448**	.376**	.451**
Hyperactivity			.541**	.506**	.465**	.332**
ADHD index		.246*	.561**	.440**	.476**	.426**
CSHQ:						
Bedtime resistance			.212*	.212*		
Sleep duration			.267*	.341**		
Parasomnias	.280**	.230**				
Daytime sleepiness			.286**	.294**	.230*	
CSHQ total score			.355**	.343**	.275*	.236*
GARS:						
Stereotypy	.241*		.360**	.339**	.331**	.201*
Social interaction			.492**	.556**	.396**	.244*
Autism index			.384**	.394**	.333**	.203*
BPI:						
SIB frequency					.272**	
SIB severity					.250*	
Stereotypy frequency	.206*		.258*		.273**	
Stereotypy severity	.222*		.320**	.238*	.304*	.227*
Aggression frequency			.342**	.263*	.497**	
Aggression severity			.354**	.247*	.484**	
Behaviour Problems:						
Daily frequency-Day 1			.273*		.316**	.216*

Table E1, Continued

Summary of Pearson's R Correlations between Perceived Anxiety, Depression, and Parenting Stress, and Child Variables

Variable	HADS-A	HADS-D	PSI-Total	PSI-PCDI	PSI-DC	PSI-PD
Vineland:						
Receptive subdomain			-.315**	-.414**	-.204*	
Expressive subdomain	.216*	.241*		-.247*		
Written subdomain		.206*				
Communication domain				-.300**		
Personal subdomain			-.237*	-.304**	-.234*	
Domestic subdomain			-.374**	-.418**	-.370**	
Community subdomain			-.260*	-.350**	-.245*	
Daily living domain			-.362**	-.431**	-.358**	
Interpersonal subdomain			-.345**	-.474**	-.322**	
Play subdomain			-.329**	-.379**	-.321**	
Coping subdomain			-.300**	-.378**	-.302**	
Socialisation domain			-.368**	-.464**	-.362**	
Gross motor subdomain				-.342*		
Fine motor subdomain			-.418**	-.387*	-.404**	
Motor domain			-.354*	-.373*	-.309*	
Composite score			-.334**	-.443**	-.322*	

Table E2

Summary of Pearson's R Correlations between Perceived Anxiety, Depression, and Parenting Stress, and Parent Variables

Variable	HADS-A	HADS-D	PSI-Total	PSI-PCDI	PSI-DC	PSI-PD
Unmet service needs		.298**	.289**	.210*		.299**
Social support quantity		-.306**				
Coping Styles (COPE):						
Denial	.334**	.327**				.303**
Substance use	.272**					
Emotional support		-.275**				
Instrumental support				.239*	.223*	
Behavioural disengagement	.391**	.248**	.388**	.258*	.242*	.413**
Planning						.224*
Humour		-.210*				
Religion					-.239*	
Self-blame	.531**	.536**	.320**			.513**
PSQI:						
C1: Subjective sleep quality	.357**	.412**				.350**
C2: Sleep latency	.265*	.201*	.371**	.281**	.287**	.317**
C3: Sleep duration	.262*	.310**	.311**	.309**		.314**
C4: Habitual sleep efficiency	.359**	.257*				.255*
C5: Sleep disturbances	.321**	.341**	.208*			.235*
C6: Sleeping medication			.200*		.196*	
Global PSQI score	.489**	.458**	.416**	.245**	.249*	.485**
Health-Relevant Variables						
Participant age	-.278*					
BMI	.254*	.380**	.357**			.484**
Time spent exercising			.246*	.316**		
Units of alcohol consumed	.358**					
Current illnesses quantity	.407**	.417**	.445**	.314**	.420**	.443**
Past illnesses quantity	.310**	.264**	.325**	.248**	.347**	.265**
Family history of illnesses quantity	.163*		.239**	.197*	.283**	.153*
Alcohol consumed						-.347**

Table E3

Summary of Spearman's Rho Correlations between Perceived Anxiety, Depression, and Parenting Stress, and Child, Parent, and Health-Relevant Variables

Variable	HADS-A	HADS-D	PSI-Total	PSI-PCDI	PSI-DC	PSI-PD
Diagnoses:						
ID severity		-.318**				
GI issues severity	.198*					
ADD/ADHD severity			.250*			.238*
Epilepsy		-.198*				
Health-Relevant Variables						
Employment status	-.195*	-.237*	-.212*		-.192*	
Smoking status					.229*	
Daily activity levels	-.224*	-.203*	-.226*			-.273**
Fitness level	-.304**	-.268*				-.233*
Psychotherapy			.234*			
Use of antidepressants	.346**	.324**	.270*			.322**
Medication Usage						
Frequency of OTC medication			.239*			.321**
Anti-hypertensive medication	.278*	.240*				
Use of antidepressants	.326**	.394**	.305**			.388**
Illnesses						
Hypertension family history		.222*	.241*	.207*		.294**
Current low BP		-.244**				-.333**
Current heart attack			.233*	.258*	.228*	
CV disease family history					.259*	
Psychiatric disorder family history					.297**	
Current eating disorder	.201*	.221*	.296**	.252*	.261*	.231*
Past eating disorder		.280**	.215*		.193*	.233*
Current depression	.348**	.311**	.240*			.377**
Past depression	.262*	.313**				.234*
Past alcohol dependency			.193*	.196*		
Alcohol dependency family history					.225*	

Table E3, Continued

Summary of Spearman's Rho Correlations between Perceived Anxiety, Depression, and Parenting Stress, and Child, Parent, and Health-Relevant Variables

Variable	HADS-A	HADS-D	PSI-Total	PSI-PCDI	PSI-DC	PSI-PD
Past drug dependency			.193*	.196*		
Drug dependency family history					.219*	
Stroke family history			.204*		.249*	
Current endocrine disorder		.284**				
Past endocrine disorder				-.199*	-.194*	
Endocrine disorder family history					.203*	
Past stomach ulcer			.234*		.290**	
Current insomnia	.260*			.255*		.230*
Past insomnia				-.196*		
Current anxiety	.380**	.305**				.273**
Past anxiety	.290**		.252*	.213*	.229*	
Current fatigue					.200*	
Chronic Fatigue Syndrome (CFS) family history					.248*	
Current chronic headaches						.209*
Fibromyalgia family history					.252*	

Table E4

Summary of Pearson's R Correlations between Cardiovascular Measures and Child Variables

Variable	SBP Awake Mean	SBP Asleep Mean	SBP 24hr Mean	SBP Dip (%)	DBP Awake Mean	DBP Asleep Mean	DBP 24hr Mean	DBP Dip (%)	HR Awake Mean	HR Asleep Mean	HR 24hr Mean	MAP Awake Mean	MAP Asleep Mean	MAP 24hr Mean	PP Awake Mean	PP Asleep Mean	PP 24hr Mean
Vineland:																	
Gross motor subdomain															.433*		.403*
Behaviour Problems:																	
Daily frequency-Day 1	.244*									.249*		.267*		.243*			
Conners:																	
Inattention																.278*	
CSHQ:																	
Bedtime resistance										.268*							
Night wakings				-.239*													
Disordered breathing																	-.271*
Daytime sleepiness	-.274*	-.270*	-.267*												-.293*	-.285*	-.335**
Overall sleep problems															-.239*		-.251*
Diagnosis:																	
Age problem first noticed		.299*				.313*		-.315*					.352**	.254*			
Interventions:																	
Current quantity						-.261*							-.256*				
Tried quantity		-.251*											-.243*				
Education duration			-.308*											-.300*			

Table E5

Summary of Pearson's R Correlations between Cardiovascular Measures and Parent Variables

Variable	SBP Awake Mean	SBP Asleep Mean	SBP 24hr Mean	SBP Dip (%)	DBP Awake Mean	DBP Asleep Mean	DBP 24hr Mean	DBP Dip (%)	HR Awake Mean	HR Asleep Mean	HR 24hr Mean	MAP Awake Mean	MAP Asleep Mean	MAP 24hr Mean	PP Awake Mean	PP Asleep Mean	PP 24hr Mean
Depression (HADS-D)	.236*	.292*											.300*			.325**	
Anxiety (HADS-A)	.236*	.359**	.307*			.264*							.358**	.313*		.349**	.258*
Defensive responding (PSI)	.244*	.303*	.254*									.242*	.295*	.266*		.280*	
Parental distress	.277*	.332**	.290*									.264*	.325**	.285*		.354**	
Parental responsibility																	.285*
Unmet support needs			.317*													.400**	.288*
Unmet service needs	.380**	.392**	.412**									.370**	.358**	.395**	.325**	.499**	.356**
COPE:																	
Emotional support						-.246*											
Behavioural disengagement	.277*	.322**	.328**			.255*	.259*					.275*	.323**	.358**			
PSQI:																	
Subjective sleep quality	.285**	.344**	.285**								.237*	.254*	.325**	.255*	.271*	.461**	.284**
Sleep latency											.204*						
Sleep duration	.243*	.313**	.295**	-.254*			.208*				.233*	.227*	.310**	.291**	.214*	.383**	.249*
Habitual sleep efficiency	.264*	.279*	.299**									.261*	.299**	.295**	.206*	.283**	.241*
Sleep disturbances		.219*											.229*			.308**	
Sleeping medication									.196*	.206*							
Global score	.300**	.375**	.367**	-.206*		.237*	.217*				.257*	.280**	.385**	.353**	.277**	.416**	.323**

Table E6

Summary of Pearson's R Correlations between Cardiovascular Measures and Health-Relevant Variables

Variable	SBP Awake Mean	SBP Asleep Mean	SBP 24hr Mean	SBP Dip (%)	DBP Awake Mean	DBP Asleep Mean	DBP 24hr Mean	DBP Dip (%)	HR Awake Mean	HR Asleep Mean	HR 24hr Mean	MAP Awake Mean	MAP Asleep Mean	MAP 24hr Mean	PP Awake Mean	PP Asleep Mean	PP 24hr Mean
Participant age									-.333**	-.223*	-.354**						
BMI		.271*		-.361**								.243*			.317**		
Quantity of cigarettes										-.208*	.204*						
Recent weight loss				.273*													
Weight loss duration				.295**													
Time since menopause				-.207*				-.274*									
Illnesses:																	
Past illnesses quantity		.272*				.203*							.238*	.203*			
Family history quantity									.194*		.216*						-.207*
Health-Relevant Variables from Summary Diary:																	
Caffeine consumed	.339**	.277**	.332**		.252*	.256*	.242*					.335**	.306**	.313**	.234*	.234*	.250*
Cigarettes smoked									.331**	.272*	.321**						
Alcohol consumed															-.245*		-.254*
Exercise duration		-.218*														-.211*	

Table E7

Summary of Spearman's Rho Correlations between Cardiovascular Measures and Child and Health-Relevant Variables

Variable	SBP Awake Mean	SBP Asleep Mean	SBP 24hr Mean	SBP Dip (%)	DBP Awake Mean	DBP Asleep Mean	DBP 24hr Mean	DBP Dip (%)	HR Awake Mean	HR Asleep Mean	HR 24hr Mean	MAP Awake Mean	MAP Asleep Mean	MAP 24hr Mean	PP Awake Mean	PP Asleep Mean	PP 24hr Mean
Diagnoses:																	
ADD/ADHD severity		.282*														.291*	
Health-Relevant Variables																	
Smoking status						.206*				.311**							
Daily activity levels	-.215*		-.262*											-.240*			
Fitness level	-.228*		-.243*											-.210*	-.210*		
Caffeine frequency	.303**	.300**	.369**		.254*	.211*	.295**					.318**	.288**	.362**	.218*	.362**	.282*
Psychotherapy																	
Menopause		.253*	.205*	-.222*				-.222*					.249*			.277*	.222*
Health-Relevant Variables from Summary Diary:																	
Oral contraceptive pill													.203*				
Antidepressants	.230*	.217*	.266*										.239*	.226*	.224*	.283*	.270*
Antihypertensive medication						.211*											
Predicted stress	.267**	.276*	.283**		.203*	.217*	.240*					.252*	.266*	.287**		.311**	
Stress experienced	.294**		.322**				.254*					.251*	.232*	.298**	.284**		.295**

Table E8

Summary of Spearman's Rho Correlations between Cardiovascular Measures and Illnesses

Variable	SBP Awake Mean	SBP Asleep Mean	SBP 24hr Mean	SBP Dip (%)	DBP Awake Mean	DBP Asleep Mean	DBP 24hr Mean	DBP Dip (%)	HR Awake Mean	HR Asleep Mean	HR 24hr Mean	MAP Awake Mean	MAP Asleep Mean	MAP 24hr Mean	PP Awake Mean	PP Asleep Mean	PP 24hr Mean
Current hypertension	.339**	.241*	.310**		.206*		.212*					.323**	.228*	.279*	.301**	.333**	.304**
Past hypertension				-.230*	.210*	.310**	.244*	-.284*	.227*		.212*	.204*	.287**	.222*			
Current low BP	-.363**	-.402**	-.388**		-.325**	-.416**	-.377**			-.214*		-.340**	-.420**	-.393**	-.228*	-.292**	-.223*
CV disease family history									.212*								
Psychiatric disorder family history	-.207*														-.217*		-.203*
Past eating disorder									.217*		.235*						
Eating disorder family history		-.218*		.217*													
Current depression	.256*	.258*	.274*										.246*	.216**	.296**	.314**	.300**
Past depression										.204*							
Depression family history	-.249*	-.225*	-.272*									-.201*			-.256*	-.318**	-.298**
Past cancer								-.212*									
Cancer family history				-.269*				-.224*									
Endocrine disorder family history									.229*		.313**						.220*
Current stomach ulcer										-.204*							
Stomach ulcer family history	-.246*		-.240*		-.259*							-.236*		-.231*		-.289**	
Current IBS															-.234*		-.205*
IBS family history									.286**		.304**						

Table E8, Continued

Summary of Spearman's Rho Correlations between Cardiovascular Measures and Illnesses

Variable	SBP Awake Mean	SBP Asleep Mean	SBP 24hr Mean	SBP Dip (%)	DBP Awake Mean	DBP Asleep Mean	DBP 24hr Mean	DBP Dip (%)	HR Awake Mean	HR Asleep Mean	HR 24hr Mean	MAP Awake Mean	MAP Asleep Mean	MAP 24hr Mean	PP Awake Mean	PP Asleep Mean	PP 24hr Mean
Current insomnia																..235*	
Insomnia family history	-.198*														-.216*		
Current anxiety																.260*	
Anxiety family history	-.213*														-.226*	-.228*	-.240*
Current circulatory problems									-.238*	-.295**	-.211*						
Circulatory problems family history											.216*						
Current diabetes	.223*		.235*				.264*		.291**	.395**	.405**	.231*	.227*	.273*			
Diabetes family history									.294**		.264*					-.207*	-.218*
Fatigue family history	-.235*	-.220*			-.221*							-.230*			-.203*	-.218*	-.218*
Current CFS										-.204*							
CFS family history											.208*						
Current chronic headache				.225*													
Past chronic headache												-.197*					
Current fibromyalgia	-.216*														-.265*		-.250*
Past fibromyalgia																.248*	
Fibromyalgia family history									.263*		.265*						

Table E9

Summary of Pearson's R Correlations between Cortisol and Child and Health-Relevant Variables

Variable	Cortisol AUC_G	Cortisol AUC_I
GARS:		
Communication	-.386*	
Autism index	-.323*	
Vineland:		
Fine motor subdomain		-.516*
BPI:		
SIB frequency	-.427**	
SIB severity	-.397**	
CSHQ:		
Sleep onset delay	-.307*	
Night wakings	-.313*	
Parasomnias	-.441**	
Overall child sleep problems	-.369*	
Unmet service needs		.269*
Health-Relevant Variables		
Participant age		.356*
BMI		
Quantity of current illnesses	-.418**	
Quantity of past illnesses		.433**

Table E10

Summary of Spearman's Rho Correlations between Cortisol and Health-Relevant Variables

Variable	Cortisol AUC_G	Cortisol AUC_I
Oral health	.302*	
Caffeine frequency		.371**
Illnesses:		
Past low blood pressure		.393*
Low blood pressure family history	-.264*	
Past CV disease	-.267*	
Psychiatric disorder family history		.329*
Eating disorder family history	-.274*	
Current depression	-.354*	
Past cancer	-.267*	
Current endocrine disorder	-.373**	
Past endocrine disorder		.267*
Endocrine disorder family history		.345*
Past stomach ulcer		.416**
Past IBS		.309*
Past anxiety		.290*
Current diabetes		-.269*
Current fatigue	-.432**	
Past fatigue		.372**
Current chronic headaches	-.299*	
Chronic headaches family history		.326*

Table E11

Summary of Pearson's R and Spearman's Rho Correlations between sAA and Health-Relevant Variables

Variable	Alpha-Amylase
Pearson's R Correlations	
Participant age	.269*
BMI	.250*
Average exercise duration	.251*
Cigarettes smoked	-.243*
Spearman's Rho Correlations	
Menopause	.272*
Alcohol dependency family history	.300*
Current anxiety	.257*
Use of antibiotics	.271*
Alcohol consumed	-.184*
Smoking	-.183*