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Stress, Coping, Emotional Intelligence, Emotional Health and Cortisol in Premature Infants, at Age 23

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STRESS, COPING, EMOTIONAL INTELLIGENCE,
EMOTIONAL HEALTH AND CORTISOL IN
PREMATURE INFANTS, AT AGE 23

BY

JOYCE ELLEN HICKEY

A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF THE
REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

IN

NURSING

UNIVERSITY OF RHODE ISLAND

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DOCTOR OF PHILOSOPHY DISSERTATION

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UNIVERSITY OF RHODE ISLAND
2017

Abstract

Stress, Coping, Emotional Intelligence, Emotional Health and Cortisol

In Premature Infants, at Age 23

Little is known about the psychobiological processes of adults, at age 23 who were born prematurely. Compared to infants born at term, premature infants face additional obstacles of immature body systems, more neonatal stress and are at risk for developmental delay and possible parental overprotective patterns. The Developmental Origins of Health and Disease theory asserts that early prenatal and neonatal stress disturbs hypothalamic-pituitary-adrenal (HPA) axis function, which in the long run, affects later health and behavior. The purpose of this secondary analysis is to examine neonatal stress of prematurity on coping, emotional intelligence, physiological responses, developmental milestones and emotional health at age 23 years in a sample at risk for HPA dysfunction.

Prematurely born age 23 year olds, categorized into four groups of medical risk were compared to a term-born group on measures of: Daily Hassles Stress, Coping Response Inventory-Adult, Bar-On EQi, Adult Self Report and salivary cortisol responses in the Trier Social Stress Test. Significant differences in cortisol slope, between the term and premature medical risk group with high stress coupled with lower and upper socioeconomic status, were found as well as differences in total cortisol AUCg levels between premature groups. Significant differences in cortisol slope values were found for all 23 year olds with Adult Self Report psychological clinical problems. This study supports the HPA Axis stress response as a biomarker of premature birth and of psychological clinical problems. Preventative and clinical

interventions for young adults to reduce and actively manage problems can promote adaptive emotional health behaviors and later physical health.

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CHAPTER ONE

Little is known about the psychobiological processes of adults, at age 23, who were born prematurely despite the fact that 1 out of every 9 infants is born too early (Atzil, Hendler & Feldman, 2011; CDC, 2015; Mannisto, Vaarasmaki, Sipola – Leppanen, Tikanmaki, Matinolli, Pesonen, Raikkonen, Jarvelin, Hovi, & Kajantie, 2015). Compared to infants born at term, premature infants, face additional obstacles of immature body systems and more neonatal stress, and are at risk for developmental delay, and possible parental overprotection patterns (Clarke, Cooper & Creswell, 2013; Grunau, 2013; Kopp & Rethelyi, 2003; Pinquart, 2014).

Developmentally, in the United States, 23 year olds are expected to achieve independence and form intimate relationships, yet there is limited knowledge about their emotional intelligence and related psychobiological processes while more is known about their stress levels, coping strategies, and emotional disorders (Clarke, et al, 2013; Ingels, Glennie, Lauff, & Wirt, 2012; Pinquart, 2014; Simpson, 2009). Intrapersonal, interpersonal, adaptability, stress management effectiveness, as well as general emotional health are all involved in reaching adult developmental milestones (Granger & Kivlighan, 2003; Simpson, 2009). These important young adults milestones require the ability to: communicate effectively with others, be sensitive to others, maintain emotional self-control, use both knowledge and experiences to cope, manage stress, work and assume responsibilities (Arnett, 2013; Di Fabio & Saklofse, 2014; Simpson, 2009).

The abilities required to meet young adult developmental milestones are captured in definitions of emotional intelligence (EI) and also incorporates the involvement of

the brain's prefrontal cortex executive functioning which further evolves during this time period (Kristensen, Parker, Taylor, Keefer, Kloosterman, & Summerfeldt, 2014; Arnett, 2013; Davis & Humphrey, 2012; Armstrong, Galligan, & Critchley, 2011; Lishner, Swim, Hong & Vitacco, 2011; Aldao, Nolen-Hoeksema & Schweizer, 2010; Stuss, 2009; Tarasuik, Ciorciari & Stough, 2009; Ciarrochi, Deane & Anderson, 2000). The Hypothalamic – Pituitary -Adrenal (HPA) Axis biological response to stress and prefrontal cortex executive functioning abilities are theorized to be involved with emotional intelligence (EI), functioning, stress, coping, and emotional health (Frodl & Stuart, & Pretorius, 2007; Smith & Vale, 2006; Herman, Ostrander, Meuller & Figueiredo, 2005).

Young adults who were born prematurely carry the consequences of their early birth. As infants, their immature physical systems were responding to ongoing physical pain from neonatal medical procedures, a longer hospitalization and a multitude of environmental stimuli with unstable and ineffective bodily system responses (Grunau, 2002). The foundations for brain growth and functioning and what will become their usual stress response were being laid at this point in time and will likely influence lifelong development including meeting young adult milestones.

However, there is a need for research examining the mechanisms of stress and its relationship with adult emotional health outcome to assure the well being of young adults and their attainment of developmental milestones (Mannisto, et al., 2015; Simpson, 2009; Tarasuik, et al., 2009). There is minimal research into emotional health and its association with biological processes functioning existing to help in identifying the precise timing and targets of health promoting interventions.

Theoretical Framework

The Developmental Origins of Health and Disease (DOHaD) theory of HPA dysregulation postulates that the stress of prematurity and during early life critical brain growth periods (i.e. formation completes during young adulthood) effects the HPA Axis structure and functioning (Eiland & McEwen, 2012; Sullivan, Hawes, Winchester & Miller, 2008; Barker, 2007; Miller, Chen & Zhou, 2007; De Boo & Harding, 2006; Pujol, Vendrell, Junque, Marti-Vilalta, & Capdevila, 1993). Stress responses from the HPA axis are adaptive (allostasis) and if occur frequently, particularly with prematurely born infants and their underdeveloped systems, will result in injury or “wear and tear” (allostatic load) on the body (McEwen, 2003 & 2006; McEwen & Seeman, 2006). If the stress is ongoing or chronic than the “wear and tear” on the body will result in illness (McEwen, 2003 & 2006 McEwen & Seeman, 2006). Cortisol is the primary HPA hormone and it can be measured non-invasively and reliably in saliva (Helhammer, Wust, & Kudielka 2009; Turner-Cobb, 2005; Granger & Kivlighan, 2003). When the body is stressed, the HPA axis releases higher levels of cortisol resulting in metabolic imbalances involving blood sugar and insulin, higher blood pressure, faster heart rate, mood changes, thyroid imbalances and weakened immunological responses (Bruyere, 2009).

A normal salivary cortisol stress reactivity response consists of a peak elevation of salivary cortisol concentrations 10 minutes after the cessation of the test stressor with a decrease to pre-stress levels approximately 90 minutes after the start of the test (Kirschbaum, Pirke & Hellhammer, 1993). In addition, heart rates peak during the

protocol's stressful task and then drops to baseline once the stressor stops (Kirschbaum, Pirke, & Hellhammer, 1993). In a recent study, prematurely born age 6 -10 years olds were found to have an exaggerated cortisol response when faced with a social stressor in a reliable laboratory paradigm, and more emotional and memory problems (Quesada, Tristao, Pratesi & Wolf, 2014). Biological stress responses (i.e. cortisol) play a reciprocating role whether the stress is physical or psychological.

Psychological stress (i.e. social evaluation and the perception of uncontrollability in the situation) activates the cortisol response (HPA Axis), which in turn effects the physical systems. Conversely physical stress activation (i.e. pain) of the cortisol system (HPA Axis) is associated with psychological changes in affective and cognitive processes (Smith & Vale, 2006; Dickerson & Kemeny, 2004).

This study uses a standardized, widely used and well-researched laboratory test for inducing moderate psychobiological stress responses called the Trier Social Stress Test (TSST), (Kudielka, Hellhammer & Kirschbaum, 2010; Kirschbaum, Pirke, Hellhammer, 1993). In a meta-analysis of 208 laboratory stress studies, the TSST was found to repeatedly induce changes in the concentration levels of cortisol (both serum and saliva), as well as other major HPA Axis endocrines, and to significantly cause an increase in heart rate (Kirschbaum, 2010; Kudielka, Hellhammer & Kirschbaum, 2010). Two key components of the TSST's protocol are well studied and known to be needed to induce a reliable and strong activation of the HPA Axis measured in salivary cortisol. The components are the psychological stress of the threat of social evaluation, and the perception of uncontrollability in the situation (Kirschbaum, 2010; Dickerson & Kemeny, 2004). Over fifteen years of TSST

research has shown an increase by 50-300% over baseline in endocrine, immunological and cardiovascular parameters (Kirschbaum, 2010).

Young adults born early with immature body systems may not just *simply grow out of it* and catch up to those born full term (URI, 2011). Subtle, minor and clear differences in attention, hyperactivity, and emotional and socialization effectiveness have been found during childhood and adolescence (Healy, 2010). Guided by the DOHaD theory, the developmental challenges of independent living at age 23 years call for greater knowledge about how mechanisms of stress and capacity for response are seen in former premature infants. There is minimal research on 23-year-old outcomes of stress, coping, emotional intelligence, emotional health, stress reactivity responses and the progression of emotional disorders. More knowledge about the relationship of premature birth, the neuroendocrine stress response, self-reported stress, coping and emotional intelligence will expand our understanding of the well being of young adults and their attainment of developmental milestones. (Mannisto, et al., 2015; Simpson, 2009; Tarasuik, et al., 2009).

Purpose

The Developmental Origins of Health and Disease (DOHaD) framework provides the theoretical perspective for the proposed study. DOHaD asserts that early prenatal and neonatal stress seen in HPA axis function affects later health and behavior. It offers a mechanism to enable the understanding of salient young adult developmental performance abilities. This is a secondary analysis of a well- characterized cohort of premature infants categorized by neonatal illness into four groups of prematurely born infants (Appendix A Measurements) and one group of full-term infants (for a total of

5 groups) who were assessed at age 23 years in a research protocol, which included measures of stress, coping, emotional intelligence, emotional health and the Trier Social Stress Test. The purpose of the study is to: 1. Compare the effect of prematurity on stress, coping, emotional intelligence and emotional health at age 23 years. 2. Examine the neuroendocrine functioning of the stress response and the stress recovery period at age 23 years. 3. Examine emotional health with HPA Axis stress reactivity responses accommodating for prematurity and gender. The knowledge gained from this study can help to inform how neonatal stress of prematurity effects young adult coping, emotional intelligence, physiological responses, developmental milestones and emotional health at age 23 years in a sample at risk for HPA dysfunction. The results can help to identify who may be at risk, the role of the neuroendocrine systems as an underlying mechanism, and suggest clinical interventions to be taken to avoid risk factors and promote future adult health outcomes (Rice, 2012; Kirschbaum. 2010; Sullivan, 2008).

Aims and Hypotheses

In a sample of young adults at age 23, former premature infants with a wide variation in diagnoses of neonatal illnesses and a full term group, the aims of the study, with related hypotheses are:

Aim 1. Compare the effect of prematurity on stress, coping, emotional intelligence and emotional health.

Hypothesis 1. Higher self-reported stress scores, higher use of avoidance coping types, lower emotional intelligence scores and more emotional health disorders will be found for the adults at age 23 years born prematurely

compared to the term-born adults.

Aim 2. Compare the salivary cortisol response to social stress between premature and term-born infants using stress paradigm of the Trier Social Stress Test (TSST).

Hypothesis 2. Adults at age 23 who were born prematurely will have a prolonged stress recovery period of the TSST.

Aim 3. Examine the relationship between effect of emotional health and on the stress recovery period of the TSST measured in salivary cortisol.

Hypothesis 3. The stress recovery period for adults at age 23 years with emotional health problems will be prolonged compared to adults without emotional health problems when prematurity is controlled.

Summary

This study used a well-characterized, longitudinal sample of preterm and full term born infants who have been followed from birth in a series of research studies. The study used neonatal data and self-report of stress, coping and emotional intelligence, clinical diagnosis of emotional health, and neuroendocrine function during a social stress paradigm at age 23 years in a secondary analysis. The study is congruent with the original, larger, study using the same theoretical framework and relevant variables to examine self-reported stress, coping, emotional intelligence and emotional health with the primary biomarker of HPA axis system, cortisol. The overall aim of the study is to examine self-reports of important emerging adult independent function with neuroendocrine activity in the well-standardized social stress test, the TSST. The brain areas most affected by stress are the same areas involved in adapting to stress

and coping effectively (Compas, 2006). Researching these integrated psychobiological processes through multiple analyses will lead to further understanding of how stress effects young adults emotional development (Compas, 2006). This study has the potential to add relevant knowledge about salient developmental characteristics, elements and competencies. The following chapter (2) provides the scientific literature in support of this study. These include the DOHaD theoretical framework and developmental milestones at age 23 years old of the emerging adult who was prematurely born. The use of secondary longitudinal data for analysis will also be addressed.

CHAPTER TWO

Theoretical Background and Related Literature

In this chapter, selected theoretical perspectives for this study are delineated, specifically the Developmental Origins of Health and Disease (DOHaD), neuroendocrine function of the HPA Axis, and prematurity. Age appropriate young adult development at age 23 years will be described in relation to concepts of stress, coping, emotional intelligence, and emotional health. Prematurity effects compared to the full term born for 23 year olds will be understood within a disease developmental theory integrating both biological and psychosocial aspects.

The discussion will begin with a historical review of the DOHaD theory and the contributions up until present time. Included is a review of the construct development of emotional intelligence and the relationship of DOHaD to stress, coping and emotional health.

Theoretical Perspectives

The relatively new Developmental Origins of Health and Disease (DOHaD) theory has gained scientific acceptance within the last thirty years (Wandhwa, Buss, Entringer & Swanson, 2009). DOHaD postulates that fetal and neonatal stressors affect the neurological and endocrine systems adaptive responses, specifically the hypothalamic-pituitary-adrenal (HPA) axis, which is essential for physical and psychological growth (Barker & Thornburg, 2013; Carpenter, Gawuga, Tyrka, Lee, Anderson, & Price, 2010; Wandhwa, et al., 2009; McEwen, 2003; Sapolsky, 2001). The major stress pathway of the neurological system is the HPA Axis (Figure 1) which releases cortisol. Cortisol, the most dominant stress hormone that crosses the

blood - brain barrier, has the important function of adapting the body to both physical and emotional stress responses (Bruyere, 2009). Additionally cortisol is vital in the regulation of blood vessel tone, the inflammatory response, stimulation of glucose production, insulin, and metabolism (Bruyere, 2009). HPA functioning is altered by stress during structural growth periods resulting in permanent programming of early life stress responses that contribute to disease formation later in life (Sullivan, Hawes, Winchester & Miller, 2008).

Figure 1

Hypothalamic-Pituitary-Adrenal Axis

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Premature birth (≤ 37 weeks gestation) occurs at a rate of one out of every nine births, is the leading cause of infant deaths and long-term neurological disabilities in children (CDC, 2014). The normal duration of pregnancy is 9 months (280 days) with full term birth occurring at 40 weeks gestation (Taber's, 2009). Preterm birth, either naturally or by cesarean section, is "*arbitrarily defined as before 37 weeks*" (WHO, 2015; Johansson & Cnattingius, 2010). Preterm birth can be further subdivided into: moderately premature (32- <37 weeks), very premature (28-32 weeks), and extremely premature (<28 weeks), (WHO, 2015). The characteristics of low birth weight and rates of fetal growth has also been used to define prematurity. In preterm research studies, the combination of gestation weeks and birth weights are used to avoid any misclassification especially with infants who have growth restrictions (Johansson & Cnattingius, 2010). Low birth weight is $<2500\text{g}$ (5 lbs. & 8 oz.), very low birth weight is $<1500\text{g}$ (3 lbs. & 4 oz.), and extremely low birth weight is $<1000\text{g}$ (2 lbs. & 3 oz.), (Johansson & Cnattingius, 2010).

Surviving preterm born infants may have intellectual disabilities, neurological problems, respiratory, visual, hearing and digestive problems (CDC, 2014; Martin & Osterman, 2013). Premature infants have experienced prenatal stress, often from health risk factors in the mother and postnatal stress from months-long intensive care in the Neonatal Intensive Care Unit (NICU). These early stress experiences evoke broad biological responses in the underdeveloped premature infant's neurophysiological systems including brain growth occurring during uterine

development (Phillips, 2001) and peak growth spurts well into the mid-twenties of age (Epstein, 1986). Thus, the premature infant's neuroendocrine system is frequently activated leading to the inductive development and empirical evidence in support of DOHaD theory.

Historical Evidence and Perspectives

DOHaD evolved from Barker's original "Fetal Origins Hypothesis" which originated inductively from epidemiological studies. (Barker, 1990; 2004, 2007; Barker & Thornburg, 2013; Wandhwa, et al., 2009; Gluckman & Hanson, 2007; Reynolds, 2007; Hofman, Regan & Cutfield, 2006). During this time period multiple retrospective mortality and epidemiological studies from different countries showed evidence that adult height and geographical differences were related to infant mortality caused by heart disease (Barker, 1990, Barker, Erickson, Forsen, & Osmond, 2002; Phillips, 2001). An influential study, consisting of 499 people at age 50 born in England, revealed their current blood pressure measurements and hypertensive risk factors were strongly related to the measurements of their hospital-recorded placenta and birth weights (Barker & Osmond, 1986; Barker, 1990). A lack of evidence was found for the role of some commonly involved environmental variables in heart disease, such as a high fat diet, and this finding prompted an alternative hypothesis (Barker, 1995, 2007; Barker & Osmond, 1983). As a result of this landmark study, a paradigm shift representing a new conceptualization of disease causation occurred (Barker, 2007). This shift in scientific thinking about adult diseases, which was defined as degenerative in nature and viewed as a result of gene and environmental interactions, occurred and resulted in the inclusion of biological programming during

fetal and infant life (Barker, 1990) as a plausible explanation (Gordis, 2009).

Barker's (1995) initial assumption was: "*fetal under-nutrition in middle to late gestation, which leads to disproportionate fetal growth, programs later coronary heart disease* (p 171)" lead to further studies from this hypothesis. Later Barker (2004) refined this to the hypothesis:

"Cardiovascular disease and type 2 diabetes originate through developmental plasticity, in response to undernutrition. As it is now known that growth during infancy and early childhood is also linked to later disease 'developmental origins hypothesis' is now preferred (p. 114). "

Barker, defined a process of developmental plasticity as: "*a critical period when a system is plastic and sensitive to the environment, followed by loss of plasticity and a fixed functional capacity*" (Barker, 2007, p 415). According to Barker, developmental plasticity also has three features: 1. The response will depend on the nature of the environmental cue. 2. There are critical time periods for different systems when changes will occur and these changes may be temporary or permanent. 3. Duration of these critical time periods are different depending on the structure with the brain periods longer. These changes can be gross, substructure or functional.

The imbalance of fetal nutrients and oxygen was thought to result in an alteration of not only metabolic yet endocrine functioning leading to smaller birth weights and a variety of adult pathologies (Drake, Tang, & Nyirenda, 2007; Phillips, 2007; De Boo & Harding, 2006, Gibson, Carney & Wales, 2006). Despite the scientific acceptance of this explanation, methodological issues surrounding the use of body measurement, birth weight or gestation age, and studies designed without a well-characterized cohort

utilizing prenatal and adult health outcomes, as well as observational and prospective designs, added to concerns about confounding variables (socioeconomic status, diet, cigarette smoking, physical exercise and selection bias), statistical effect sizes (attrition and statistical over adjustments) and publication bias (Skogen & Overland, 2012; Erickson, 2006; Godfrey, 2006). A 2003 meta-analysis study addressing publication bias relating to low birth weight and higher blood pressure found a weaker association than initially determined yet maintained support for the fetal origins hypothesis (Skogen & Overland, 2012). Despite a weaker association found, while addressing some of the common confounding variables as alternative explanations, better research methodologies resulted and improved the replication of findings (Gordis, 2009).

Further studies from a variety of countries designed to control for confounding variables supported the association between low birth weights as a fetal antecedent to diseases (Barker & Bagby, 2005; Vohr, Wright, Dusick, Mele, Verter, Steichen, Simon, Wilson, Broyles, Bauer, Delaney-Black, Yolton, Fleisher, Papile & Kaplan, 2000). At the same time debates occurred focusing on the idea that the only important applicable time period for DOHaD was during pregnancy and the theory was useful in explaining only cardiovascular diseases (Gluckman & Hanson, 2006; Godfrey, 2006). Indeed, DOHaD flourished in explaining cardiovascular risk (Bryan & Hindmarsh, 2006). Later, researchers significantly correlated low birth weight with increased risk in a number of diseases that are part of the metabolic syndrome (Hofman, Regan & Cutfield, 2006) such as truncal (middle body) obesity, hypercholesterolemia, atherosclerosis, coronary artery disease, insulin resistant

diabetes mellitus, cardiomyopathy, congestive heart failure, autoimmune disease, anxiety, depression, chronic pain and headache (Fricchione, 2015). Replicable cross sectional studies of relationships between disease formations with metabolic illnesses supported further study of the HPA axis associations.

The results from a retrospective longitudinal study of Helsinki, Finland male adults, gave credence to the findings that smaller infants have a higher rate of cardiovascular disease for men in adulthood (Ericksson, Forsen, Tuomilehto, Osmond & Barker, 2001, Godfrey, 2006). The sample size consisted of 4,630 men born in Helsinki (1934-44) and utilized child welfare clinic medical health records with multiple time points of childhood through adult weight recordings, height measurements and hospital admissions for coronary heart disease (Ericksson, et al., 2001). Overall, low birth weight was associated with heart disease, low weight gain was associated with an increased risk of heart disease and rapid weight gain after age 6 was associated with further risk (Ericksson, et al., 2001). As a result of this study, determining what changes occurred in prenatal growth and those that occurred later lead to focusing on the interactions of both prenatal and postnatal environments in the development of adulthood cardiovascular diseases (Ericksson, 2006; Godfrey, 2006).

Concurrently animal researchers showed that exposure of rats during pregnancy and after birthing, along with their offspring, to a variety of stressors resulted in elevated stress-induced cortisol levels in the off-spring and disease development (Phillips, 2001). This study added a “dose-response” relationship, or intensity and timing of the exposure of stressors to the DOHaD research literature. Animal studies, pinpointed the HPA Axis response to stressors introduced during critical times of rat

brain growth, that occurred after birth and produced permanent changes in the animals HPA Axis response (Matthew, 2002; Phillips, 2001). As mentioned earlier, in humans, critical brain growth occurs during uterine development (Phillips, 2001) and continues with peak growth spurts well into the mid-twenties of age (Epstein, 1986).

Sterling and Eyer in 1988 coined the word “allostasis” (Sterling & Schulkin, 2004) based on research with monkeys while studying high blood pressure. Allostasis was now a new paradigm to explain arousal pathology and replaced homeostasis conceptually. Allostasis involves regulation by: varying parameters and variations in anticipated demands. A new core assumption now was that physiology is sensitive to social relations. Allostasis also depended on higher-level brain functioning, other than basic physiological automatic responses, and involved prefrontal cortex regulation. Anticipatory regulation for anxiety and satisfaction was found to rely on the prefrontal cortex through neuronal mechanisms.

McEwen, in 1989, further developed these principles through “allostatic load” and is credited with the advancement of the theory by publishing research findings related to human autonomic, central nervous system, endocrine and immune system activity (Sterling & Schulkin, 2004). McEwen (2006) implicated stress to an event as an individual biological response factor in the development of a disease. In addition to acute stress events, McEwen defined the effects of general “wear and tear” (p 367) on the body as allostatic load that targets the HPA Axis, releasing an end product of cortisol and can lead to the development of adulthood diseases (McEwen, 2003 & 2006).

The main hypothesis of the DOHaD theory, involves one sensitive brain area of

prenatal and postnatal development occurring in adulthood disease development by resetting the glucocorticoid endocrines, which is the Hypothalamic-Pituitary –Adrenal (HPA) Axis (Sloboda, Newnham, Moss and Challis, 2006). McEwen (2008) further delineated this dimension of the theory by postulating that stress hormones have a central effect in health and disease by providing protective, damaging and mediating effects.

These mediating stress effects can be from a physical, psychological, emotional, cognitive, intellectual, major life events, environmental or social/caring interactions. Biologically, individuals can either adapt to acute stress (allostasis) or become overloaded (allostatic load) with chronic stress resulting in pathophysiological changes (McEwen & Seeman, 2006). See Figure 2.

Figure 2

Stress Response and Development of Allostatic Load

Figure 2. Reprinted by permission from Macmillan Publishers Ltd; Neuropsychopharmacology, 2000, by B., McEwen, Allostasis and allostatic load: Implications for neuropsychopharmacology, *Neuropsychopharmacology*, 22, 108-124. Copyright 2000 by Nature Publishing Group.

The brain is viewed as not only the controller of the stress response yet conversely as the target (Rubinow, 2006). The HPA Axis as programmed is effected by the totality of lifelong stressors (cumulative risk) and negative effects on the brain and body (allostasis/allostatic load) leading to the long-term consequences of adulthood disease (Manzanares, Monseny, Ortega, Montalvo, Franch, Gutierrez-Zotes, Reynolds, Walker, Vilella & Labad, 2014; McEwen, 2012, Sullivan, et al., 2008). In other words, the adaptability to a stressor or anxiety rather than the initial reaction will predict long-term outcomes and allostatic load becomes the unifying concept between cumulative risk and HPA dysregulation (Manzanares, et al, 2014; McEwen, 2012, Sullivan, et al., 2008).

McEwen did initially base his theory on the idea of homeostasis, which conceptually is a bodily system that is stable and unchanging (Dictionary.com, 2015) and postulated there is an optimal level and ideal set point (McEwen, 2004). This explanation evolved to include the idea of variation (allo) of levels and set points achieving a balance in the total system (Dictionary.com, 2015). McEwen points out: Homeostasis is about adjusting this level while allostasis is about the brain coordinating body-wide changes to achieve stability through change (McEwen, 1998 & 2004). Thus adaptation is a central concept of the theory. Stressors, according to McEwen result in experiences that are either acute or chronic. Acute stress is the “fight or flight” response or those responses resulting from major life events. Chronic stress is defined as the accumulation of minor and daily stresses.

There are four types of allostatic load (Figure 3): normal, repeated, lack of adaptation and inadequate (McEwen, 2000 & 2007). Endocrine and metabolic

Figure 3

Four Types of Allostatic Load

"Four types of allostatic load are illustrated. The top panel illustrates the normal allostatic response, in which a response is initiated by a stressor, sustained for an appropriate interval, and then turned off. The remaining panels illustrate four conditions that lead to allostatic load: 1) Repeated "hits" from multiple novel stressors; 2) Lack of adaptation; 3) Prolonged response due to delayed shut down; and 4) inadequate response that leads to compensatory hyperactivity of other mediators, e.g., inadequate secretion of glucocorticoid, resulting in increased levels of cytokines that are normally counter-regulated by glucocorticoids). Figure drawn by Dr. Firdaus Dhabhar, Rockefeller University."

Figure 3. Reprinted by permission from Macmillan Publishers Ltd; Neuropsychopharmacology, 2000, by B., McEwen, Allostasis and allostatic load: *Implications for neuropsychopharmacology*, *Neuropsychopharmacology*, 22, 108-124. Copyright 2000 by Nature Publishing Group.

responses protect the body from allostatic load in the short term through homeostatic adaptation called allostasis. Chronic stressors or allostatic load, whether physical, psychological or a combination, result in structural brain changes that effect our physiological and behavioral responses and lead to the development of adulthood diseases (Ewen, 2003 & 2006).

The stress experience or the stress response that the individual has to a potential stressor, is the focal point (McEwen, 1998) and added to “Barker hypothesis” of prenatal stage development, the future time determinants of adulthood diseases (Skogen & Overland, 2012). Stress as defined by McEwen is a “*state of real or perceived threat to homeostasis*” and stressors are “*aversive stimuli*” while “*maintaining homeostasis through activation of complex responses involving the endocrine, nervous and immune system*” is the stress response (McEwen, 2006). Luthar, Cicchetti and Becker, 2000, are clearer in their definition of the stress response, as a dynamic equilibrium, meaning an ability to sway and not as a fixed or static state. When a good adjustment is achieved across different domains of the stress response, in the face of significant adversity, then “*resiliency*” is achieved (Luthar, Cicchetti & Becker, 2000; Ficchione, 2015). Thus, the capacity to maintain allostasis while challenged by mental and physical aversive stimuli to well being, constitutes resiliency (Ficchione, 2015). One Mind Body Medicine equation (hypothesis) is formulated as (Ficchione, 2015):

Stress (Allostatic Loading)
Resiliency Factors = Selective Vulnerability: Propensity to physical and mental illness

Ficchione (2015) defines resiliency factors as: “*relaxation response, mindfulness; social support/prosociality; cognitive skills; positive psychology; spirituality; exercise; nutrition; healthy habits*”.

According to McEwen (1998) there are two factors that determine the individuals stress response: how the situation is perceived and the individuals’ general state of health (determined by genetics, behavior and lifestyle choice), (See Figure 2).

Matthews (2002) added that the timing and intensity of the aversive event and/or an intervention also effects HPA axis development and functioning. This focus on later stress experiences added environmental triggers to critical or sensitive growth time periods as a second possible causal pathway to disease suggesting the involvement of more than one critical time period (Skogen & Overlad, 2012). Stressors, occurring prenatally result in adaptive changes within the HPA-Axis, become permanently programmed, and impact health during adult life (Reynolds, 2013; Sullivan, Hawes, Winchester & Miller, 2008; Barker, 2007) while later environmental triggers and responses add to the allostatic load depending on coordination with sensitive growth time periods (Skogen & Overland, 2012).

The body’s stress response helps individuals adapt to a problem and marshal the resources to respond which includes releasing response coordinating hormones. A unifying and central relationship is if the stress response is activated too frequently or under utilized then the stress-response itself can be more harmful than the stressor and this concept is called allostatic load (McEwen, 1998 & 2004). There has been

controversy over the labeling of this phenomenon yet the underlying concept has not been challenged. McEwen (2004) does identify features of some stress processes that do not change in order to help and adds those processes that do vary in the context of life cycles, individual experience and responses to the physical and social environment (McEwen & Wingfield, 2010). The challenge, according to McEwen, in the definition is that allostasis adds to homeostasis a focus on how individuals have access to bodily resources to respond to problems with the environment (McEwen, 2004).

Welberg and Seckl, 2001, found that stress during pregnancy could permanently alter behavioral and/or physiological reactivity to stressors (Sullivan, et al, 2008). The authors extensively reviewed available research of epidemiological, animal biological, human biological, anxiety, cognition, neural mechanisms, under-nutrition, interactions with postnatal environments and glucocorticoid studies. In terms of HPA function, the evidence showed:

“Birth weight correlates closely with HPA measures from infancy (206), through adolescence and young adulthood (207) to old age (208). These data suggest that low birth weight associates with both increased basal and ACTH-stimulated cortisol levels (207, 209). Taken as a whole, these findings are compatible with the hypothesis that fetal overexposure to glucocorticoids whether exogenous DEX or endogenous cortisol may underlie at least in part the connection between the prenatal environment and adult stress-related and behavioral disorders (Welberg & Secki, 2001, p 123).”

As this evidence became available another shift in thinking about adulthood disease formation occurred and became widely accepted by the scientific community

(Skogen & Overland, 2012; Salonen, Kajantie, Osmond, Forsen, Yliharsila, Paile-Hyvarinen, Arker & Eriksson, 2011; Gluckman & Hanson, 2006; Godfrey, 2006). The scientific community was now in consensus that relevant life periods are on a continuum that includes during pregnancy, infancy and throughout the life span (Gluckman & Hanson, 2006; Godfrey, 2006). The focus of the theory was now based on two main assumptions. The first assumption is early life events that occur during periods of critical biological growth partially determine future adulthood disease development and secondly, this has implications for both disease development and promotion of health (Gluckman & Hanson, 2006; Godfrey, 2006). DOHaD theory research then branched into three major areas of interest: 1. Maternal, fetal and postnatal nutrition, 2. Preterm birth and, 3. Epigenetics (or gene modification), (Wadhwa, et al., 2009; Waterland & Michels, 2007).

DOHaD theory has been applied to a variety of diseases and health concerns: behavioral, cancer, cognitive, diabetes, metabolic, muscular, neurological, psychological and respiratory (Barker & Thornburg, 2013; Gluckman & Hanson, 2007; Hofman, Regan & Cutfield, 2006; Barker, 2005 & 2004). The theory is widely used in behavioral medicine and specifically with interventions directed at reducing stress responses (Benson, 2015). Researchers are also exploring the effects of stressors, as measured by cortisol levels and magnetic resonance imaging, with major psychiatric disorders such as post-traumatic stress disorders, anxiety responses, stress responses, cognitive functioning, eating disorders, childhood disorders, personality, long-term effects of child abuse, psychosis and addictions (Nosarti, Murray & Hack, 2010). Indeed, anxiety, an autonomic nervous system response triggered by HPA

functioning, is a general symptom of stress and co-occurs with other psychiatric disorders, especially depressive disorders (APA, 2000).

Theory Analysis

DOHaD a relatively new theory addressing the origins of adulthood disease is widely accepted, utilized in clinical interventions and research studies. Originating from epidemiological study results DOHaD has developed into a major disease causation theory. The original theory was intended to explain one aspect of disease causality and to be applicable to multiple diseases including those “*future entitles yet unknown*” (Barker, 2007). Extending the original Fetal Origins Hypothesis, beyond the initial hypothesis of environmental influences during pregnancy has an effect on later development, expanded the perspective beyond biological determinism (Skogen & Overland, 2013). The addition of stress responses through further refinement of the HPA Axis dysregulation hypothesis, multiple critical time periods and life span influences took into consideration the role of other causal issues. In addition to physical stressors, an individual’s perception of stress as threatening or uncontrollable has been shown to activate the HPA axis as well as coping styles choices affects on later life stress-related disease development (Figure 4). The continued development along this thinking moved DOHaD to a fuller life course perspective with multiple causal factors including emotional health (individual perceptions of stress interpreted as threatening or not) environmental (such as parenting) and for some the addition of the biological bases of mammalian evolutionary attachment (social influences) perspectives (Skogen & Overland, 2013; Fricchione, 2011).

DOHaD theory defines a plausible biological temporal relationship between

disease formation and the role of the HPA axis. Research results show an association, of a dose-response relationship of intensity and timing of stressors, and replication of

Figure 4

The Cardiovascular Toll of Stress (Emotional and Physical Stressors, HPA Axis & Health)

Figure 4. Reproduced with permission of Lancet Publishing Group; Brotman, D.J., Golden, S. H., & Wittstein, I.S. (2007) The cardiovascular toll of stress, *The Lancet*, 370, 1089a1100.

findings in specificity to stress-related illness, although the majority of research in the United States, has been at discrete time periods in a person or population's life. DOHaD theory states the necessary condition of structural and functional changes prenatally and whether any of this is irreversible remains to be seen (Skogen & Overland, 2012). Some common confounding variables as alternative explanations have been addressed and the national government (National Institute of Health) has prioritized the use of longitudinal studies to address the complexities of what factors (NIHR, 2015), may or may not be sufficient or necessary for disorders.

Literature recommendations for further research focus on utilizing regression modeling statistical strategies to address: the association between the two variables of early exposure and adult outcomes, intermediate exposures, the interaction between the early exposure and intermediate variables and to what degree the intermediate variable is related to disease outcome (Skogen & Overland, 2012). Research into the effects of stress experiences on HPA Axis development, function and dysregulation also requires addressing the roles of birth term, gender and social inequalities as confounding moderating variables (Matthew, 2002; Sapolsky, 2009). The renewed interest in DOHaD theory is leading researchers into areas of study that promise to identify risks and protective mechanisms; locate periods of transitions into pathology, develop preventive and possibly corrective interventions to intervene in the progression of disease pathologies over the course of a lifetime (McEwen & Wingfield, 2002; Sullivan, Hawes, Winchester & Miller, 2008; Ben-Sholmo & Kuh, 2002).

Socioeconomic Status (SES)

Many socioeconomic status (SES) factors are associated with low birth weight, coronary heart disease, under nutrition, low literacy rates and health disparities (Senterfitt, Long, Shih, & Teutsch 2013; Baber, Muzaffer, Khan, Imdad, 2010). Social and economic factors are considered the largest single predictor of health outcomes and influencer of health behaviors (Senterfitt, et al., 2013). Disparities between countries in preterm birth weights have been partly explained by differences in SES (Johansson & Cnattigus, 2010). Multiple studies have found that the lower the social, education, and economic position the higher the unhealthy behaviors (i.e., smoking, physical inactivity) and inability to engage in healthy behaviors (Senterfitt, et al., 2013).

The World Health Organization (WHO, 2015) includes determinants of SES consisting of: the physical environment, the person's individual characteristics and behaviors (including how they "*deal with life's stresses and challenge*, (p 1)", social support networks, genetics, available health services and gender. Additionally, a WHO (2003) sponsored study, found middle-class office workers and lower ranking staff has more disease and die earlier than higher positioned workers. The WHO report (Wilkinson & Marmot, 2003) focuses on ten main areas of what is known:

1. Life expectancy and shorter life spans occur further down the social ladder.
2. Stressful circumstances lead to worry, anxiety, inability to cope that is damaging to health.
3. Foundations are laid in childhood: "*slow growth and poor emotional support raise the lifetime risk of poor physical health and reduce physical, cognitive and emotional function in adulthood*, (p 14)".
4. Poor life quality shortens lives.

5. Stressful workplaces increase the risk of disease. 6. Health risks of unemployment increases the rates of premature death. 7. Supportive relationships contribute to health. 8. Individuals' addictions to alcohol, drugs and tobacco numb difficult conditions and lead to downward mobility. 9. Healthy food is a political issue. 10. People's dependence on cars has increased resulting in less walking and social contact and more traffic accidents and air pollution.

One of the earliest criticisms of the interpretations from DOHaD studies is the the confounder of SES could explain results used to support the theory (James, Nelson, Ralph, & Leather, 1997; Skogen & Overland, 2012). James, et al, (1997) found lower socio- economic groups have more premature and low weight births, more illnesses, more risk factors and less nutritional diets. The DOHaD theory emphasizes that chronic exposure to stress mediators of the HPA Axis and the sympathetic nervous system effects multiple organs resulting in disease (Dowd, Simanek & Alello, 2009) although SES confounding variables in earlier studies were not adequately controlled or interpreted.

Allostatic load has been used to explain part of the association between SES, health and disease. Lupien, King, Meaney, & McEwen (2001), as one goal of three, explored the possibility that because lower SES status involved higher stress and fewer coping resources then morning salivary cortisol levels would differ from other SES groups. The cross-sectional experimental design study, utilized 307 children from a school setting, with 6 age groups (6-16 years old) and two categories (low and high) of SES. Overall findings showed that lower SES in children related to higher cortisol levels with the impact of SES on cortisol absent after transition to high school.

The authors identified four possible social explanatory factors of: changing status, influence of peers, influence of youth culture, and resilience.

In contrast a systematic review of the literature, extending up until June of 2009, on SES and biomarkers of physiological systems, was conducted by Dowd, et al., (2009) to address SES, cortisol levels and indirect measures of allostatic load. A total of 26 studies met the inclusion criteria of reported associations between an indicator of SES and cortisol, and/or allostatic load. Fourteen of these studies utilized salivary cortisol secretions. The findings overall were mixed with little evidence that lower cortisol related to lower SES and lower SES related to higher allostatic load measurements. Overall, the authors found more studies with no associations of cortisol to SES than the intuitive finding of lower SES associated with higher cortisol levels. The unexpected findings were attributed to differences in the nature of this relationship or inconsistencies in measurements and analysis of both cortisol and SES. Standardization of cortisol procedures and analysis, variations in SES indicators used, and the exclusion of subjects using stimulants of cortisol such as smoking, were major recommendations for future research. Although the review focused on cortisol level daily patterns and indirect measurements of allostatic load, both laboratory stress induced and dexamethasone challenges, were felt to provide more controls for research on the differences of SES on HPA function.

Pluobidis, Benova, Grundy & Layton, 2014, identified four major hypotheses from the literature about the associations between SES and later life health: 1. Early life SES directly effects later life health. 2. Early life SES indirectly effects later life SES. 3. Early life and later life SES effects health through accumulation of risk.

4. Early life health indirectly effects later life health via later life SES. A sample of aged 50-53 years was taken from an English longitudinal study on aging. Multiple measurements of early life and later life SES, health, and fibrinogen levels (indicator of aging) were obtained. The four major hypotheses were compared through statistical modeling. In general, results found early life SES extends directly until the beginning of old age and predicts health at age 65 and older yet fibrinogen levels will vary.

Co-existing with SES are social risk factors defined as (Msall, Sullivan & Park, 2010): “*suboptimal home and community environment, poverty, domestic violence, drug addictions, crime, hunger, and poor quality housing* (p 224).” The conditions of low SES along with access to care issues, coping with multiple adversities, helplessness and low self-esteem; contribute to the risk of preterm births (p 225) along with ethnicity, family history, maternal characteristics, multiple pregnancies and air pollution (Johansson & Cnattigius, 2010; Msall, Sullivan & Park, 2010).

Low SES is consistently associated with poor health and disease yet how this gets translated into biological risk is uncertain and studies have shown inconsistent and at time weak results (Pluobidis, Benova, Grundy & Layton, 2014; Senterfitt, Long, Shih, & Teutsch 2013; Baber, Muzaffer, Khan, Imdad, 2010; Dowd, Simanek & Alello, 2009; Wilkinson & Marmot, 2003; Lupien, King, Meaney, & McEwen, 2001). Methodological issues in past studies have helped confound the influence of SES and effected some interpretations of DOHaD theory evidence.

In this study, participants’ selection criteria at birth, as designed in the original research, involved representation from all SES groups in each variable of birth

status (preterm and full-term born) to control for this effect. Additionally later SES status at age 23 was assessed for possible individual differences and SES group variations from the prenatal time period. Multiple measures of SES status, in addition to income, were used and included standardized instruments, education level, occupation level categorization and neighborhood ratings (Farrington, 1991).

Race and Ethnicity

The Center for Disease Control (CDC) found differences in preterm birth by race and ethnicity and statistically analyzed the relevant differences, using percentages, and z tests at the 95% confidence level (Martin & Osterman, 2013). The percentage results were: 1. Black infant preterm birth rate (17.1%) was 60% higher than for White infants (10.8%). American Indian/Alaska Native (13.6%) and Hispanic (11.8%) preterm birth rates were higher than White infants. 2. Black infants had double the early preterm birth rate (6.1%) than Whites, 25% higher than Hispanics (Reagan & Salsberry, 2005), and other ethnicities (2.9%). 3. Black infants were 40% “*more likely to be born late preterm than White infants with Hispanic infants more often than White infants*, (Martins & Osterman, 2013).”

Reagen and Salsberry (2005) studied the health disparities of preterm births among Blacks, Hispanics, and Whites focusing on social contexts of neighborhood disadvantage and cumulative exposure to income inequality while controlling for individual risk factors. Neighborhood poverty and housing vacancy rates increased the rates of premature births for Blacks while income inequality directly effected only Hispanics (Reagan & Salsberry, 2005). Notwithstanding these findings, the close relationship of social risk factors from low SES to ethnicity (Msall, Sullivan & Park,

2010) confounds the separation of SES from ethnicity effects. Likewise, other epidemiological studies have shown, that among the multiple causes for spontaneous preterm births, being a member of the Black race, is also a risk factor (Goldberg, Culhane, Iams & Romero, 2008).

Mustillo, Krieger, Gunderson, et al., 2004, found self-reported experiences of racial discrimination by Black women were related to premature birth weights and may contribute to disparities in perinatal health between races (Black 50%: White 5%). One landmark study accounting for social disparities, showed that even college educated Black women have an increase rate of premature births when compared to White college educated women (Schoendorf, Hogue, Kleinman, et al., 1992). In past racial and ethnic disparities research, studies have separated their focus on either the social construct of race or the biological processes (Kramer & Hogue, 2009).

A systematic review of the research literature, in 2009, focused on integrating the racial biological and social patterning of premature births, with the expressed purpose of ‘*understanding the etiology of black-white racial disparities in preterm birth*’ (Kramer & Hogue, 2009, p 85)”. Over 1,459 citations were reviewed spanning from 1960-2009. Studies utilizing ultrasound-based measurements and data cleaning methodology approaches that decreased misclassification were utilized. Conceptual framework reviews lead to 3 primary biological pathways mediating the racial disparities in preterm birth: placental dysfunction, HPA dysfunction and maternal-fetal inflammation. Pre and peri-conceptual maternal health as well as genetic and epigenic pathways studies were included in the review. Overall the researchers found evidence to support socially patterned maternal stress as a cause of

racial disparities. The identification of few studies addressing genetics and the challenges of controlling for multiple causal explanations prompted the authors' suggestions for future research. The suggestions included incorporating biological markers into socially focused preterm birth studies as well as improved epidemiological design studies.

In this secondary analysis longitudinal study, the original racial and ethnic composition of the sampling is predominately White and reflective of the population and geographical location in Southeastern New England from 1985 to 1989 (Sullivan, et al., 2008). The homogenous composition of this study population will be applicable to the White racial group and not reflect the disparities inherent between Black and Hispanic populations. The identification of the White racial group preterm health outcomes from 1985-1989 may contribute to further knowledge of health advances made since that time period and could possibly be utilized to compare the associated magnitude of racial disparities today.

Gender

Globally, males are slightly more likely to be born prematurely than females (Katz, Lee, Kozuki, et al, 2013). Decades of past research have shown males born prematurely have higher mortality and morbidity and the phenomena is often referred to as "male disadvantage" (Brothwood, Wolke, Gamsu, Benson & Cooper, 1986; Stevenson, Verter, Fanaroff, et al., 2000; Banga, Barche, Singh, Sheehan & Vasylyeva, 2015). The confirmed risks of high blood pressure and placenta abnormalities to the pregnant mother carrying a male fetus is thought to occur secondary to sex differentiation hormones in utero and at conception (Katz, Lee,

Kozuki, et al., 2013, Ingemarsson, 2003). Moreover, Sweden national figures show death rates are higher for males by 55- 60% when born between 23 and 32 gestational weeks (Ingemarsson, 2003). Immediate complications of respiratory distress syndrome are greater for prematurely born males and cognitive recovery after intracranial hemorrhage is less when compared to premature females (Ingemarsson, 2003). Similarly, in the United States, males are also more likely than females (OR = 1.21; 95% CI: 1.02 - 1.42) to be born at 33 to 36 weeks (McGregor, Leff, Orleans & Baron, 1992). To put it another way, if a male and female are born at the same prematurely gestational age then male infants risk becoming more seriously ill than females (SMFM, 2015).

While prematurity survival rates have increased, the prematurely born at 25 weeks will develop disabilities (1:10) such as lung disease, cerebral palsy, blindness or deafness; 50% disabilities; and more commonly cognitive and neurological impairments (Banga, Barche, Singh, Sheehan & Vasylyeva, 2015). A retrospective chart review of 160 (male 59% and female 41%) pediatric records at a Texas clinic focused on children and adolescents born prematurely and any gender differences in medical diagnoses. The sample consisted primarily of White (39.2%) and Hispanic (38.0%) races born prematurely and between 10 and 21 years old at the time of the chart review. Gestational ages were divided into two groups of 32-37 weeks and < 32 weeks with birth weight divided into 7 groupings ranging from extremely low birth weight to large for gestational age. The incidence of neonatal complications between genders was assessed according to: jaundice, metabolic complications, respiratory distress syndrome, sepsis, intracranial hemorrhage and hypertension.

Long-term morbidities included ICD-9 diagnoses of: asthma, allergic rhinitis, cardiac defects, behavioral issues, developmental delays, growth delay and kidney anomaly and diseases. Even though, more preterm born males were at weights appropriate for their gestational age, the study found males had a higher incidence of neonate complications especially: jaundice (63.1 vs. 36.8%; $p = 0.02$), metabolic issues (64.2% vs. 35.7%, $p = 0.03$), and respiratory distress syndrome (60.5% vs. 39.4%, $p = 0.02$), (Banga, Barche, Singh, et al., 2015). In contrast, prematurely born females weights were primarily small for gestational age. No differentiation between genders for neonatal diagnoses of intracranial hemorrhage, sepsis or hypertension were identified. The only significant gender difference in long-term morbidities found was notably in behavioral issues for males and mostly diagnosed with attention deficit hyperactivity disorder (6% vs. 2%; $p < 0.01$), (Banga, Barche, Singh, et al., 2015).

The prematurely born female chances of survival are higher than males yet Black prematurely born females, weighing about 2.2 pounds or less, have a higher rate of survival than their White peers (UF, 2006). Researchers (UF, 2006) who studied vital statistics from Florida, between the years of 1996 and 2000 utilizing records of 5,076 babies born in the state, found females at lower birth rates fared better with Black females faring better overall. Although the researchers are yet unable to explain why this racial and gender phenomena exists it is known that female premature babies in general have more developed lungs than males (UF, 2006).

Finally, there are a few decades of history of the vulnerability of preterm males over preterm females for increased mortality and morbidity. Studies have shown

despite advancements in preterm neonate care this phenomenon continues to exist as a risk to full term pregnancy and neonatal complications with male births. A few endocrine and biological adaptation explanations have been proposed without any known etiology of this gender-related health disparity (Banga, Barche, Singh, et al., 2015). In this study, overall gender differences as well as gender and birth status interaction are analyzed to identify the direction and strength of this effect (Baron & Kenny, 1986).

Prematurity and Development at Age 23 as Emerging Adults

Multiple factors including SES, race, gender, environment and lifestyles, to mention a few, influence the health and the formation of physical diseases and emotional health in all adults. The prematurely born Age 23 emerging adult entered this world with the disadvantage of multiple immature bodily systems. Additionally, as has been previously stated, human brain growth occurs during uterine development (Phillips, 2007), the newborn's central nervous system (CNS) evolves rapidly and peak growth spurts continue well into the mid-twenties of age (Epstein, 1986) resulting in multiple sensitive time periods of critical influence. In preterm infants who require extensive neonatal intensive care, it is possible that the HPA Axis is repeatedly activated which may result in permanent programming of early life responses (Maniam, Antoniadis, & Morris, 2014; Reynolds, 2013; Sullivan, Hawes, Winchester & Miller, 2008). Routine medical procedures activate the newborns stress response system to react and moderate levels of endocrine hormones have been found (Jensen, Beijers, Riksen-Walraven & de Weerth, 2010) that contributes to alterations within the HPA Axis or fetal programming from cell death, and failed or

delayed responses of the central nervous system (CNS), (Sullivan, Hawes, Winchester & Miller, 2008). See Figure 5

Figure 5

Prematurity, Postnatal Stress and HPA Function

Figure 5. Reprinted by permission Mary. C. Sullivan. 2008-2013. In “Risk and protection in trajectories of preterm infants: Birth to adulthood (Grant # NIH R01 NR003695-14).” Bethesda, MD, National Institutes of Health, National Institute of Nursing Research.

Structural differences in the brains of premature low birth weight infants with alterations continuing into adulthood have been found by researchers (Nostarti, Murray & Hack, 2012). Measurements of brain pathology using a variety of biomarkers, such as salivary cortisol levels as an endocrine marker of the HPA Axis activation is in wide use and may serve as a transitional marker for psychological

pathology (Turner-Cobb, 2005).

Feldman, Weller, Sirota & Eidelman (2002) in Israel, studied the effects of mother to infant (or Kangaroo Care) skin-to-skin contact on both prematurely and full term born infants. Specifically they examined “*infants’ capacity to regulate sleep and wake states, organize behavior, regulate negative emotions, modulate arousal, coordinate attention to mother and an object, and sustain effortful exploration of the environment* (p 194)”. The infants born prematurely showed an abnormal response to stimuli and often could not inhibit reactions. Three theoretical perspectives were included: There is unique time windows for input required for optimal development of the central nervous system and behavioral organization. Sensory development is sequential and lastly maternal proximity organizes sleep, rest and behavior inhibition. A prematurely born group with intervention ($n = 73$) and a matched control group ($n = 73$) without intervention was used. Pre and post interventions as well as multiple time point measurements were used. The premature infants who received skin-to-skin contact from the mother were found to benefit by improved behavior organization and emotional regulation when they reached full term. Hence, self regulation and the ability to adjust behaviors to the situation is a challenge to the preterm infant, requires environmental control and sets up regulation parameters overtime (M.C. Sullivan, personal communication, November 11, 2014).

Affect regulation of emotional experiences to serve a purpose or goal contribute to meeting developmental milestones and adult maturation while emotional influences on decision-making have been found during adolescent to contribute to behavioral (alcohol and nicotine dependence), emotional and clinical disorders (Dahl,

2001). Prematurely born children are at increased risk for behavioral and emotional health problems along with associated psychiatric disorders especially anxiety, depression, inattention and social difficulties (Johnson & Marlow, 2011; Strang-Karlsson, 2011). Children born at extreme prematurity have been found to experience a 50-70% higher rate of attention and behavioral problems in school despite normal IQ scores (Lynn, Cuskelly, O'Callaghan & Gray, 2011). Even children born near term (34-35 weeks gestation) have a 36% increased risk for developmental delay or disability at kindergarten age (Rabin, 2009). Preterm born 17-year-old late adolescents have a higher percentage of psychological problems when compared to the United States national age-related statistics (NIMH, 2007a & 2007b; ADAA, 2007): 11% with ADHD compared to 3-5%; 12.1 % diagnosed with depression compared to 5%; 9.8% diagnosed with anxiety disorders compared to 3.1% (Sullivan, 2008). Thus, prematurity is associated with behavioral and emotional health issues, as well as, psychiatric disorders from preschool through adolescence age, and have risk for continued problems in adulthood.

Miller, Sullivan, Hawes & Marks (2009), reported on their prospective, longitudinal sample of 186 children, at age 12, grouped into four preterm perinatal morbidity groups (healthy preterm without medical or neurological illness, medical preterm with clinical illness but without neurological abnormality, neurological preterm with severe illness and small for gestational age preterm with or without medical problems) and healthy full-term comparison group. A variety of biological, social and physical environmental factors were measured utilizing a battery of tests and neonatal medical data sources. Differences for neurological status, motor status

and health at age 12 were significant with abnormal high rates in the four preterm groups compared to the full term group. Total health outcomes of the four preterm groups were 3.4 times more likely than term births to have overall abnormal health status at age 12.

A secondary analysis of this data (Wright & Sullivan, 2011) demonstrated that prematurity measured by birth weight was associated with childhood psychiatric symptoms at age 12. Additionally, the mother's perception of their premature child's vulnerability and psychiatric symptoms correlated positively at ages 4, 8, and 12.

Sullivan, Msall & Miller, (2012), found a higher percentage than the United States' statistics for psychological problems in their age 17 cohort of the study related to attention deficit hyperactive disorder (ADHD), depression and anxiety. This prospective study reported on the same cohort at age 17 consisting of 215 infants born between 1985-1989 with preterm birth weights <1850 grams and grouped by neonatal morbidity then compared them with a full term group. Outcomes of functioning and disability included body functions, body structures, activities and participation. Contextual factors were identified according to the World Health Organization International Classification of Functioning (The ICF Model, WHO, 2002). At age 17, 180 of the 215 adolescents, completed the standardized assessment process that analyzed health, neurological, chronic conditions, psychological, environmental-socioeconomic, personal-neonatal morbidity and gender status. Overall results indicated that physical long-term effects of prematurity were not only confined to infants with very low or extremely low birth weight but also included small for gestational age and preterm infants without neonatal complications. Higher

percentages of psychological problems, 11% ADHD (4.1%), 12.1% (5-8%) depression, and 9.8% (3.1%) anxiety disorders were found.

Emerging Adults

The age period between 18-25 years old is now termed the period of “emerging adulthood” due to cultural delays in reaching developmental milestones (Arnett, 2013). In the past, these ages were considered part of young adulthood, when the taking on of adult roles were not delayed. Emerging adulthood, is now a separate category in the life span characterized by: identity exploration, instability, self-focus, feeling in-between adolescence and adulthood and feeling hopeful about future possibilities (Arnett, 2013). More importantly, emerging adults do not exist in all cultures and only exist in cultures (middle income) that allow the putting off of adult roles and responsibilities (Arnett, 2013).

Recent national behavioral trends in transitioning into adulthood have shown delays in traditional major life events such as age at marriage and parenthood, instability of residence, higher rates of enrollment in college education and a decline in emerging adults working for pay (Ingels, Glennie & Lauff, 2012; Arnett, 2013). Arnett (2000 & 2013) characterizes the self-views of emerging adults, in addition to not perceiving themselves as an adolescent or an adult, as also not fully accepting responsibility and not making independent decisions. This may be especially difficult for those who were born prematurely. In addition during the transition from adolescence (ages 10-18) into emerging adulthood (ages 18-25) extensive related endocrine system changes occur and influence bodily processes (Arnett, 2013). This turbulence in endocrine hormones, involving all brain structures, adds to the life

experiences influencing brain growth and emotional health. An emerging adult born premature may not have the flexibility or adaptability of the HPA axis responses to achieve allostasis and resiliency.

Globally emerging adults are experiencing life as less meaningful and health professionals are increasingly concerned about emerging adults negative behavioral choices to deal with stress as a way of coping (Hutchinson, Stuart & Pretorius, 2007). American adolescents and emerging adults have a higher rate of risk behaviors than other countries (Arnett, 2013). Additionally, emerging adult college students were more likely than older students to become angry or hostile about negative life events instead of becoming more anxious and depressed (Jackson & Finney, 2002).

Emerging adults born premature who may have physical difficulties, learning problems and limitations in social skills have an additional level of coping complexity during this developmental period (Sullivan, 2008). Considering these challenges at age 23, it may be expected that prematurely born emerging adults will have difficulty coping with adult stressors.

During emerging adulthood, exploration and changes occur that often lead to lasting life choices (Arnett, 2000) with stressors, coping styles and neurophysiological responses of these life choices effecting overall health (Lovallo, 2005; Somerfield & McCrae, 2000). Differences in stress exposure, appraisals of stress and coping styles have been identified in adults with immune system disorders, cardiovascular, depression disorders, and include a variety of physical and mental diseases (Cohen et al, 2007; McEwen, Gray & Nasca, 2015; Segerstrom & Miller, 2004). Anxious adults with comorbid depression have been found to use more emotion oriented

coping than individuals without a comorbid diagnosis (Man, Dugan, & Rector, 2012). The role of avoidance coping has been associated with the generation of stress that a decade later leads to depressive symptoms (Holahan, Moos, Holahan, Brennan & Schutte, 2005).

Given that higher percentages of psychological problems were found at the age 17 original study time point of both gestationally small and premature infants and that growth of the brain was not fully completed, then obtaining stress, coping, emotional intelligence and emotional health measurements by examining the age 23 cohort will provide additional developmental data.

Stress and Coping

Multiple definitions of stress and stressors exist and are often criticized as being “circular, formless or varied (Aldwin, 2009)”. Indeed, stress is defined according to each multiple discipline perspectives and applied to divergent topics (Aldwin, 2009). Stress definitions have also been classified as: a response (Styles: physiological), a stimulus (Holmes & Rahe: adaption to life events), a transaction (Lazarus & Folkman: appraisals and coping), a dynamic process, state-like or trait-like and acute or chronic (Rice, 2012; Lyons 2012; Butler, 1993).

Acute and chronic psychological stress in this study are defined by the DOHaD theory. Acute stress is characterized by the ‘fight or flight’ response and is short term with transient over arousal, gastrointestinal symptoms, muscular problems and combinations of the three stress emotions of anger or irritability, anxiety or depression (Miller & Smith, 2015). The American Psychological Association (2015) also defines an “episodic acute stress” characterized by an “*individual always in chaos, in*

a hurry, full of nervous energy and type A personalities.” Chronic stress, is the daily wear and tear of allostatic load and can lead to “*suicide, violence, heart attack, and stroke* (Miller & Smith, 2015)”.

Related to types of stress are the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5; APA, 2013) broad definition of anxiety disorders. Anxiety disorders have “*features of excessive fears, anxiety and related behavioral disturbances* (p 189)”. “*Fear is the emotional response to real or perceived imminent threat, resulting in arousal of the flight or fight response, and anxiety is the anticipation of the future* (p 189), (APA, 2013).”

Measuring stressors is often done by checklists and interviews (Gutman & Nemeroff, 2011). A number of functional tests of HPA activity have been developed including the dexamethasone (DST) suppression test, the corticotrophin-releasing factor (CRF) stimulation test and the combined (DEX/CRF) test. These tests are invasively administered or use oral administration (Gutman & Nemeroff, 2011). The Trier Social Stress Test (TSST) a reliable non-invasive psychological challenge test was developed to test HPA axis reactivity to psychological stressors (Gutman & Nemeroff, 2011). A standardized laboratory protocol involves a 10-minute public speech and a mental arithmetic test. The TSST combines both uncontrollability and evaluative threat, which are the conditions of DOHaD theory to activate the HPA axis response (Kudielka, 2008). As a result the response to TSST is both physiological and psychological including increased anxiety and negative mood (Kirschbaum, 2010).

TSST testing has shown that total plasma cortisol levels are overall higher in elderly women when compared to elderly men (mean age 67.3 years), younger men

and women (mean age 23.5 years) and without any differences in the patterns of reactivity (Kudielka, Buske-Kirschbaum, Hellhammer & Kirschbaum, 2004).

Schommer, et al., (2003) found habituation to psychosocial stressors unchanged overtime even if an individual was found to be a “high” or “low” cortisol responder to the TSST. Meaning, that the cortisol response to stress pattern remains uniform when activated with repeated stress. Additionally differences in cortisol response to TSST have been found in many psychological disorders (McGirr, Daiacu, Berlim, Pruessner, Sable, Cabot & Turecki, 2010).

Maltreated female youths (ages 12-16 year) show a different cortisol pattern in response to the TSST (McMillan, et al., 2009). Youth without a history of maltreatment showed an increase of cortisol followed by a gradual decline, in contrast to youths with a history of maltreatment, who showed no increase in cortisol in response to the TSST and no decline. Emerging adults, at age 23, who were born preterm are expected to have a pattern of salivary cortisol response of little to no increase and little to no decline after exposure to the TSST. Total cortisol levels may vary in comparison to the full term born by being higher.

Lazarus and Folkman's *enduring* model (Carver, 2011) of psychological stress and coping responses focuses on the process between the person, environment, their appraisal of and context of the event and is consistent with DOHaD theory (Lazarus, 1999; Park & Folkman, 1997). Psychological stress is what happens when a person is defeated in obtaining their goal commitment, intention or values (Lazarus, 1999). Stress mediating processes include coping responses defined as problem and emotion focused resulting from the primary and/or secondary appraisals used and are

considered psychologically effective if they work for the individual (Lazarus, 1999).

Moos and Holahan, (2003), added dispositional (inclined to cope) factors resulting in two orientations: focus and method of coping. The focus is either approach or avoidance while the methods of coping are cognitive and behavioral (Billings & Moos, 1981; Moos, 1995; Moos & Holahan, 2003). Approach coping is defined as problem solving attempts to deal with and resolve stressors while avoidance coping is defined as emotion focused attempts to avoid dealing with, thinking about, and managing emotions associated with the stressor (Moos & Holahan, 2003).

Combining the focus and methods of coping results in eight coping subset styles listed in Table 1 and defined in Table 2, and are measurable with standardized self-report instruments.

Moos and Holahan's definition of coping is one of three models (Aldwin, 2009; Rice, 2012). Two of the three other models explaining coping are: the environmental system, addressing ongoing life stressor and social resources, and the personal system involving individual demographics (Rice, 2012). The third model, transactional, incorporates personal and contextual factors of the stress situation with coping skills that affects the psychosocial functioning and maturation at the individual's next stage of development (Rice, 2012). Determinants of coping responses are the types and the severity of the stress, social resources and demographics (Rice, 2012). Gender, age, education, SES, cognitive abilities, problem-solving skills and personal control or regulation will effect both the focus and method of coping.

Table 1

Coping Styles

Type of Coping	Approach Coping	Avoidance Coping
Cognitive	Logical Analysis	Cognitive Avoidance
	Positive Reappraisal	Acceptance/Resignation
Behavioral Rewards	Seeking Guidance and Support	Seeking Alternative
	Problem Solving	Emotion Discharge

Table 1. From “Dispositional and Contextual Perspectives on Coping: Toward an Integrative Framework,” by R. H. Moos and C. J. Holahan, 2003, *Journal of Clinical Psychology*, 59 (12), p.1391. Copyright 2003 Wiley Periodicals, Inc. Adapted with permission.

Table 2

Definitions of Coping Styles (Moos & Holahan, 2003)

COGNITIVE APPROACH COPING	Combination of logical analysis and positive appraisal strategies
Logical Analysis	Attention to one aspect of the situation Utilizing past experiences Thinking about possible actions and their consequences
Positive Appraisal	Accepting situation and restructuring it in a favorable way

BEHAVIORAL APPROACH COPING	Dealing directly with situation through concrete actions
Seeking Guidance and Support	Seeking guidance and support from others
Problem Solving	Actions derived from the approach strategy, and organized information about the problem along with the allocation of resources, monitoring progress and evaluating results (Cherry, 2013)
<hr/>	
COGNITIVE AVOIDANCE COPING	Combination of cognitive avoidance and acceptance or resignation.
Cognitive Avoidance	Minimizing or denying the situation and/or consequences
Acceptance/Resignation	Accepting the situation and/or consequences and deciding it cannot be altered.
<hr/>	
BEHAVIORAL AVOIDANCE	Involves seeking alternative rewards, venting feelings and/or engaging in risky behaviors.
Seeking Alternative Rewards	Replacing losses with involvement in new activities resulting in an alternative source of satisfaction
Emotional Discharge	Openly venting feelings of anger and

frustration

Behaviors that temporarily reduce tension

involving acting impulsively and/or doing

something risky

Table 2. From “Dispositional and Contextual Perspectives on Coping: Toward an Integrative Framework,” by R. H. Moos and C. J. Holahan, 2003, *Journal of Clinical Psychology*, 59(12), p.1391. Copyright 2003 Wiley Periodicals, Inc. Adapted with permission.

Gender Differences in Stress and Coping

The American Psychological Association (APA, 2010) reports differences in the way males and females react to, manage, and view their ability to deal with stress. In general: females report more stress than males (especially married females), more physical symptoms of stress, and identify money and economy more often as sources of stress while males report work more often as a source (APA, 2010).

Matud (2004) examined stress and coping differences between genders and found outcomes consistent with decades of past research. There were no significant differences in life event frequencies experienced between genders, yet females reported life events as more negative ($p = <0.05$), less controllable ($p = <0.01$) and also reported more chronic stress ($p = <0.01$) than males. The major stressors for females were family and health related while males' stressors were focused on relationships, finances and work events. Females utilized emotional and avoidance coping styles more frequently while males used less emotional coping ($p < 0.001$). Females experienced more psychological distress ($p < 0.001$) and somatic symptoms ($p = <0.001$) than males.

Tamres, Janicki and Helgeson (2002) in their meta-analysis of gender coping differences reviewed 50 English language empirical studies between the years of 1990-2000, with actual stressor measurements rather than hypothetical situations, to examine coping responses. The authors categorized the studies' definitions of coping behaviors into problem-focused and emotional-focused with avoidance, approach-based and other components. Females showed a wider repertoire of types of coping behaviors with higher usage of most types than males. Significantly, females used more verbal, social support seeking, ruminating and positive self-talk than males. A few differences between genders were found with females more often than males using avoidance responses for coping with other's health stressors while males used avoidance responses for coping with relationship stressors.

Gender differences in coping patterns at emerging adulthood (ages 18-25 years) have been found to reflect established cultural qualities (Davis, Burleson & Krusewski, 2011; Kim & Sasaki, 2014) with endorsement of masculinity as a significant predictor in problem-oriented coping (Lipinska-Grobelny, 2011). A recent decline in female gender depressive symptoms in emerging adults is thought to be associated with greater autonomy and empowerment (Frye & Liem, 2011) although in general, females had been found to have higher emotional coping than men (Durm & Glaze, 2002).

DOHaD, Stress and Coping

DOHaD theory focuses on the HPA Axis physiological and biochemical mechanisms and how these interact, as well as, the resultant effects on higher nervous system processes such as behaviors, cognitions and emotions. Importantly, DOHaD

theory defines stress as a real or perceived threat (physical or psychological), a stressor as an aversive stimuli and the stress response as the activation of numerous complex body systems through the HPA axis. Accordingly, stress, coping responses and affective states are thought to influence physical pathology by direct and indirect

Figure 6

Relationship of Hippocampus to HPA Axis

Figure 6. Reprinted by permission from Macmillan Publishers, Ltd: Nature Neuroscience, "How Adversity Gets Under the Skin" by Steven E Hyman, 2009, *Nature Neuroscience*, 12, 241-243. Copyright 2009 by Macmillan Publishers, Ltd.

influences on the biological processes and behavioral responses with the HPA Axis seen as the gateway (Cohen, Janaki-Deverts & Miller, 2007; Heindel & Vandenberg, 2015; Lovallo, 2005; Somerfield & McCrea, 2000). The roles of allostasis and

allostatic load above all, is the link provided as a mediator of the accommodation to the stressor, being that system-wide physiological changes are made and the HPA Axis interconnects with other brain areas including the prefrontal cortex (PFC) to fit or adapt to the situation (McEwen, Nasca & Gray, 2015; McEwen, 1998, Ganzel, Morris & Werthington, 2010), (See Figure 6). Figure 5 illustrates “*the HPA axis under the excitatory control of the amygdala and inhibitory control of the hippocampus* (Hyman, 2009)”.

Importantly, studies of individuals experienced in meditation, show related inverse changes in functional brain imagery and mapping of brain activity (Hoffman, et al., 1982). When relaxation increases, heart rate, blood pressure and hippocampus signal activity decreases (Hoffman et al., 1982). Individuals with years of meditation experience also show thickening of the frontal cortex of the brain (Lazar, et al., 2005). The researchers think genomic changes are taking place from meditation relaxation since, in addition to sympathetic nervous system responses, chemical changes related to gene changes had been identified. The gene changes are believed to be a counterpart of the stress reduction response (Dusek, et al., 2008).

The resultant system-wide and HPA-Axis responses to stress (appraisal of threat) and the stressor (aversive stimuli) allows for the inclusion of appraisal, cognition and emotional states as key elements of the stress process. Hence, DOHaD theory advances an integrated model of stress through multilevel biomedical and psychosocial models of stress (Ganzel, Morris, & Werthington, 2011). Inherent in dealing with stress are the efforts to manage the potential and threatening aspects of the situation or circumstances, whether physical or psychological, and any detrimental

consequences to health (Moradi, Pishva, Ehsan, Hadadi, & Pouladi, 2011).

Psychologically, coping is defined as changing cognitively and behaviorally to manage internal or external demands that extend beyond the persons existing resources (Lazarus & Folkman, 1984).

Emotional Intelligence

A variety of emotional intelligence definitions were found in a systematic literature review, using the keywords “emotional intelligence”, from the Cumulative Index of Nursing and Allied Health Literature (CINAHL), Cochrane Library, ProQuest Dissertations, PubMed and the Web of Science. The majority of publications were from the disciplines of psychology and business with proliferation of the topic in psychology beginning in the early 2000’s (Matthews, Zeider & Roberts, 2004). Recently, the nursing literature on EI has shown an interest in the epistemology, application to educational and the professional competences uses of the construct (Por, Barriball, Fitzpatrick & Roberts, 2011; Akerjordet & Severinsson, 2010; Smith, Profette-McGrath & Cummings, 2009; Freshwater & Stickley, 2004; McQueen, 2004). A large body of literature exists in the business arena where EI is extensively used in hiring, employee assessments, promotions and education of managers (Ashkanasy, Ashton-James, & Jordan, 2004; Freedman, 2010).

One aspect of EI in which all agree is the involvement of an emotional awareness of self and others and emotional regulation or management (Armstrong, Galligan, & Critchly, 2011; Matthews, Zeider & Roberts, 2007 & 2004; Bar-On & Parker, 2000). A broad common definition of EI is the capacity to identify, process and manage emotions to contribute to a successful life (Armstrong, Galligan, & Critchly, 2011).

Emotional regulation is used interchangeably with emotional management and is defined as the conscious or unconscious control of emotion, mood, and affect that is most commonly achieved through coping strategies (Kowalczyk, 2015; Thompson, 1994). The construct of EI developed out of the research on intelligence to bridge a gap in the role of emotions and is widely used today in education and business settings (Mayer, Salovey, & Caruso, 2004).

Multiple studies have indicated a relationship between stress, coping styles, EI and emotional health (Por, Barriball, Fitzpatrick & Roberts, 2011; Tricky, Far hall, Wertheim, Hinch & Ong, 2011; Ciarrochi, et al., 2002; Gerits, Derkson, Verbruggen & Katzo, 2005). Studies have found that individuals with high EI report less perceived stress, have better health and report feelings of well being (Ciarrochi, et al., 2002; Hertel, Schutz & Lammers, 2009; Pau and Croucher, 2003). Individuals with low EI reported more stress and difficulties with coping (Gohm 2005; Schutte, Malouff, Thorsteinsson, Bhullar, & Rooke, 2007). Ciarrochi, Deane & Anderson, 2000, found stress associated with higher reports of depression, hopelessness, and suicide ideation in college students ($N = 302$) with high emotional perception scores and found more suicide ideation in college students who scored low in managing others emotions.

The reciprocal relationships between stress, coping and the hippocampus and how it relates to emotional intelligence warrants further investigation as recent studies are finding smaller hippocampal volumes in individuals with long-term Post Traumatic Stress Disorders (PTSD), (Woodward, et l., 2006), (See Figure 6). Impairment has been found in fear responses of individuals with PTSD involving dysfunctional

activation of the HPA Axis and especially within the ventromedial prefrontal cortex (Milad et al., 2009). EI can predict health functioning as well as distress and traumatic stress (Singh & Sharma, 2012). Additionally, oxidation of cells occurs from psychosocial stress and reflects an increase in cell aging (Fricchione, 2015). Singh and Sharma (2012) also found high levels of salivary cortisol improve coping strategies in individuals with high EI while Tang, et al., (2007) found a significant decrease in stress-related cortisol with 5 days of 20-minute meditation improving attention and self-regulation.

Infants born prematurely have a variety of health impairments and a prevalence of neurodevelopmental disabilities in general (Allen, Cristoalo & Kim, 2011). Attention issues and ineffective executive functioning has been found in premature low birth weight infants suggesting that regulation of these functions in the cerebral cortex may have been impaired (Kessenich, 2003). This area of the brain is also utilized in regulating emotions, interpreting and organizing information for responses which influences the level of an individuals' EI (Contrada & Baum, 2011). Premature emerging adults who may have perinatal injury and experienced sensitive time periods of stress effecting the HPA Axis, then are exposed to increasing vulnerabilities to stress, coping, and allostatic load during this time period, may have responses that negatively effect EI (Allen, Cristofalo & Kim 2011; Hack, 2009; Ozer & Benet-Martinez, 2006). EI studies of emerging adults, ages 18-25 years old, are scarce and the majority of studies are cross-sectional with mixed adult age ranges of the participants. Current measurement of the abilities to lead a successful life often are functional and relate to developmental milestones determined by age-appropriate

roles, succeeding in schoolwork, succeeding in the work environment, social maneuverability and forming intimate relationships to mention a few (Sullivan, Msall, & Miller, 2012). Given the identified vulnerabilities of prematurely born infants then measuring and describing EI in this population at age 23, including gender differences, may lead to further understanding of possible predictive functions, critical time periods of risk, protective factors and types of effective clinical interventions. *“What is not known is what are the long-term outcomes for adults who were born prematurely and how does stress, biology, caregiving and social factors over a lifetime effect the developing premature born infant?”* (Sullivan, 2008-2013)”.

EI Theoretical Perspectives

Historically, the modern origins of EI can be traced back to John Dewey (1902) “*moral motive*” and then Thorndike’s 1902 conceptualization of “*social intelligence*”, which at a rudimentary level, involves the ability to understand and manage other people while getting along socially (Cantor, 2000; Zeidner, Matthews & Roberts, 2009). Social intelligence was later eclipsed by interest in EI that included differentiating between cognitive, emotional and other factors. EI began to capture scientific interest in academic journals during the early 1990’s through publications by Mayer and Salovey’s model as a way of joining emotions to intelligence studies (Mayer, Salovey & Caruso, 2000). Culturally, Goleman popularized EI in the 1995 publication of the book *Emotional Intelligence* that integrated emotions with intelligence. The importance of both emotion and reason intuitively appealed to the public and some in the scientific community (Matthews, Zeider and Roberts, 2004). Although the scientific community is studying EI there is not agreement on whether it

is a scientific endeavor or if there is a science of EI (Zeidner, Matthews & Roberts, 2009). A discussion of the theories, measurements, and applications of EI to this controversy follow.

The term EI is used in three ways (Mayer, Salvey, & Carusi, 2000): 1. The popular meaning is that reason and emotion can be personally integrated and an individual can achieve self-improvement. 2. To describe personality traits through connecting parts of the mind to life outcomes from mental mechanisms, models of self, and self-relevant and general traits. 3. To define a set of abilities dealing with processing emotional information. Three theorists have influenced the development of EI knowledge, the use in practice, and research across multiple disciplines: Daniel Goleman, Mayer & Salovey, and Raven Bar-On (Smith, Profetto-McGrath & Cummings, 2009).

Goleman's theories developed from the business sector and popularized the concept of EI through use of work competency measures that covered a broad range of personality measures (Mayer, Salovey & Caruso, 2004). EI to Goleman consist of five parts: “*knowing emotions, managing emotions, motivating oneself, recognizing emotions in others, and handling relationships*, (Goldman, Boyatzis and Hay Group, 2005).” The five parts have been divided into 25 different competencies and include mapping of these competencies. Goleman's model is a combination of theoretical concepts and construct derived from multiple psychological perspectives resulting in a popular, trait and ability mixed model. The model has wide spread popular appeal and is viewed as over inclusive by some in the scientific community. Goleman, Boyatzis and Hay Group (2005) developed the self-report, Emotional and Social

Competency Inventory (ESCI), to assess the emotional and social competencies of outstanding leaders and provided guideline for using the ESCI. Courses and certifications in EI are also available. Extensive construct and validity studies within business organizations worldwide have been conducted. Overall, reliability has shown high internal consistency with limited evidence for test-retest reliability. EI has good construct validity with a variety of personality constructs (Wolf, 2005).

Mayer, Salovey & Caruso, 2000, ability model views EI as operating across both cognition and emotional systems as a “*unitary intelligence*” (Mayer, Salovey & Caruso, 2000; DeFabio & Saklofske, 2014). Emphasis is on EI as a “*concept of an intelligence that processes and benefits from emotion*” and is composed of “*mental abilities, skills or capacities*”. The unifying system has 4 branches: emotional perception (perceive, attend, decipher and express), emotional integration (uses cognitive system), emotional understanding (understanding and reasoning) and emotional management (flexible guidelines to manage emotions). EI is conceptualized as a mental ability and measured with objective tasks. The Mayer-Salovey-Caruso Emotional Intelligence Test (MSCIET) measures EI by having the individual perform tasks and solve emotional problems in 4 areas: perceiving emotions, facilitating thought, understanding emotions and managing emotions (Mayer, Salovey & Caruso, 2004^b). The original measurement had criticisms relating to scoring and reliability and a second version addressing some of these issues are now in use (Mayer, Salovey, Caruso & Sitrenious, 2001).

Bar-On, 1997, generally classified as a trait model and by some others as a mixed model defines EI as an “*array of non-cognitive capacities, competences and*

skills that influence one's ability to succeed in coping with environmental demands and pressures" (Bar-On, 1997; DiFabio & Saklofse, 2014; Por, Barriball, Fitzpatrick & Roberts, 2011). Bar-On and Parker (2000) view the key elements of EI as stress management and the ability to adapt. The self-report trait EI, in this study, was assessed with the Bar-On EQ-I. The 133 likert-like item questionnaire provides a total EI score ($M = 100$, $SD = 15$), and five composite scores for the principle dimensions with 15 related content scale scores (Bar-On, 2002 & 2006; Di Fabio & Saklofske, 2014; De Weerdts & Rossi, 2012). The five composite score areas are: intrapersonal, interpersonal, adaptability, stress management and happiness (Bar-On, 2002 & 2006; Di Fabio & Saklofske, 2014; De Weerdts & Rossi, 2012). The 15 content scale scores are: emotional self-awareness, assertiveness, self-regard, self-actualization, independence, empathy, interpersonal relationship, social responsibility, problem solving, reality testing, flexibility, stress tolerance, impulse control, happiness and optimism (Bar-On, 2002 & 2006; Di Fabio & Saklofske, 2014; De Weerdts & Rossi, 2012). The model evolved from personality psychology and is measured through a standardized self-report test that assesses self-perceptions rather than actual abilities (Mayer, Salovey, & Caruso, 2000). Studies show the EQi has a significantly stronger association with mental health than other measures (Gohm 2005; Schutte, Malouff, Thorsteinsson, Bhullar, & Rooke, 2007). Raven Bar-On EQi test is used in this study to assess EI, stress management, adaptability and general mood.

A major issue and controversy with EI definitions is they vary so determining what processes, behaviors and outcomes are related makes operationalization of the

construct difficult if not possible and limits hypothesis testing (Bar-On & Parker, 2000). A construct is deliberately and consciously invented or adapted for a specific purpose and identifies what meets inclusion or exclusion by categorization (Mishra, 2013; WSU, 2013). In addition, constructs serve as an indirect link between the abstraction defined and observed manifestations, in contrast to concepts which are directly linked with observation (WSU, 2013). Construct validity includes assessing the test content, response processes, internal structure, association with other variables and consequences of its use (Furr & Bacharach, 2014). Construct validity in EI is, at best questionable, as definitions of EI measurements are based on multiple theories and not on an accurate and agreed upon representation of the entire domain (Stough, Saklofske, & Parker, 2009). As with many constructs in the social sciences, face validity and content validity refers to the scores derived from each test item and not the test, and is often also an issue with other social science constructs (Stough, Saklofske, & Parker, 2009). Conceptualization of EI on face validity is appealing yet construct validity across measurements has not been achieved. Research has shown that ability based and self-reported trait EI tests are not correlated yet differing models of trait self-reported EI were found to be correlated despite describing similar and not identical constructs (DiFaboe & Saklofske, 2015). EI developed from personality theories and maybe redundant with personality constructs (Stough, Saklofske & Parker, 2009).

DiFaboe & Saklofske, 2014, administered a battery of eight tests to assess emotional intelligence (including Raven Bar-On), self-evaluating, resilience, and life satisfaction, to 164 Italian high school students, and utilized multiple regression for

the data analysis. Significant results were found for the ‘Big 5’ personality factors followed by trait self-reported emotional intelligence tests as the most predictive of self-evaluation, resilience and life satisfaction. An equally significant finding, was the higher the self-reported trait EI score then the more positive, in control, able to deal with adversity and emotional resources available were perceived by the individual.

Summary

In summary, the construct of EI, is primarily based on personality theories, and measurements lack construct validity across tests. On the other hand, EI theories are used in business, educational and health settings, and are capturing components or factors related to success in dealing with stress, coping, emotional management and getting along with others socially. The Bar-On EQi, captures emotional and psychological functioning, fits with DOHaD theory, and was utilized in this study to assess the emotional development of emerging adults and any differences between the prematurely and full term born. Although definitions of EI vary and a unified construct validity has not been achieved, EI is in wide use, and the appeal may be the ability to capture assessments of traits of a mature personality as demonstrated in the study above conducted by DiFabeo & Saklofske (2014). Additionally, emotional intelligence scores level off and usually without further gain in adulthood, and this could be related to personality development theories (Garner, Qualter, & Whitely, 2011). Thus, the measurement of EI at early or emerging adulthood is very relevant.

The Daily Hassles Questionnaire will provide a standardized measure of usual life stress events. Moos & Billings (1981) determinates of coping styles provides the

coping types and strategies used by an individual in stressful events, and carried over from any previous stressful experiences while fitting conceptually with EI (Moradi, Pishva, Ehsan, et al., 2011). Bar-On and Parker (2000) view the key elements of EI as stress management and the ability to adapt. Elements from each of these perspectives, along with coping strategies and stress responses have been implicated in health issues.

Emotional Health

Health, well-being, mental health and quality of life are similar concepts, which include biology and the individual's psychological state, to utilize goal directed behaviors and involve multiple causal networks (CDC, 2013). Emotional health is defined as an individual *"adjusting to new situations and achieving the desired outcomes"* (GUCCHD, 2016). Higher emotional intelligence maybe related to better emotional health and is linked to aspects of better psychosocial functioning (Schutte, Malouff, Thorsteinsson, Bhuller & Rooke, 2007). Drug use, being bullied and violence are a few of the behavioral risk factors that threaten emerging adults emotional health globally (Lister, Merrill, Vance, West, Hall & Crookston, 2015; Orleans, 2008). There is a range of emotional behavior dependent on an individual's adaptation to stress and coping style, resulting in effects on feelings of well-being, functioning and health behaviors (Glanz & Schwartz, 2008).

Childhood physical and emotional abuse is related to childhood psychiatric disorders and also effects cortisol (HPA) stress reactivity (MacMillan, Tanka, Duku, Vaillancourt & Boyle, 2013; MacMillan, et al., 2009). Children and adolescents mental health, according to the CDC (Perou, et al., 2013) is characterized by meeting

developmental and emotional milestones, social development, effective coping skills, and being able to function at home, school and other areas of their life. A similar definition of emotional health exists for emerging adults (Arnett, 2013). A number of studies of emerging adults show their identified markers of transition are: *“accepting responsibilities for oneself, making independent decisions and becoming financially independent”* (Arnett, 2013; Nelson, 2003).” Additionally, social and emotional loneliness is common and often used for self-reflection and mood management (Arnett, 2013). High EI scores were associated social functioning, mental health, and vitality (Rey & Extremera, 2013). EI has been found to have a moderate positive relationship with coping styles and buffered stress in work situations (Gujral, 2013).

Health experts have agreed that most physical problems begin in the early teens through the early 20's and relate this to behavior patterns developed during this time period (Arnett, 2013). As a result, these patterns of behavior, become established and effect adulthood responses. Health educational programs, directed at risky behaviors have shown mixed results (Arnett, 2013). Effective psychosocial protective factors have been identified, such as exercise that improves mood, weakens the stress response and is thought to promote neurogenesis, while reinterpreting negative stress stimuli through cognitive positive reframing involves memory, control of emotion, and underlying neurobiology (Feder, Nestler & Charney, 2009).

Tsaousis & Nikolaou, 2005, through two studies ($N = 365$ & $N = 212$) with mean age ranges of 25-36 years old measured: 1. EI and general health in one group and 2. EI, general health and health related behaviors (smoking, drinking, and exercise) in another group. EI was found to be negatively associated with poor general health,

negatively correlated with smoking and drinking and positively correlated with exercise.

Burnett, et al., in 2011, conducted a meta-analysis of peer-reviewed research article findings from 1995-2010. The purpose was to look at “*the prevalence of psychiatric diagnoses in preterm and full-term children, adolescent and young adults*. (Burnett, 2011).” Of 719 articles only 5 passed inclusion and exclusion criteria resulting in 734 preterm and 634 full term (controls) individuals. The study reported high odds ratio of risk for anxiety, depression and other psychiatric disorders in the prematurely born.

England’s young adults (between the ages of 18 and 25 years old) were found to have resilience-related coping skills by utilizing social support when compared to older adults (ages > 64 years old) who were more resilient and utilized emotional regulation and problem solving (Gooding, Hurst, Johnson & Tarrier, 2011). EI research has shown an association with better health and is a plausible health predictor (Martins, Ramalho & Morin, 2010; Schutte, et al., 2007).

Adolescents with visible or invisible chronic health conditions (disabilities) were found to do less well in several psychological study outcomes than adolescents without disabilities (Wolman, Resnick, Harris, & Blum, 1994). One similar invisible condition is Alexithymia, broadly defined “*as an inability to express one’s feelings* (Merriam-Webster Medical Dictionary, 2015)”. Some researcher postulate this condition is due to deficits in cognitive processing of emotions resulting in undifferentiated poorly regulated emotions and representative of the opposite of emotional intelligence (Velasco, Fernandez, Paez & Campos, 2006; Taylor, Bagby &

Parker, 1991). Recent research developments have associated alexithymia with maladaptive emotional regulation, low EI, reduced rapid eye movements, somatic illness, and disease development (Taylor, 2000; Taylor, Bagby & Parker, 1991).

Given the potential for disabilities due to HPA axis, emotional dysregulation in emerging adults born prematurely, coupled with previously mentioned higher incidences of psychiatric disorders, then a range of developmental functioning, well-being and psychopathologies are possible. Indeed, if self-emotional regulation is a part of EI, then the prematurely born are at risk for self-regulation and mental health disorders especially Attention Deficit Hyperactive Disorder (ADHD), (Sullivan, personal communication, November 11, 2014).

Summary

In this study measures for assessing stress responses, coping types, emotional intelligence and emotional health are utilized. The TSST provided physiological responses by inducing a moderate stress situation along with salivary cortisol levels to assess HPA regulation. The DHS provided self-reports of everyday stress experiences as representative of developmental milestones as well as stress magnitude scores in all perinatal birth groups. The Bar-On EQi (1997), total EQi score indicates the emotional intelligence levels in the perinatal birth groups. The ASR provides a emotional health score for all participants. Comparisons of emotional health and stress reactivity of salivary cortisol levels determine if any differences between perinatal birth groups exist.

HPA Axis and the Stress Response

The stress response consists of activation of two brain pathways triggered by the hypothalamus. The first pathway involves the autonomous nervous system and release of epinephrine causing the physiological response of fight or flight. This pathway's response is described by Selye's theory of adaptation to stress definition of immediate responses (Arnold & Kverno, 2009).

The second pathway involved in the stress response is the anterior HPA Axis. Stress, directly and indirectly affects the HPA Axis, initiating the stress hormonal response at the hippocampus level (Figure 7). The primary hormone regulating this axis is the glucocorticoid, cortisol (Edmunds & Mayhew, 2009).

The HPA Axis, consisting of the hypothalamus, pituitary and adrenal gland, regulates cortisol secretion from the adrenal glands whenever we are stressed and produces a physiological response (Bruyere, 2009). Corticotrophin-releasing hormone (CRH) from the hypothalamus acts as a chemical messenger to the pituitary gland. In turn the pituitary gland responds to the CRH and releases adrenocorticotrophic hormone (ACTH) into the blood stream. The circulating ACTH in the bloodstream reaches the adrenal gland that then produces and releases cortisol. The cortisol circulates in the bloodstream until the demand for it is met. Physiologically, there is a cascade of effects including increased cardiac output, pupil dilation, and shunting of blood from the digestive tract and kidneys to vital organs ultimately resulting in decreased fluid loss, increased glucose, and decreased brain nor-epinephrine. Once the demand is satisfied the cortisol signals the pituitary to

stop producing ACTH.

Figure 7

Corticotropin Releasing Factor System

Figure 7. Reprinted with permission by Sage College. In W. Lovallo, (2005), Stress and health: Biological and psychological interactions (p 116). USA: Sage Publications. Copyright 2005 by Sage College.

The production of cortisol in the HPA axis is regulated through a negative feedback loop system:

1. Low serum cortisol levels activate the pituitary gland to release

adrenocorticotrophic hormone or acetylcholine (ACTH).

2. ACTH stimulates the adrenal cortex to increase cortisol.
3. A high level of serum cortisol decreases ACTH production and results in a decrease of cortisol production (Edmunds & Mayhew, 2009).

When the HPA Axis is functioning effectively the endocrine response will be quick and adaptive. If the HPA Axis is exposed to abnormal allostatic loads, defined as repeated stress, lack of an adaptive response, or an inadequate response then dysregulation occurs or allostasis is not achieved, resulting in variations in the cortisol levels (Barker, 1990 & 2007).

Cortisol is the end product of the HPA Axis chemical transactions (de Weerth, Zijl & Buitelaar, 2003). Cortisol levels follow a circadian or diurnal pattern in normal adults totaling a secretion level of 10 mg. a day (Edmund & Mayhew, 2009). Cortisol levels are highest in the early morning, peak 60 to 90 minutes after awakening, and are lowest from evening to midnight (Edmund & Mayhew, 2009).

The processes involved in the HPA Axis negative feedback loop are complex and influenced by a number of multifaceted variables. The timing of sampling during this process of cortisol circulation will affect the interpretation of results nonetheless standardized timeline comparisons and interpretations are available for adults and children (de Weerth, Zijl & Buitelaar, 2003).

The cortisol feedback loop, also involves circulation to the frontal cortex and limbic areas, which effects affective responses and past experiences (Lovallo, 2005). Differences in the frontal-limbic system as part of the central cortisol feedback system

will in turn influence the differences in the stress response (Lovallo, 2005).

Summary

In summary, cortisol is a key stress response glucocorticoid within the HPA Axis and prefrontal cortex negative feedback loop response. Cortisol can be accurately measured in the saliva as well as the blood, and patterns of responses are standardized. Changes in HPA Axis responses may help identify periods of transition into disease states. Salivary cortisol, in this study, was measured to obtain the HPA stress response pattern in a sample of former premature infants and a term born comparison group at age 23 years. The cortisol response was examined in association with self-reported stress and coping patterns, as well as emotional intelligence and emotional health.

HPA Axis Function in Prematurity

Prematurity rates in the United States contribute to national health care issues and costs both in the short and long term. The rate of premature birth for two and one-half decades, from 1980-2006, increased by 20% (Martin, Osterman & Sutton, 2010). Twelve percent of all births in 2010, were premature, totaling 523,033 births for the year (CDC, 2010). Currently the mortality rates for premature newborns with low birth weights has decreased by around 2% due to newer preventative and treatment approaches, but long-term morbidity remains (Child Trends Databank, 2011). This decrease in mortality rates has increased the rates of immediate preterm birth consequences such as: adrenal insufficiency, intraventricular hemorrhage, patent ductus arteriosus, respiratory distress syndrome, bronchopulmonary dysplasia, necrotizing enterocolitis and retinopathy of prematurity (March of Dimes, 2008).

The long-term outcomes for these infants of decades of premature birth will continue to influence social, educational, community, and family systems well into the future. Prematurity in general has multiple immediate birth-related health issues, and requires continued vigilance throughout childhood (Saigal & Doyle, 2008; Sullivan, et al., 2008; Hack, Taylor, Drotar, Schluchter, Carter, Andreias, et al., 2005; Saigal, Burrows, Stoskopf, Rosenbaum, & Streiner, 2000). More recently, prematurity effects are proposed to contribute to the development of adulthood illnesses (Hack, 2009; Saigal & Doyle, 2008).

In addition to the specific issues surrounding prematurity, the study of cortisol levels as an indicator of the HPA Axis functioning in those born at term has implications for application to health issues. Identifying any difference between premature and full term birth HPA Axis functioning by screening for alterations in cortisol levels in combination with stress, coping, emotional intelligence and emotional health measurements may identify vulnerable individuals of all birth group types before expression of any variations (Dedovic, Duchesne, Dager & Pruessner, 2010).

Cortisol levels in newborns have been hard to standardize due the rapid shifting of development, instability of the newborn system and brain volume growth of 15mL/week between 29 and 41 week's gestation (Main, 2010). No normal range of preterm cortisol levels exist because of a variety of fluctuation in preterm and full term physiological homeostasis (Ng, 2011). There is agreement that around 1 year of age preterm cortisol levels begin to stabilize into a pattern approximating the adult diurnal cycle (de Weerth, Zijl & Buitelaar, 2003). Under normal conditions, with full term

infants, cortisol levels reveal a circadian rhythm with the adult pattern of diurnal decline from morning to evening at around 3 months of age (Turner-Cobb, 2005).

Attempts to collect salivary cortisol levels from newborns have met with issues of lack of sufficient saliva quantity and concerns about interference of administered sweet solutions (Morelius, Nelson & Theodorsson, 2004). Morelius, Nelson & Theodorson (2004), successfully obtained 113 preterm and term infant's saliva using less than a 10mL sample. In combination with altering the detection limit of the radio-immunoassay they were able to analyze very low concentrations of cortisol in newborns without interference from oral glucose solutions.

Jansen, Beijers, Riksen & de Weerth (2010), analyzed 48 peer reviewed empirical studies (1978-2008) providing pre and post stressor cortisol levels at various ages. In the first 13 weeks or 3 months post-natal the mean cortisol reactivity effect to painful stimuli was the highest and continued to decline with advancing infant age groups (3 & 6 months, 6 & 12 months and 12 & 24 months).

Tollenaar, et al. (2010), obtained daily eye swab cortisol levels from 300 infants at the ages of 6 weeks, 5 months and 12 months. The infant's cortisol levels declined over the year however intra-individual variability was large and stabilized between 5 to 8 months.

Knowing that the newborn's system, whether preterm or full term, is continuing to evolve at a rapid pace, understanding the processing of a variety of stressors during critical growth periods become paramount. Prematurity is a stressor itself due to the immaturity of the central nervous system and the neonatal course, which may include neonatal illness and a long intensive care stay. Routine medical procedures activate

the newborns stress response system to increase cortisol secretion. Physical stressors to the infants HPA Axis have resulted in subsequent moderate increases of cortisol levels (Jansen, Beijers, Risken-Walraven & de Weerth, 2010). The combination of increased cortisol levels, contributing to cell death and a failed or delayed response of the central nervous system (CNS) results in alterations within the HPA Axis or fetal programming (Sullivan, Hawes, Winchester & Miller, 2008).

Sullivan, Hawes, Winchester and Miller (2008), define the immediate effects of premature birth or neonatal programming and include individual differences such as genetic, developmental and social/care giving, which influence the neonates behavioral and neuroendocrine response (See Figure 8). This response is heightened

Figure 8

Premature Birth: Fetal/Neonatal Programming

Figure 8. Reprinted by permission Mary. C. Sullivan. 2008-2013. In “Risk and protection in trajectories of preterm infants: Birth to adulthood (Grant # NIH R01 NR003695-14).” Bethesda, MD, National Institutes of Health, National Institute of

Nursing Research.

or hypersensitive and contributes to the neonatal allostatic load. Long-term exposure to repeated negative developmental stress increases the allostatic load on the HPA Axis and if not counteracted by positive or protect factors, such as mediated by mother-child attachment security, then adult outcomes of disease occur. Sullivan, et al. (2008), attributes adulthood disease development to factors both positive and negative, defined as cumulative risk and protection, which affect long-term outcomes of functional, emotional, executive brain and work performance (See Figure 9).

In summary, early trauma with long term neurological consequences that are dysfunctional, especially when paired with the proper timing of a stressor, at a critical time in the development of a child, may result in health, emotional and behavioral consequences exhibited in adulthood.

Figure 9

Cumulative Effects of Caregiving and Social Environments Over Time

Figure 9. Reprinted by permission Mary. C. Sullivan. 2008-2013. In "Risk and

protection in trajectories of preterm infants: Birth to adulthood (Grant # NIH R01 NR003695-14).” Bethesda, MD, National Institutes of Health, National Institute of Nursing Research.

HPA Axis Function in Stress and Anxiety

Anxiety is a complex response to a stressor that includes emotional, cognitive, behavioral and physiological responses. Diagnosed anxiety disorders, which last longer than 6 months, affect 40 million adults age 18 and older each year (NIMH, 2009). Anxiety disorders commonly occur with mental and physical illnesses, as well as alcohol and substance abuse. Individuals may complain of uneasiness, fearfulness or nervousness, increased heart rate and increased respirations, to mention a few signs and symptoms.

Walter Cannon’s well-known fight or flight response, further developed later by Hans Selye, prepares the body to respond by increasing blood pressure, heart rate, cardiac output and other responses as indicated in HPA functioning noted earlier (Halter & Vascarolis, 2010). If the individual’s underlying neurological functioning or HPA Axis functioning is dysfunctional then mediating effects, positive and negative or cumulative protective and risks factors, will effect this response according to the DOHaD theory. These mediating stress effects can be from a physical, psychological, emotional, cognitive, intellectual, major life events, environmental and social/caring interactions.

If the individual’s anxiety is transient and without dysfunction then no permanent change will occur in the HPA Axis functioning. If there is an early trauma, such as prematurity, with long term neurological consequences that are dysfunctional, paired with the proper timing of the stressor during critical developmental periods of the

child, then the consequences will be evident in adulthood (Turner-Cobb, 2005). In other words, the adaptability to a stressor or anxiety rather than the initial reaction will predict long-term outcomes.

What is known during childhood, is the relationship of low birth weight in preterm births and subsequent psychopathology is coactive with psychiatric problems but not necessarily medical problems (Nomura & Chemtob, 2007). Additionally directionality of cortisol levels in childhood is known and has been determined by their behavior of engaging or withdrawing responses (Turner-Cobb, 2005). Often a child born prematurely will have a blunted cortisol response. A blunted cortisol response, is defined as a failed, less intense, less concentrated or delayed cortisol response (Bruehl, Wolf, & Covit, 2009; Sullivan, et al, 2008).

Various adult studies exist that measure cortisol levels in a variety of psychological pathologies, emergency situations, and anxiety reduction interventions (Harvey, Nathens, Bandiera & LeBlance, 2010; Rapaport, Schettler & Bresse, 2010; Yu, 2010). At the other end of the life spectrum, Lanze, Mantella, Shi, et al., (2010), report that researchers have found that one-third to one-half of elders develop generalized anxiety disorder later in life, when aging neurological processes interfere with brain cortex messages to the HPA Axis.

Although they are careful to report that multiple neurological pathways affect health, the utilization of the selective serotonin re-uptake inhibitor drug escitalopram was effective in symptom reduction and reducing cortisol levels in subjects that had diurnal pretreatment cortisol levels above normal peak and daily total levels (Lanze, Mantella, Shi, et al., 2010).

Elevation of cortisol levels in healthy adults has been found when the subject is exposed to a standardized transient psychological stress test, the Trier Social Stress Test (TSST) to trigger an acute anxiety or stress response. If a stressor or anxiety response is elicited then the corresponding standardized timed cortisol level responses reflect this change. A baseline diurnal cortisol level is obtained for comparison to the stress induced cortisol response to measure reactivity of the HPA Axis. This psychological stress induced measurement is useful to look at the level of stress or anxiety triggered (and the HPA Axis response) and the intensity level of the cortisol release.

Stress, coping, emotional intelligence and emotional health differences have been found in some studies with prematurely born children. Given that the prefrontal cortex and limbic system is part of the corticotropin releasing factor system, then involvement of these structures and function effects will also influence emotional, affective, appraisal and coping in the emerging adult (See Figure 10).

Summary

Structural differences in the brains of premature, low birth weight infants with alterations continuing into adulthood have been found by researchers (Nostarti, Murray & Hack, 2010). The concept of allostatic load and research studies related to DOHaD theory links (Sullivan, 2008-2013): “1. *Repeated infant stress responses to prematurity and neonatal experiences with increased allostatic load.* 2. *The cumulative effects of the social environments overtime with disease formation*”.

Measurements of brain pathology using a variety of biomarkers, such as magnetic resonance imaging and free saliva cortisol levels, have yielded general structural and

functional data. Salivary cortisol levels as a major endocrine marker of the HPA activation is in wide use to measure stress responses, allostatic load, allostasis, health,

Figure 10

Stress, Coping, Prefrontal and Limbic System, HPA Axis, Cortisol, Emotional Intelligence and Emotional Health

Figure 10. Adaptation from Dorine Felder with permission, (2003), "*Schematic overview of the hypothalamic-pituitary-adrenal (HPA) axis. Stress activates the HPA-axis and thereby enhances the secretion of glucocorticoids from the adrenals*". CC BY-SA 4.0 (<http://creativecommons.org/licenses/by-sa/4.0>), via Wikimedia Commons. Retrieved December 15, 2016.

disease and psychiatric pathology. (Turner-Cobb, 2005). Salivary cortisol sampling is non-invasive and easily, as well as, reliability measured.

The TSST, is another well established standardized protocol for provoking the physiological and emotional stress responses in a laboratory setting for acute stress reactions. Measurements of TSST cortisol stress responses, in early adulthood (age 23) allows us to examine differences in HPA Axis functioning between former premature and full term infants and prior to later adulthood and later aging process interactions.

Emerging adults, at Age 23, have multiple intrapersonal, interpersonal and stress adaptability reactions to master in order to reach developmental milestones and will require effective emotional intelligence and emotional health. Societal changes in when emerging adults met the traditional milestone goals and the globally observed increased risk-taking behaviors also warrant examining emerging adults coping styles. Emerging adults coping styles to deal with stress will not only influence their current emotional health yet will be carried with them into adulthood. Stress, coping, the cortisol stress response, emotional intelligence, and emotional health are all involved in obtaining developmental milestones at Age 23 for emerging adults. Results from this study will inform us not solely of current emerging adults' emotional health status, yet also importantly, if there are any prematurity effects to address that may point to risk for later adult disease.

Secondary Data Analysis

This secondary analysis is derived from the dataset of a prospective longitudinal cohort designed study of preterm and full term infants: *Risk & Protection in Trajectories of Preterm Infants: Birth to Adulthood* (Sullivan 2008-2013). The project received full IRB approval from the University of Rhode Island, Women & Infants Hospital and Rhode Island Hospital. The study also received a Certificate of Confidentiality from the National Institutes of Health. The subjects ($N = 213$) were born at Women & Infant Hospital between 1985-1989 and continually followed at 9 time points in research studies: 1 month, 18 months, 30 months, age 4, age 8, age 12, age 17 and age 23. The sample retention rate at age 23 was 85% ($N = 180$).

The original researcher's theoretical perspective was derived from DOHaD theory and the purpose was to follow preterm (with ranges of gestational age and perinatal morbidity) and full term born infants to age 23 to examine "*the impact of prematurity, medical history, and environments on achievements and deficits during young adulthood* (Sullivan, 2008)". The original study had 3 specific aims with related hypotheses that focused on determining the effects, relationships, and outcomes of young adults (age 23 years old) in terms of: health (especially cardiovascular), functional performance, emotional intelligence, executive function, work competence, growth, neurological morbidities and psychological processes. This secondary analysis adds an in-depth subset analysis of findings related to stress, coping, EI, emotional health and psychiatric disorders of the infant born prematurely at age 23 (Miller & Brewer, 2003).

Compatibility of the primary data with this secondary analysis is not an issue and

will still provide information on the differences over time between 23 year olds born pre or full term (Miller & Brewer, 2003; Yea & Niemeier, 1996). No data collection expenses will be incurred in this secondary analysis study as did in the original longitudinal study. The first wave of the recruited participants did not change over time and the retention rate (85%) and those lost to attrition at age 23 (15%) remained less than 20% allowing confidence in interpreting the differences between birth groups (Gordis, 2009; Miller & Brewer, 2003; Yea & Niemeier, 1996).

The initial cohort was well characterized and the study procedures and measurements were standardized and well defined. An “exposed” group of prematurely born and a “non-exposed” group of full term born infants comprised the two comparison groups and were selected for birth status, neonatal illnesses, gender and wide representation of socioeconomic status. The original researchers followed protocols without changes in research staff resulting in minimization of personnel effects threatening the internal validity (Miller & Brewer, 2003; Menard, 2007). As with all longitudinal studies, all factors involved over time, are not captured yet the original design accounted for common confounding variables associated with changes in socioeconomic status between birth and age 23 and gender differences (Menard, 2007).

The two time points used in this secondary analysis add a distinctive period over time and allow for identifying levels of stress adaptability, coping styles, emotional intelligence levels and emotional health status (Singer & Willet, 2003). Measurements included self-report, interview, observational coding, and standardized protocols used over the course of the longitudinal study were age appropriate, and

minimized the effects of the setting and participants' acclimation (Menard, 2007). Furthermore, the current researcher had limited involvement in the original research nonetheless had access to the principal researchers for clarification, understanding and information related to the study design, data, meaning and decision processes (Miller & Brewer, 2003)

Ethical, confidentiality issues and use of the participants' identity cleansed data was addressed in the original study consent form at all time points and are not accessible to this researcher (Miller & Brewer, 2003). Notwithstanding the availability of the original dataset, the selection and in-depth understanding of the dependent variables required extensive analysis. In addition, modeling analysis of change variables is possible with this design (Singer & Willett, 2003). What is unique about this secondary analysis is it begins with the identified preterm and full term birth groups before any manifestations of emotional issues and psychiatric disorders develop and focus on the time point of the age 23 year old participants.

Summary

This study examined all dependent variables both alone and in combination that may influence the prematurely born emerging adult in developing emotional health issues. Before the research plan for this study could be fully developed, a theoretical and concept analysis of the Developmental of Health and Disease Theory, HPA Axis functioning and Emotional Intelligence was done to clarify the concepts and constructs and provide direction in measurement. Issues in conducting a secondary analysis were reviewed. In Chapter 3 the secondary analysis and original study description, sample characteristics, methodology, and data analysis plan to investigate any

differences in emotional health between the preterm and full term born emerging adult is described.

CHAPTER 3

Methodology

This study is a secondary analysis of a larger longitudinal study aimed at determining the effects of prematurity, risks and protective processes on adult trajectories and health outcomes at age 23. The original study's theoretical foundation is the Developmental Origins of Health and Disease (DOHaD) and the variable measurements used are consistent with the purpose of this secondary analysis. This secondary study is a descriptive correlational design of the effect of premature birth and its relationship to stress, coping, emotional intelligence, emotional health and the stress response of neuroendocrine functioning at age 23. In this chapter, sample and neonatal demographics, self-report measures of stress, coping and emotional intelligence, assessment of emotional health, and timed measures of salivary cortisol through a standardized laboratory social stress paradigm, the Trier Social Stress Test (TSST) are defined. The study procedures and data analysis plans are outlined.

Research Design

The University of Rhode Island, Office of Research Integrity, determined this secondary data analysis, did not require Institutional Review Board (IRB) oversight: # 910108-1 (See Appendix L).

Sample

The infant participants were born at Women's and Infants Hospital in Rhode Island between 1985-1989, continually followed in a series of research studies supported by the National Institutes of Health (Lester, 1985; McGrath, 1989, 1994,

1998, 2003; Sullivan, 2008 - 2013). The infant sample included 213 infants in a 5-group design of: (1) full term healthy infants (FT), (2) preterm infants without neonatal illness (HPT), (3) preterm infants with neonatal medical illnesses (MPT), (4) preterm infants with neonatal neurological illness (NTG) and (5) preterm infants with small for gestational age (SGA) with and without neonatal illness, (Sullivan, 2008-2013). The original racial and ethnic composition of the 213 sampling, consisted of 105 females and 108 males, and was reflective of the population and geographical location in Southeastern New England from 1985 to 1989 (Sullivan, 2008-2013). This original sampling included varied degrees of neonatal morbidity in preterm birth. The study maintained a 97% retention rate from age 4 to 23 years (Sullivan, 2008-2013). At age 23 years, 180 subjects were recruited for participation in the 10th longitudinal follow-up study with a retention rate from birth of 85%, and 96% from the prior age point of 17 years. Two time points, birth and age 23 years, the 10th wave of this longitudinal study comprised the sample for this study.

The data (Appendix Measurements) utilized for this study will consist of neonatal data from the original five groups and demographics. Age 23 years data are, the self-report measures of the Daily Hassles (DHS-R) for stress, Coping Response Inventory (CRI-A) for coping styles, the Bar-On Emotional Quotient Inventory (Bar-On EQi) for emotional intelligence, emotional health status from the Adult Self Report (ASR) and salivary cortisol levels collected according to the established protocol from the Trier Social Stress Test (TSST).

The neonatal sample inclusion and exclusion criteria as well as the characteristics of 5-group design are well described (Sullivan, 2008-2013; Winchester, Sullivan,

Roberts & Granger, 2016). For this study, inclusion criteria included the ability to participate and complete the study protocol and measures. Any 23-year old participant with severe medical problems, such as cerebral palsy with wheelchair assist, or low intelligence with or without physical limitations was excluded. Females who were pregnant, tested positive using urine pregnancy screening, or <8 weeks post - partum were excluded from the original data collection. Additionally any 23-year old young adult with positive urine testing for substance use was excluded.

Measurements

The following measurements are proposed for the current study.

Demographic Data

Demographic data included neonatal birth data at age 23 including the Hollingshead Four Factor Index of Social Status (HH), (Holmes & Rahe, 1967). HH was used to estimate the socioeconomic status of unmarried individuals, female and male heads of households and families (Hollingshead, 1977; Gottfried, 1985). It is widely used in medicine and public health to differentiate socioeconomic status (Adams & Weakliem, 2011). The four factors examined consist of: education, occupation, sex/gender and marital status that are used to derive a mathematical estimate of social status (Hollingshead, 2011).

The occupation scale is scored between 1 and 9, nine being the most highly regarded occupations and one being less highly regarded careers. Examples of occupations which would receive a score of nine are architects, lawyers and physicians, while occupations that would earn a score of one are janitors, dishwashers and personal attendants. A seven-point scale is employed to determine educational

status. A seven indicates the completion of a graduate degree or higher while a one represents completion of less than a seventh grade education. To acquire a social status score the occupation score is multiplied by 5 and the education score is multiplied by 3 (Hollingshead, 1977). In families with two gainfully employed persons the total score is divided by 2 to obtain a social status score. The range of computed social status scores (8-66) are then divided into five categories of: lower (8-19), lower-middle (20-29), middle (30-39), upper-middle (40-54) and upper (55-66), (Hollingshead, 1975).

The Hollingshead Four Factor Index of Social Status is used as a benchmark to compare other measures of socio-economic status (Adams, J., & Weakliem, D., 2011). The Hollingshead reliability correlates with other indices of SES with ranges of .73-.89 (Cirino, Chin, Sevcik, Wolf, Lovett & Morris (2002). The social scores achieved are considered the best valid measure of socioeconomic differentiation available (Cassidy, Drotar, Ittenbach, Hottinger, Wray, Wernovsky, Newburger, Mahoney, Mussatto, Cohen, & Marino, 2013; Adams, J., & Weakliem, D., 2011).

Stress

Stress (psychosocial) measurements include everyday and major life experiences as captured by the Daily Hassles Scale-Revised (DHS-R). The DHS-R, developed by Kanner, Coyne, Schaefer & Lazarus, (1981), focuses on everyday stressors instead of major stress life events and psychological and somatic symptoms (Holm & Holroyd, 1992). Measures of daily life hassles in 53 items (e.g., time alone, your spouse, your health) on a 4-point scale, ranging from “none or not applicable” to a “great deal.” The item scores are summed to give a score for the

overall severity of hassles, ranging from 0-159. Internal consistency was .93-.97 over 2 years. Mean day-to-day correlation was .77 and mean monthly correlation was .82. Daily hassles severity is related to depression, anxiety, PTSD and role stress. The DHS-R has been previously used in studies with preterm born children (Msall & Park, 2008).

Coping

The Coping Response Inventory-Adult Form (CRI-A) measured coping responses, at Age 23 (Billing & Moss, 1981; Moos, 1995; Moss & Holahan, 2003). The CRI-A form, developed by Rudolf Moos, is a 48-item self-report inventory measuring cognitive and behavioral responses to stress. It measures coping orientation focus (approach or avoidance) and methods of coping used (cognitive or behavioral)

The CRI-A is appropriate for use in both healthy adults 18 years of age and older, and those with psychiatric, substance abuse and medical diagnoses. The instrument may be administered in structured interview or self-report format. The participants select and describe a recent stressor (within the last year) and use a four-point scale varying from “not at all” to “fairly often” to rate their reliance on each of the 48 coping items. Eight types of approach and avoidance coping responses are derived from the items. Approach responses include logical analysis, positive appraisal, seeking guidance and support, and problem solving. Avoidance responses include cognitive avoidance, acceptance or resignation, seeking alternative awards, and emotional discharge. Within each of the approach and avoidance responses, the first two scales are representative of cognitive coping strategies and the third and

fourth scales of behavioral coping strategies (Moos, 1993).

Additionally, ten appraisal items assess if the stressor was expected, viewed as a threat or challenge, the perceived cause and if resolution has occurred. Participants respond to questions aimed at examining the context of the stressor using a four-point scale varying from “definitely no” to “definitely yes” (Moos, 1993).

The CRI-A is interpreted using T-scores, with possible T-scores ranging from 20 to 80 + with a mean of 50 and a standard deviation of 10. A T-score of ≤ 34 is considerably below average and a score of ≥ 66 is considerably above average. Internal consistencies range from 0.61 to 0.74 for males and 0.58 to 0.71 for females. Cognitive avoidance internal consistency as measured by Cronbach’s alpha is .71 and emotional discharge is .60. Avoidance coping “*across time (1-year stability = .56; 6-year stability = .51) and across life domains (ie., interpersonal, health, and financial stressors), has been found to be stable* (Holahan, Moos, Holahan, et al., 2005). “ Test-retests alphas showed moderate stability over 12 months (average $r = 0.45$ for males and 0.43 for females). Content and face validity was built into the CRI-A by formulating definitions of specific domains, preparing items to fit the construct definitions, and selecting items that were conceptually and empirically related to a dimension. Reliability and validity have been extensively researched and found to be adequate (Moos, 1993 & 2004).

Emotional Intelligence

The Bar-On Emotional Quotient Inventory (Bar-On EQi) was used to measure the emotional intelligence of all participants. The Bar-On EQi is a self-report measure of emotional and social behaviors and the participants potential

to deal with daily environmental demands and pressures (Bar-On, 2002 & 2006; Sullivan, 2008). It has been used world-wide in a variety of settings with diverse populations and translated into 30 languages since being developed in the 1980's (Bar-On, 2006). The Bar-On EQi contains 133 items, on a 4-point Likert-like scale (ranging from very seldom to true), resulting in 5 composite scales and 15 subscales (Sullivan, 2008; Bar-On, 2006). The 5 composite scales are: *intrapersonal, interpersonal, adaptability, stress management and general mood* (Bar-On, 2006)". The intrapersonal scale measures self-awareness and self-expression as a composite assertiveness, independence and self-actualization subscales. The interpersonal scale measures social awareness and interpersonal relationships as a composite score of the social responsibility and interpersonal relationships ("*the establishment of mutually satisfying relationships and relating well with others*") scales (Bar-On, 2006). Stress management measures emotional management and regulation as a composite score of the stress tolerance and impulse control subscales. The adaptability score measures change management as a composite score of the reality testing, flexibility and problem solving. The general mood composite score measuring self-motivation is derived from the subscales of optimism and happiness. Finally an overall total emotional quotient score is generated.

The 133 items are rated on a five-point Likert-type scale with choices ranging from "very seldom" or "not true" to "very often true" or "true". The raw scores are converted into standard scores with a mean of 100 and standard deviation (*SD*) of 15. The higher the score the more potential for effective emotional and social functioning

and the prediction for meeting daily demands and challenges (Bar-On, 2006).

The Bar-On EQi questionnaire has a built in validity correction factor that adjusts for response bias. Internal consistencies range from .69 - .96 with all composite scales correlate highly with the total EQ-I scores (.67 to .93), (Dawda & Hart, 2000). Test-retest reliability at six months ranges from .72 to .80 (Bar-On, 2006). Ten subscales from a confirmatory factor analysis are the strongest measures of the construct: self-regard, interpersonal, impulse control, problem solving, emotional self-control, “*emotional self-awareness, flexibility, reality testing, stress tolerance, assertiveness and empathy* (Bar-On, 2006).” The remaining 5 subscales are facilitators of emotional and social intelligent behaviors: independence, self-actualization, social responsibility, optimism and happiness. The Bar-On EQi has been extensively researched and validated with evidence strongly supporting construct, divergent, discriminant, criterion, and convergent and predictive validity.

Emotional Health

The Adult Self-Report (ASR) was used to determine emotional health. The ASR for ages 18-59 is extensively researched and used widely in research studies for well over twenty years (Achenbach, 2003). The ASR assesses social competence and behavior problems in adults ages 18-59. The participant completes the ASR and report their own functioning, problems and substance use. It provides normative scales for functioning, syndromes, substance use, internalizing, externalizing and total problems (ASEBA, 2011). The syndrome scales are then profiled to international expertly derived *Diagnostic and Statistical Manual of Mental Disorders Fourth Edition Text Revision (DSM IV-TR)* oriented scales (Achenbach, 2003).

The ASR (2003) is composed of data relating to friends, spouse or partner, family, job, education and a list of self-descriptors. The descriptor list contains 126 items rated on a 3-point scale consisting of “not true, somewhat, or sometimes true and very true or often true”. Raw scores are converted in *t* - scores are categorized into clinical risk screening ratings of normal ($t = <59$), borderline ($t = 60-63$), and clinical ($t = 64$ and above). Classification parameters resulted in true positives (sensitivity) = 80%; true negative (specificity) = 95%; false positives = 20%; false negatives = 5%.

Category scores are related to DSM IV-TR disorder criteria. Internalizing behaviors include anxiety, depression and withdrawn behaviors while externalizing behaviors include aggression, rule breaking and intrusive behaviors. The DSM-oriented problem scales are depressive; anxiety problems; somatic; avoidant personality; AD/H and antisocial personality.

Reliability is excellent: 1-week test-retest correlations were .80 - .90, 2 years was .69 with none below .71. Internal consistency range was .78 - .85. Discriminant validity was demonstrated in the referred and non-referred samples and with DSM IV-TR categories.

Stress Reactivity

The stress reactivity responses were measured by the Trier Social Stress Test (TSST). The TSST, developed by Kirschbaum, is a standardized protocol for inducing stress to subjects in studies and deliberately activates the HPA axis response (Kirschbaum, 2010; Kirschbaum, Pirke & Hellhammer, 1993). It was used in this study to assess physiological reactivity to what the participant perceives as a

challenging situation which has the potential for being negatively judged by others (Kudielka, 2008; Sullivan, 2008 - 2015). In addition to invoking reports of negative mood changes and increased anxiety, the HPA Axis response of endocrine, immunological and other biological markers have been well documented (Stolerman, 2009). It is widely used and involves a well-scripted task the participant is asked to engage in hypothetically while others are evaluating their verbal response. The task consists of a brief preparation period (10 minutes), a test period where the participant delivers a speech for a mock interview (5 minutes) and finally a period of calculating mental arithmetic (5 minutes) in front of an audience. While completing the tasks the participants are also being video recorded to later assess their behaviors while under stress. In the original study their behavioral presentation will be evaluated according to pre-established definition levels of: humor, anxiety, non-verbal but otherwise engaged, defensive, aggressive, scorn, frustration and distraught.

Salivary cortisol samples are collected at the following intervals: prior to the task (baseline), 15, 30, 45, 60, 75 and 90 minutes after the task has started (Sullivan, 2008-2013). A mandatory part of the protocol is participants are debriefed after completion of the test (Kirschbaum, 2010). The TSST has been used with children, adults, healthy subjects and clinical populations. It is among one of the few tests that assess both behavioral and psychobiological responses to situations that are perceived as uncontrollable and socially threatening (Kudielka, 2008; Dickerson & Kemey, 2004).

HPA Axis Biomarker: Salivary Cortisol

Salimetrics, Inc., Expanded Range High Sensitivity (ERHS) Salivary Cortisol

Enzymes Immunoassay Kit for research was used to collect samplings during the TSST (Salimetrics, Inc., 2008). Sensitivity and specificity of the Salivary Cortisol Enzyme Immunoassay is excellent, as well as, a widely used solid technology, and the “gold standard among researchers. The laboratory procedures include duplicate assays for each cortisol sample so that each saliva sample is tested twice on the assay plate. This renders two data points per sample and a mean for each sample tested in duplicate. Duplicate assays minimize the potential of with-in subject variability. Each assay captures the full range of salivary cortisol levels (0.003 to 3.0 ug/dL) while using only 25 uL of saliva per test (Salimetrics, Inc., 2008). The lower limit of sensitivity was determined by interpolating the mean minus 2 standard deviations (*SDs*) for 10 sets of duplicates at 0 ug/dl standard. For sensitivity, the minimal concentration of cortisol that can be distinguished from 0 is < .003 ug/dL. The correlation between saliva (Salimetrics ERHS Salivary Cortisol EIA) and serum (Diagnostic Systems Laboratory) was highly significant ($r = .91, p = .0001$). During testing, a potential hydrogen (ph) indicator alerts if cortisol levels are artificially inflated or lowered (< 3.5 or ≥ 9.0); possible blood contamination is screened; and temperature is controlled. Specificity of antiserum varies but is excellent. The EIA is accurate in diagnosis of Cushing Syndrome (Raff, Homan, & Skoner, 2003; Lucke, Loucks & Berga, 2007). Sensitivity/Specificity of 100 %/95 % was reported for salivary cortisol to distinguish diagnosis of Cushing’s syndrome and obesity in an age-matched sample of children (Castro, Elias, Martinelli, Antonnini, Santiago & Moreira, 2000). Various adult studies exist that measure cortisol levels in a variety of psychological pathologies, emergency situations, and anxiety reduction interventions

(Harvey, Nathans, Bandiera & LeBlance, 2010; Rapaport, Schettler & Bresse, 2010).

In the primary study, a careful research protocol was followed. A foam oral swab was used to collect saliva and placed under the participant's tongue for 2 minutes. Approximately 1 mL of saliva saturated the foam swab and then it was inserted into a labeled collection tube and stored in a freezer. The saliva samples were placed into a cryostorage box with a minimum of 5 lbs. of dry ice and shipped via Federal Express Priority for analysis by Salimetrics, Inc. Samples were typically submitted to Salimetrics quarterly (every 3 months) but more frequently when necessary. Shipping instructions according to www.salimetrics.com were followed.

Once analyzed, specimen data were entered into a spreadsheet by Salimetrics, Inc. and reported to the Principal Investigator via e-mail in the form of an Excel file with two values for each saliva sample and the mean in the form of a concentration (e.g., cortisol = 0.652 ug/dL). Saliva samples were discarded according to Pennsylvania Department of Environment Protection regulations 30 days after testing was completed. Data from the log were entered into MS Excel by study staff. The only identifier between the participant and the saliva sample was a predetermined label provided by Salimetrics, Inc., who had no access to data to link the sample to the participant. It was the responsibility of the research staff to link the participant's unique identification number with the Salimetrics predetermined label.

Summary

Data for the proposed study include neonatal and demographic data from infancy and self-report questionnaires, 8 sequential salivary cortisol samples from the TSST standard laboratory protocol from the same participants at age 23 years. The

measures are well developed with good psychometric values. Questionnaire scoring and subscale information are provided in the Appendix Measurements

Procedure

Researcher

In the study, the researchers were kept blinded to all identifying information which was coded by the primary study staff. The coded database was not associated with identifying information. The anonymous complete dataset for the study was provided by the project director.

Recruitment

The infancy sample was recruited during the mother's post-partum stay or during the infant NICU stay. The full term infants were identified during the post-partum and in the same time frame as the preterm infants. The criteria for recruitment were neonatal diagnoses, birth weight < 1850 grams (4 pounds), maternal history (no history of mental illness), and English as a primary language. Parent(s) were then invited to participate by research nurses. Fewer than 10% of the parent(s) declined participation.

Neonatal Group Criteria was: (1) full term healthy infants (FT); (2) preterm infants without illness (healthy preterm group: HPT); (3) preterm infants with medical illness (bronchopulmonary dysplasia, respiratory distress syndrome, necrotizing enterocolitis and sepsis: MPT); (4) preterm infants with neurological illness (meningitis, hydrocephalus, and grade 3 or 4 intraventricular hemorrhage: NPT); and preterm infants who were small for gestational age (SGA). Additionally specific protocols for neonatal diagnostic labeling were used. The range of premature infants

in weeks and grams were from 28.3 to 40 weeks gestational age and 1149 (2.5 lbs.) – 3420 (7.5 lbs.) grams in weight. Socioeconomic status, incorporating both maternal and paternal education and occupation, was measured with the Hollingshead Four Factor Index.

Infants were followed in a series of research studies at ages 18 months, 30 months, 4 years, 8 years, 12 years, and 17 years. At age 23, the original researchers who had worked with the participants for more than 10 years, made recruitment contact again. Sample retention was high with 96% of the sample from age 17 years and 85% of the birth sample participating at age 23 years. The research protocol included a home visit and hospital research laboratory visit.

Demographics

At age 23, demographic information was updated to include name, address, home and cell telephone numbers, e-mail address, social security number, marital status, education level/history, occupation/history, source(s) of financial support, lists of people living in home, pregnancies (for female subjects) and number of children, closest relative information, primary care provider information, and use of medical and community support services. The participant also filled out his or her own Hollingshead Four Factor Index of Social Status.

The Daily Hassles Scale-Revised (DHS-R), the Coping Response Inventory (CRI-A), the Bar-On EQi and the Adult Self-Report (ASR) were administered to each participant, as part of the larger study protocol of tests and questionnaires, during their first laboratory visit. Laboratory visits were scheduled according to standardized time periods. Each participant followed the same schedule or sequencing of

questionnaires, food breaks and examinations throughout the specified time period allowing for consistency in protocol and comparison between participants. This battery of tests was administered after completion of the history and physical in a small private room free from distractions. The research assistants monitored, timed and administrated the sequencing of tests according to study protocols.

TSST Protocol

The Trier Social Stress Test (TSST) followed standardized and extensive protocols relating to timing during the day, timing with other testing and testing protocols. Levels of cortisol are heightened in the morning and decline throughout the day. To reduce the potential of varied cortisol levels among subjects, administration of the (TSST) was standardized to 1 pm for all participants. Upon arrival the research nurse (RN) greeted the participant, explained the laboratory protocol, confirmed consent and addressed questions. To avoid confounders in possible stimulation of the HPA axis, the RN inquired about health-related limitations (colds, flu), physical activity, smoking, medications, large meals, and hormonal treatments within the last 2 hours. The RN had informed participants of these restrictions when the testing was scheduled. Participants were not informed in great detail about the exact procedure of the TSST. The RN inquired about the participant's ideal occupation as this was necessary information for the speech segment of the TSST protocol. A baseline salivary cortisol sample was collected 20 minutes after arrived (and the time noted) followed by the TSST.

The participants were escorted to an assessment room and told there would be two parts to this segment: (1) mock job interview and (2) arithmetic problem. All scripts

were followed verbatim. For the mock job interview, the participant was instructed by the researcher with a scripted standardized comment. The participant was given 10 minutes to prepare for the interview and informed that no notes could be used and they needed to make an impression on the committee (composed of 4 research assistants) who would decide on the acceptance of the application.

The mock job interview was allowed 5 minutes, only one RA would talk and direct the participant and the rest would be silent, with focused eye contact and a serious demeanor that excluded any non-verbal encouragement. A dummy video camera was set up for the mock interview and the participant was asked to *“please step behind this line, and begin your speech.”* All research assistants followed protocols for their roles, behavior, eye contact, facial expressions and speech. If there was a pause in the speech, then at 20-seconds the participant was told: *“You still have time, please continue.”* Should there be another pause and the participant has nothing to add then the RA will ask a standardized set of questions until the 5-minute time period ended. At the end of the job interview time the RA announces that the second part of the test will begin and states it had nothing to do with the job interview.

The verbal instructions to the participant for the arithmetic test is also scripted and in part includes:

“We ask you to count backwards to 0 in 13 number steps, starting at 1687.

It is important to do it fast and correctly. Should you miscalculate, you will be told to start again at 1687. Any questions? Please begin.”

During this time the RA made notes on paper for errors in calculations. If the participant miscalculates, the RA will respond with *“Error. 1687.”* After 5-minutes

of mental arithmetic, the RA stopped the TSST.

The second cortisol sample was collected 20 minutes after the TSST. In the interim, the RN conducted the health history interview. After the collection of the cortisol sample there was a break and then additional questionnaires were administered and collected. The third cortisol sample was collected after the questionnaires were completed, approximately 30 minutes after the collection of the second sample (50 minutes after the TSST). The RN then begin the debriefing of the TSST with:

“The first thing I’d like to tell you is that the job interview speech and the arithmetic part were supposed to be difficult. We did this to elicit a stressful situation for you. When people encounter stressful events, their bodies respond with an accelerated heart rate and the body produces cortisol. We will be measuring your cortisol through your saliva. We were not really recording audio or video for preverbal stress or behavioral analysis. Also, the judge is actually one of our research assistants. Let me introduce you to him/her.”

After, the debriefing, a healthy snack was given and further assessment inventories were administered. The physical examination followed and further salivary cortisol samples were collected at 60, 75 and 90 minutes.

Summary

All measurements representative of the dependent and independent variables to be utilized in this secondary analysis have been reviewed along with the original study procedural protocols. Use of this data, from the original measurements with good psychometric prosperities, remains a strong fit to the aims of this study despite

the longevity of the original study design.

Analysis

All data was analyzed using the IBM SPSS Statistics Standard Grad Pack (SPSS), Version 22 descriptive statistics. The SPSS is used widely in social science research to obtain descriptive statistics (mean, standard deviation, range), graphic depiction, and hypothesis testing (Chi Square, ANOVA, ANCOVA, MANOVA and MANCOVA).

First frequency statistics were performed for each variable. The frequency and pattern of missing data were reviewed. Missing data imputation was considered when $> 10\%$ data were missing. Distribution skewness, kurtosis, box-plots were examined and transformations considered when needed. Correlations were examined between all dependent variables. Correlations $> .80$ were considered to be highly correlated and decisions were made to use one variable (FGSE, 2016). The significance level was $p \leq .05$. Analysis of Variance (ANOVA) was used to determine any variations within and between the groups (LoBiondo-Wood & Haber, 2010) in all self-report variables. AUCg was used with the cortisol endocrine data as a measurement of hormonal output (Fekedulegn, et. al., 2007). The AUCg is the total area under the plotted curve of all the multivariate measurements and allows for identification of the differences between the single measurements and the distance of these measurements from ground (Spence, Cotton, Underwood & Duncan, 1976). Measurements from ground (see Figure 11) take into consideration the distance from zero instead of baseline data points and allows for total salivary cortisol response comparisons between participants. (Fekedulegn, et. al., 2007). Finally, the level of

effect power range recommended to reduce any Type I (is effect when there is none) or II (no effect when there is) error at the 0.05 significance criterion is 0.80 – 0.95 (Lakens, 2013). The study's power was calculated at the 0.80 significance criteria.

Figure 11

Area Under the Curve with Respect to Ground (AUCg)

Figure 11. From: Catherine Raymond, Marie-France Marin, Anne Hand, Shireen Sindi, Robert-Paul Juster, and Sonia J. Lupien, "Salivary Cortisol Levels and Depressive Symptomatology in Consumers and Nonconsumers of Self-Help Books: A Pilot Study," *Neural Plasticity*, vol. 2016, Article ID 3136743, 12 pages, 2016. Figure 4, p 9. doi: 10.1155/2016/3136743 <http://doi.org/10.1155/2016/3136743> Retrieved December 15, 2016. This is an "open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited."

Aims and Hypotheses

Aim 1: Compare effect of prematurity on stress, coping, emotional intelligence and emotional health. Compare independent categorical variable of birth groups with the dependent continuous variables: DHS summed scores ranging from 0-159 for severity of stress, the CRI-A mean scores ranging from 20-80 + with M 50 (SD 10) for avoidance coping, the Bar-On EQI total mean score of 100, (SD 15) for emotional intelligence effectiveness and the ASR mean score of ≥ 60 for risk to emotional health.

Hypothesis 1. Higher self-reported stress scores, higher use of avoidance coping styles, lower emotional intelligence scores and more emotional health disorders will be found for the adults at age 23 years born prematurely compared to the term-born adults. Hypothesis 1 will be significant if DHS-R total scores are higher, CRI-A avoidance t -scores are higher, EQi total scores are lower, and ASR scores are higher for the premature groups.

Aim 2: Compare the salivary cortisol response between premature and full-term born infants in the social stress paradigm of the TSST. Descriptive statistics including line graphs by groups will be examined first. Analysis of Variance (ANOVA) will be used to determine differences in salivary cortisol responses as measured by AUCg and at 6 sample times from the TSST paradigm at 15, 30, 45, 60, 75 and 90.

Hypothesis 2. Adults at age 23 who were born prematurely will have a prolonged stress recovery period for the TSST. Hypothesis 2 will be significant if the AUCg mean is higher during the stress recovery period for TSST in the prematurely born age

23 year old adult.

Aim 3. Examine the relationship between effect of emotional health and on the stress recovery period of the TSST measured in salivary cortisol. Three levels of emotional health (normal > 59 , borderline $60 - 63$, and clinical ≥ 64) as measured by the ASR will be examined for the salivary cortisol samples across 6 sample times.

Descriptive statistics including graph lines by groups will be used followed by ANCOVA where the effects of prematurity and gender will be controlled to understand whether cortisol AUCg and slopes differ due to emotional health.

Hypothesis 3. The stress recovery period for adults at age 23-years with emotional health problems will be prolonged compared to adults without emotional health problems (as determined by ASR *t*-scores) when prematurity is controlled.

Summary

The measurements used for this secondary analysis are consistent with the aims of the primary study. Measurements and procedures for all dependent and independent variables were reviewed. The measures have demonstrated good psychometric properties. In the original study, the research team developed and trained on the procedure protocols. Reliability between team members in the protocol were consistently assessed and were $\geq 90\%$. The design of this study allows for correlational and multivariate modeling. The use of neonatal data enables predictive analysis.

CHAPTER FOUR

Data Analysis

The analysis of data was designed to address the aims of the study with the overall goal to determine how premature birth effects stress, coping, emotional intelligence and emotional health at age 23 years old. Self-report responses were compared to salivary cortisol levels obtained during a moderate psychological stress inducing standardized test, the Trier Social Stress Test (TSST). One hundred and sixty-six 23 year-old young adults participated in this study with 142 participants completing the self-report and TSST protocols. The results are presented as follows: sample, demographics, data integrity, independent variables, dependent variables and analysis of the hypotheses.

Sample Demographics

The original sample ($N = 213$) was recruited as infants who met a priori criteria for five perinatal birth groups. The infants and families participated in a series of research studies through infancy, toddlerhood, preschool, school age, and adolescence. By age 17 years, 180 participants from the original study sample participated in the research protocol for an 85% retention rate from birth. At age 23 years, 180 participated resulting in a retention rate of 85%. Overall, this study maintained an 84% retention rate from birth to age 23.

Forty-two participants were lost to attrition, moved, or decided not to participate. There were approximately 10 participants who were unable to complete some of the protocol due to medical and/or developmental disabilities. For this secondary

analysis, the sample consists of 166 who completed the self-report measures and the TSST. Thus, 92% (166 of 180) of the age 23 sample is represented in this study. Of the fourteen subjects who did not participate, three with IQ scores < 70 were excluded ($M = 60.67$, $SD = 6.66$) due to severe cognitive limitations, four were unable to complete the self-report measures, and seven chose to only complete some of the full protocol. There were differences in perinatal birth group in gender between those who participated at age 23 and those who did not [$N = 166$: $X^2 = (1,4) = 11.919$, $p = .018$ & $n = 42$: $X^2 = (1,4) = 4.270$, $p = .371$ respectively]. More females (91%) than males (62%) participated at age 23.

One-hundred sixty-six subjects who participated in this study had a mean age of 23.19 years ($SD = 1.015$; range 22 – 27) and were predominately white (87%) as shown in Tables 3 & 4. There were approximately equal numbers of males, $n = 79$ (48%), and females $n = 87$ (52%). The majority of participants were single (80%), graduated high school (91%), and were educated beyond high school (70%). Seventy-two percent were employed in unskilled, skilled, semiprofessional and professional/executive positions or were students (Table 4). As a group the 23 year-old participants' socioeconomic status was moderately high ($M = 40.58$, $SD = 13.61$) on the Hollingshead Index (Hollingshead, 1975) range of 40-54. All socioeconomic status levels were represented in the distribution as follows: High 15%, moderately high 43%, average 19%, moderately low 16% and low 8%. Many participants were still partially dependent on their parent(s) for economic support, which partially explains the moderately high status at age 23. Those who

participated in the TSST and self-report of stress measures had normal intelligence scores ($M = 100.33$, $SD = 12.9$), (Table 3).

Table 3

Age 23 Demographic Data: Age, SES and IQ

Variable	<i>N</i>	<i>M</i>	<i>SD</i>
Age	166	23.19	1.015
SES	159	40.58	13.611
IQ Age 17	149	100.33	12.897

Table 4

Summary of Age 23 Demographic Data ($N = 166$)

Variable	<i>n (%)</i>
Perinatal Birth Group	
FT	41 (24.7)
HPT	24 (14.5)
MPT	49 (29.5)
NPT	26 (15.7)
SGA	26 (15.7)
Gender	
Male	79 (48)

Variable	<i>n</i> (%)
Female	87 (52)
Race	
African American	14 (9)
White	138 (87)
Native American	2 (1)
Asian	1 (< 1)
More than 1 race	3 (2)
Educational Level	
High School	
GED	7 (5)
Diploma	137 (91)
Prep School	2 (1)
Vocational School	10 (6)
Community College	6 (4)
College Graduate	64 (41)
Graduate School	3 (2)
Transitional Program	1 (< 1)
Employed	114 (72)
Occupational Category	
Unskilled to skilled	77 (48)

Variable	<i>n</i> (%)
Semiprofessional	37 (23)
Professional/executive	6 (4)
Student	39 (25)
Marital Status	
Single	126 (80)
Cohabiting or married	31 (20)
Separated, divorced, widow	1 (< 1)
SES	
High 55-66	24 (15)
Moderate High 40–54	68 (42.8)
Average 30–39	30 (18.9)
Moderate Low 20–29	25 (15.7)
Low 8–19	12 (7.5)
IQ Score Categories at Age 17	
> 130	0
116 – 130	16 (11)
85 – 115	113 (76)
70 -84	20 (13)
< 70	0

Summary

The sample for the study is 166 young adults aged 23 years who have participated since birth in a series of longitudinal studies of premature infants. Ninety-two percent of the full sample completed the measures of interest for this study, although this varied as discussed below. All perinatal birth groups are represented with near equal number of males and females. The sample race is primarily characterized by white participants who have completed a high school education and represent all level of socioeconomic status.

Data Integrity

Distribution

Frequency statistics were performed for 45 variables, resulting in a range of 154-166 participant completions for all variables, except cortisol salivary samples (Range 142-149 participants completed). Participant missing data on dependent variables ranged from 11-24. Mean score, standard deviation values, box plots, histograms and Q-Q Plots were examined for all study variables to determine distribution patterns (Ghasemi & Zahediasi, 2012).

There was slight skewness in the range of distributions of most variables (Bannon, 2013). Coping variables of logical analysis, seeking guidance and support, problem solving, cognitive avoidance, and acceptance or resignation were slightly negatively (right tailed and higher than median) skewed (Bannon, 2013). The remaining coping variables of positive appraisal, seeking alternative rewards and emotional discharge, in contrast, were slightly positively (left tailed and lower than

median) skewed. Additionally, stress scores (DHS-R), emotional health (ASR) and the TSST cortisol salivary levels were slightly positively skewed (left tail and lower than median) while emotional intelligence (EQi) scores were slightly negatively skewed (right tail and higher than median). The kurtosis of the dependent variables ranged from slightly platykurtic to slightly leptokurtic distributions (Bannon, 2013).

The Kolmogorov-Smirnov and Shapiro-Wilk statistical test results were used to determine the level of normality in all variable value distributions (Bannon, 2013, p 97) and although not normal distributions they were in the expected range of distributions. Outlier scores for each variable were identified through means and standard deviations, boxplot displays, and Q-Q plots without significant severe scores detected.

Missing Data

Little's Missing Completely at Random (MCAR) test ($X^2 = 330.307$, $df = 338$, $p = .607$) indicated there was not evidence for a pattern of missing data and at minimum not for the primary independent and dependent variables (IBM, 2011). Therefore, the results were not significant, the available evidence used failed to reject the null hypothesis in the variables measured, and the likelihood of a Type II error was diminished (Garson, 2015). A type II error or "false-negative" (Banerjee, Chitnis, Jadhav, Bhawalkar & Chaudhury, 2009) results when the researcher accepts a false result from the population studied (Banergee et al., 2009).

Overall, the entire sample missing data showed no differences in mean values between the participants who completed the dependent measurements and those that

did not, indicating no systematic study bias. Imputation of missing data were a consideration for two measurements with $\geq 10\%$ missing data: Intelligence (IQ) scores with 10.2% missing and all eight TSST salivary cortisol measures (ranging from 10.2%-14.5%). Missing IQ scores were only used for demographic results and not in further data analysis. Missing TSST salivary cortisol levels were automatically dropped from the analysis methods used in this study, by excluding cases pair-wise in any means, correlational analysis and analysis of variances (Howell, 2016).

Power Analysis

An a priori power analysis was used to determine the required sample size, power level and size of effect to be determined in order to prevent the possibilities of a Type I (reject when true) or II error (accept when false) (Faul, Erdfelder, Buchner & Lang, 2009; Faul, Erdfelder, Lang, & Buchner, 2007; Mayer, Erdfelder, Buchner & Faul, 2007). G*Power (3.1 version) was used as the power analysis statistical program for this study and is widely used in quantitative research studies (Bannon, 2013; Faul, et al., 2007 & 2009; Myer, et al., 2007; Thomas & Krebs, 1997). An *F* test ANOVA with fixed effects, omnibus, one - way, analysis of “Sensitivity: Compute required sample size” was used. The decision to use ANOVA was based on the statistical need to analyze the variance for all three aims of the study. The overall sample effect size *f* was determined by using an α error probability of 0.05, power (1- β error probability) of 0.08 and the total sample size of 166 for each (5) perinatal birth group (Range of 26 – 49 participants; UCLA, 2016).

Based on G*Power definitions of effect size (small = .01, medium = 0.25 and

large = 0.5) a medium effect size, $f = 0.2721144$ and F_{crit} of 2.4278100 was obtained for this study (Buchner, Erdfelder, Faul, & Lang, 2014). See Figure 12.

An ANCOVA analysis for 5 groups with 2 covariates for effect size f and F_{crit} values at the $p = .05$ level was calculated and plotted for sample sizes with up to 170 participants using power error probability of 0.8 (Figure 13).

Additionally, effect size f and F_{crit} values for ANOVA and ANCOVA analyses were calculated and distributions plotted for varying sample, group and covariate sizes (Table 5 & Appendix B: Power Analyses). The effect size f 's ranged from 0.272 - 0.335 (medium), with $p = 0.05$, and the F_{crit} values ranged from 1.99 – 2.44 with sample sizes from 142-170.

Figure 12

Distributions Plot, Power Analysis: ANOVA, $N = 166$

Figure 13

ANCOVA: 5 Groups and 2 Covariates Sample Size and Effect f Plot

Table 5

Power Analyses

Test	N	Ndf	Ddf	F_{crit}	Effect Size f Interpretation
ANOVA	166	4	161	2.4278100	0.2721144 Medium
	142	4	137	2.4377490	0.2949706 Medium
ANCOVA	170	7	163	2.0661634	0.2968223 Medium
	166	6	160	2.1556612	0.2921407 Medium
	166	7	159	2.0676042	0.3005374 Medium

Test	<i>N</i>	<i>Ndf</i>	<i>Ddf</i>	F_{crit}	Effect Size <i>f</i> Interpretation
	66	8	158	1.9974370	0.3082074 Medium
	142	6	136	2.1658823	0.3169486 Medium
	142	7	135	2.0780690	0.3261991 Medium
	142	8	134	2.0081589	0.3346675 Medium

Multicollinearity

The correlation coefficients were determined utilizing IBM SPSS Statistics (Version 24; IBM, 2011). Pearson Correlational Coefficients analysis results were transformed into a correlation matrix, (Appendix C). Socioeconomic status, intelligence, birth weight, neonatal acuity scores as well as the 23-year stress, coping, emotional intelligence and emotional health predictor variables were assessed for multicollinearity. Birth weight and the Hobel Neonatal (acuity) scores had a strong negative ($r = -.850, p = 0.01$) correlation indicating that as birth weight went down the Hobel Neonatal acuity score increased (Rumsey, 2010). The Hobel Neonatal acuity score is a weighed composite score which includes birth weight as a measurement in the total score (Strobino & Baruffi, 1984). These intercorrelated demographic variables were not removed or transformed (Bannon, 2013.)

Multicollinearity ($r = \geq .80$) of the salivary cortisol values of delta, peak, slope and AUCg existed between slope and delta ($r = .993$) and AUCg and peak ($r = .905$) (Table 6). The cortisol value measurements analyzed were already winsorized and had undergone a natural log transformation.

Table 6

Correlation Matrix for Cortisol Values and Perinatal Birth Groups

Variables	(1)	(2)	(3)	(4)	(5)
(1) Delta	-				
(2) Peak	.719**	-			
(3) Slope	.993**	.726**	-		
(4) AUCg	.430**	.905**	.425**	-	

** $p = 0.01$ (2-tailed)

Khoury, et al., 2015, rigorous analysis of 219 cortisol research challenge studies, from six different journals spanning 2011-2013, found the frequent use of all TSST cortisol values as redundant. The multicollinearity between all cortisol measurements have been an issue in cortisol values research. Khoury, et al. found two dimensions to cortisol values consisting of total cortisol or cortisol change (Khoury, et al., 2015). The use of slope, AUCg and/or AUCi (natural log value) as research variables were supported by extensive statistical analysis, as providing the lowest correlations and assessing the two different dimensions (Khoury, et al, 2015). Given, the

multicollinearity found in the cortisol measurements and relevant research literature, the slope and AUCg ($r = .425$) were used in this analysis to assess cortisol changes and levels.

Homoscedasity

The continuous 23-year dependent variables of stress, coping, emotional intelligence, emotional health and the TSST cortisol stress responses were assessed for homogeneity of variance, in addition to the examination of box plots, through the statistical use of Levene's Test (Bannon, 2013; QS, 2014). Coping styles of logical analysis ($p = .02$) and acceptance or resignation ($p = .03$) were significant for differences in gender variance in the perinatal birth with gender group. Once gender was controlled for in the perinatal birth with gender group it was no longer significant for logical analysis ($p = .44$) and acceptance or resignation ($p = .29$) coping styles. All results of the homogeneity of variance test, after controlling for gender, were not significant and demonstrated that the variances of the dependent variables across groups were equal (Pallant, 2010). Appendix D includes full details of this analysis.

Linearity

Examinations of distribution plots and correlations show a linear relationship among the continuous dependent variables. Simple scatterplots demonstrated linearity of all continuous dependent variables for the categorical independent variable of perinatal group and the covariate of gender (Pallant, 2010).

Summary

The statistical checks of data integrity, test assumptions (missing data and

measures), normality (skewness, kurtosis and outliers), multicollinearity, linearity, and homoscedacity have been met. Missing data were found to be random and a priori G*Power analysis indicated an adequate sample size and with medium effect size.

Independent Variables

The descriptive analysis of the independent variables of perinatal birth group and gender follow. Socioeconomic status, as a confounding variable, was also descriptively analyzed.

Perinatal Birth Group

Of the 166 participants, the perinatal birth group sizes were: Full term $n = 41$ (24.7%), healthy preterm $n = 24$ (14.5%), medical preterm $n = 49$ (29.5%), neurological preterm $n = 26$ (15.7) and small for gestational age $n = 26$ (15.7%), Significant differences ($p = .001$) were found between the 5 perinatal birth groups at birth as expected in: birth weight, gestational age, Hobel Neonatal and Hobel Total acuity scores, duration of oxygen use, and number of hospitalized days (Table 6). Again, as expected, there were significant differences at birth for medical diagnoses

Table 7

Sample and Perinatal Group: Neonatal Demographic and Medical Diagnoses Data, N=166

Variables	Sample	Perinatal Group				
		FT	HPT	MPT	NPT	SGA
	<i>N</i> =166	<i>n</i> =41 (24.7)	<i>n</i> =24 (14.5)	<i>n</i> =49 (29.5)	<i>n</i> =26 (15.7)	<i>n</i> =26 (15.7)
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>
<u>Analysis</u>						
Birth weight in grams	1784.35 (976)	3371.7 (412.9)	1470.4 (218.3)	1281.7 (324)	1167.9 (293)	1134.8 (358.6)
Range	640-4370	2495-4370	900-1600	710-1800	720-1670	640-1915
<u>$F(4, 161) = 305.276, p = .001^*$</u>						
Gestational age in weeks	32.46 (4.8)	39.8 (.88)	31.0 (1.71)	29.4 (2.3)	28.5 (2.2)	32 (2.7)
Range	25-43	37-43	28-34	25-33	25 -33	28-36
<u>$F(4, 161) = 191.471, p = .001^*$</u>						

Table 7

continued

Variables	Sample	Perinatal Group				
		FT	HPT	MPT	NPT	SGA
	<i>N</i> = 166	<i>n</i> = 41 (24.7)	<i>n</i> = 24 (14.5)	<i>n</i> = 49 (29.5)	<i>n</i> = 26 (15.7)	<i>n</i> = 26 (15.7)
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)
<u>Analysis</u>						
Hobel neonatal score	65.82 (45.6)	1.54 (3.56)	57.6 (22.7)	89.8 (25.5)	112.3 (22.3)	76.7 (33.4)
Range	0-160	0-15	20-110	30-129	48-160	8-136
$F(4, 161) = 123.661, p = .001^*$						
Hobel birth total score	109.64 (64.6)	13 (12)	113 (34)	144 (33)	167 (29)	138 (38)
Range	0-220	0-52	51-187	81-202	86-220	34-201
$F(4, 161) = 154.005, p = .001^*$						
O ₂ Duration in hours	276.41 (540.3)	0	14 (19)	468 (636)	632 (661)	257 (572)

Table 7

continued

		Sample	Perinatal Group				
Variables			FT	HPT	MPT	NPT	SGA
		<i>N</i> = 166	<i>n</i> = 41 (24.7)	<i>n</i> = 24 (14.5)	<i>n</i> = 49 (29.5)	<i>n</i> = 26 (15.7)	<i>n</i> = 26 (15.7)
		<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)
<u>Analysis</u>							
Range	0-2568	0	0-72	0-2222	0-2568	0-2140	
<u><i>F</i>(4, 161) = 10.143, <i>p</i> = .001*</u>							
Number of days		39.42 (30.3)	3 (.418)	33 (10)	53 (24)	65 (22)	52 (31)
Hospitalized: Range		2-134	2-5	17-60	17-98	23-107	9-134
<u><i>F</i>(1, 159) = 135.467, <i>p</i> = .001*</u>							

Table 7

continued

Neonatal Medical Diagnoses	Sample <i>N</i> (%)	Perinatal Group				
		FT	HPT	MPT	NPT	SGA
		<u>Analysis</u>				
IVH	24 (14.4)					
Grade 1	8(5)	0	1 (.6)	1 (.6)	5 (3)	1 (.6)
Grade II	7 (4)	0	1 (.6)	1 (.6)	5 (3)	0
Grade III	7 (4)	0	0	0	6 (3.6)	1 (.6)
Grade IV	2 (1)	0	0	0	1 (3.8)	1 (3.8)
			<u>$X^2(16) = 72.007, p = .001^*$</u>			
BPD	21 (13)	0	0	11 (22)	8 (38)	2 (9.5)
			<u>$X^2(4) = 21.974, p = .001^*$</u>			

Table 7

continued

Neonatal Medical Diagnoses	Sample <i>N (%)</i>	Perinatal Group				
		FT	HPT	MPT	NPT	SGA
				<u>Analysis</u>		
NEC	12 (7)	0	0	7 (58)	2 (17)	3 (11.5)
				<u>$X^2(4) = 9.432, p = .051$</u>		
Sepsis	14 (8)	0	0	5 (35.7)	6 (42.9)	3 (21.4)
				<u>$X^2(4) = 13.729, p = .008^*$</u>		
Meningitis	4 (2)	0	0	0	3 (75)	1 (25)
				<u>$X^2(4) = 12.257, p = .016^*$</u>		
Hydrocephalus	3 (2)	0	0	0	3 (100)	0
				<u>$X^2(4) = 16.451, p = .002^*$</u>		

Table 7

continued

Neonatal Medical Diagnoses	Sample <i>M (SD)</i>	Birth Group				
		FT	HPT	MPT	NPT	SGA
				<u>Analysis</u>		
SES raw score	39.19 (14.6)	36.93 (14.6)	34.92 (15.3)	41.51 (13.8)	35.46 (12.9)	46.04 (14.7)
Range	8-66	11-64	8-63	14-66	17-63	11-66
				<u>$F(4, 161) = 3.070, p = .018^*$</u>		

and hydrocephalus ($p = .002$), (Table 7). A two-way ANOVA of perinatal birth group with gender determined no statistical differences at birth in neonatal demographics or medical diagnoses (Appendix E). No differences in demographic data at age 23, was determined by a two-way ANOVA of perinatal birth group with gender (Appendix F).

Gender

There were slightly more females ($n = 86, 52\%$) than males ($n = 79, 48\%$) participants in the sample. At birth there were significant results for longer duration of oxygen use, $F(1,164) = 5.416, p = .021$, and for the diagnosis of meningitis, $X^2(1) = 4.514, p = .049$, in male participants (Appendix G). In contrast, female oxygen use, $F(1,164) = 5.416, p = .021$, and for the diagnosis of meningitis, $X^2(1) = 4.514, p = .049$, in male participants (Appendix G). In contrast, female participants at birth did not have any medical diagnoses of meningitis or hydrocephalus.

At age 23, there were statistical differences of the perinatal birth groups in gender distribution, as determined by Pearson's Chi-square test for Independence, $X^2(4) = 11.919, p = .018$ (Appendix H). Cramer's V test showed a weak association, $r^2 = .268, p = .018$, between gender and perinatal birth groups. Gender correlations among all dependent variables were below $r^2 = .80$. See Appendix I for further details. Statistically significant differences between males ($n = 22, 14\%$) and females ($n = 42, 27\%$) at age 23 were found for college education, $(X^2(1, 157) = 7.058, p = .008)$, with a weak association Phi $r^2 = .212$ (Appendix J).

Socioeconomic Status (SES)

As previously mentioned, the sample participants, at age 23 years old, SES mean score was 40.58 ($SD = 13.61$) indicating a moderately high SES with the majority of participants partially supported by their parent(s) (Table 8). SES at age 23, according to perinatal birth group, had mean scores ranging from 37.38 to 46.04 or from average to moderately high between groups. No statistically significant differences were determined by a one-way ANOVA between perinatal birth groups and SES at age 23 (Table 8).

Table 8

Age 23 SES for Total Sample, Perinatal Birth Group and Gender, N = 166

Age 23 Socioeconomic Status								
Age 23	Perinatal Birth Groups ¹						Gender	
	Sample	FT	HPT	MPT	NPT	SGA	M	F
<i>M</i>	40.58	42.04	37.38	40.73	40.18	41.60	39.05	41.95
<i>SD</i>	13.61	14.64	14.65	12.92	14.54	11.74	14.82	12.37
Range	9-69	13-69	17–60	9–61	10–67	22–61	9–69	10-61
Levene’s	$F(4,154) = .828, p = .515$					$F(1,157) = 5.438, p = .021^*$		
	[* Within/between = 86.91]							
Analysis	$F(4, 154) = .476, p = .753$					$F(1, 157) = 1.807, p = .181$		

¹ FT = Full Term; HPT = Healthy Preterm; MPT = Medical Preterm;

NPT = Neurological Preterm and SGA = Small for Gestational Age Preterm

SES mean scores for gender ranged from 39.05 ($SD = 14.82$) for males to 41.95

($SD = 12.37$) for females. A Levine's test resulted in significant results for gender and SES, $F(1,157) = 5.438, p = .021$, indicating unequal group variances (Table 8). The difference (86.91) between variance of males and females was less than 4 - 5 times of the smaller group and not significant, therefore an ANOVA can still be used (oak.ucc.vau.edu, 2016). A two-way ANOVA between perinatal birth group with gender and SES, at age 23, indicated no differences between perinatal birth groups with gender in SES (Table 9).

Table 9

Age 23 Mean SES for Perinatal Birth Group with Gender, N = 166

Age 23 Socioeconomic Status for Perinatal Birth Group with Gender										
	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
<i>N</i>	17	20	10	14	26	21	16	9	6	20
<i>M</i>	45.35	39.23	31.50	41.57	38.17	43.90	38.28	43.56	39.67	42.18
<i>SD</i>	16.79	12.27	15.13	13.46	13.20	12.1	15.20	13.44	10.69	12.24
Analysis Two-way ANOVA : $F(4,149) = 1.597, p = .178$										

No statistically significant differences between SES status at birth and age 23 were determined (Table 10). Although a one-way ANOVA of SES scores at birth for each perinatal birth group indicated there were significant differences, at the $p < .05$ level, between perinatal birth groups at birth in SES, $F(4, 161) = 3.070, p = .018$,

(Table 11 and 12). Post hoc comparisons using Tukey test indicated that the mean score difference at birth in SES was between HPT ($M = 34.96$, $SD = 15.31$) and the SGA ($M = 46.06$, $SD = 14.71$) participants. Overall the participants' socioeconomic status from birth to age 23 remained fairly consistent and without any statistical significant differences between perinatal birth groups at age 23.

Table 10

ANOVA Birth SES for Total Sample, Perinatal Birth Group, Gender and Perinatal Birth Group with Gender, $N = 166$.

Birth	Sample	Birth Socioeconomic Status					Gender	
		Perinatal Groups					M	F
		FT	HPT	MPT	NPT	SGA		
<i>M</i>	39.19	36.93	34.92	41.51	35.46	46.04	38.37	39.93
<i>SD</i>	14.60	14.62	15.31	13.78	13.00	14.71	15.20	24.08
Range	8–66	11–64	8–63	14–66	17–63	11–66	8-66	11-66
Levene's		$F(4,161) = .221, p = .926$				$F(1, 164) = .768, p = .382$		
ANOVA		$F(4, 161) = 3.070, p = .018^*$				$F(1, 164) = .474, p = .492$		

Summary

The sample has uneven perinatal birth sizes, near equal percentage in gender and primarily an equal distribution of SES scores from birth to the age 23-time point.

Table 11

Analysis of Variance for Perinatal Group with Gender, N = 166

<i>*Source</i>	<i>SS</i>	<i>df</i>	<i>MS</i>	<i>F</i>	<i>p</i>
Between	2492.929	4	623.232	3.070	.018*
Within	32680.282	161	202.983		
Total	35173.211	165			

Table 12

ANOVA Birth Socioeconomic Status by Perinatal Birth Group with Gender, N = 166

	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
<i>n</i>	18	23	10	14	28	21	17	9	6	20
<i>M</i>	41.11	33.65	38.40	32.43	40.18	43.29	32.82	40.44	37.33	48.65
<i>SD</i>	16.28	12.57	18.51	12.71	14.77	12.47	11.70	14.54	17.85	13.03
Analysis	$F(4,156) = 2.279, p = .063$									

Significant gender differences were found in the birth and age 23 demographics.

Males at birth had longer duration of oxygen use and all the meningitis diagnoses.

Females at age 23 had more college education than the males.

Dependent Variables

The dependent variables were all collected at age 23 years. Descriptive analysis of the dependent variables of stress, coping, emotional intelligence, emotional health and TSST salivary cortisol samples follows.

Daily Hassles Stress Score

The Daily Hassles Scale-Revised (DHS-R) was used to assess self-reported everyday stress and the magnitude of the stress. The samples weighed stress scores were above average ratings, ($M = 31.10$, $SD = 21.51$), with an overall range of 0 - 99, (Table 13). The dispersion of the Daily Hassles t -scores when converted into stress

Table 13

Daily Hassles Stress Scores for Sample, Perinatal Birth Group & Gender

	Perinatal Birth Group			
	n	M	SD	Min, Max
Sample	163	31.10	21.51	0-99
FT	40	35.30	23.88	0-95
HPT	24	26.04	17.01	5-77
MPT	49	28.84	19.55	2-82
NPT	25	28.20	22.13	2-99
SGA	25	36.60	23.64	3-83
Gender				
Male	79	29.82	23.26	0-99
Female	84	32.31	19.79	3-91

Figure 14

Distribution of Daily Hassles Rating Categories (N = 163)

ratings demonstrates the highest frequency in the total sample of below average stress ($n = 81$, 50%) followed by above average ($n = 70$, 43%) ratings (see Figure 14).

Figure 15

Distribution of Daily Hassles Stress Scores by Perinatal Birth Group

Perinatal birth group daily hassles stress scores ranged from below average for the healthy preterm (HPT), ($M = 26.04$, $SD = 17.01$) to above average for both the full term (FT), ($M = 35.30$, $SD = 23.88$) and small for gestational age (SGA), ($M = 36.60$, $SD = 23.64$) participants, (Table 13, Figure 15). The mean daily hassles stress scores were average for males ($M = 29.82$, $SD = 23.26$) and above average for females ($M = 32.31$, $SD = 19.79$; Table 13). Male participants had a slightly wider range in daily hassles stress (0 - 99) than females (3 - 91) although male mean scores were slightly lower than females. Overall, there was a widespread variation in daily hassles stress scores in both males and females.

Combined perinatal birth group with gender daily hassles stress scores (Table 14 and Figure 16) demonstrates SGA males with the highest mean score and widest deviation ($M = 39.17$, $SD = 27.75$) followed by the FT born males ($M = 37.72$, $SD = 24.62$). The lowest daily hassles stress scores, for the entire group, were found in the HPT males ($M = 17.50$, $SD = 12.25$). Overall females in the perinatal birth groups had mid-range daily hassles stress scores. In contrast, females in the perinatal birth group with gender had both the highest and lowest daily hassles stress scores. The highest female daily hassles stress scores: HPT ($M = 32.14$, $SD = 17.66$), medical (MPT), ($M = 29.19$, $SD = 17.11$) and neurological; (NPT), ($M = 29.75$, $SD = 10.90$). Males had the highest daily hassles stress score for FT ($M = 37.72$, $SD = 24.62$) and SGA ($M = 39.1$, $SD = 22.99$) perinatal birth groups.

Table 14

Daily Hassles Stress Scores for Perinatal Birth Group with Gender

Perinatal Birth Group with Gender			
	<i>n</i>	<i>M</i>	<i>SD</i>
FT			
Male	18	37.72	24.62
Female	22	33.32	23.65
HPT			
Male	10	17.50	12.25
Female	14	32.14	17.66
MPT			
Male	28	28.57	21.51
Female	21	29.19	17.11
NPT			
Male	17	27.47	26.09
Female	8	29.75	10.90
SGA			
Male	6	39.17	27.75
Female	19	35.79	22.99

Figure 16

Mean Daily Hassles Stress Scores for Perinatal Birth Group with Gender

Summary

In summary, the sample participants daily hassles stress scores were above average. The HPT had the lowest stress while the FT and SGA born had the highest. Daily hassles stress scores for males and females were similar with both genders having wide variations in stress responses. SGA males had the highest daily hassles stress scores and HPT born males had the lowest.

Coping

For the total sample, coping styles (CRI-A) scores ranged at or slightly above the average mean ($M = 50$, $SD = 10$) and within one standard deviation. (Table 15, Figure 17). The highest total sample score for coping occurred in emotional discharge, ($M = 57.13$, $SD = 9.41$) followed by cognitive avoidance, ($M = 54.53$, $SD = 9.72$) both of which are avoidance coping. The lowest scores for coping styles of the total sample occurred with seeking guidance and support ($M = 50.37$, $SD = 8.42$) followed by

logical analysis ($M = 50.74$, $SD = 9.89$), which are both, approach coping.

Figure 17

Mean Total Sample Coping Scores

The total sample widest ranges in scores occurred for emotional discharge (39 – 88) and problem solving (27 – 67). The lowest total sample ranges occurred in logical analysis (22) and problem solving (27) while the highest of low-range scores occurred in emotional discharge (39) and seeking alternative rewards (37). The highest scores in the upper-range limits occurred in emotional discharge (88) and both seeking alternative rewards (76) and cognitive avoidance (76).

All mean scores across perinatal birth groups for the eight coping style scores were within average limits (35-65; Table 15). Perinatal birth group coping mean scores were highest for emotional discharge in the FT group ($M = 58.36$, $SD = 10.46$) and lowest for logical analysis in the NPT group ($M = 46.40$, $SD = 9.05$).

Table 15

Distribution of Coping Style (CRI-A) Scores for Total Sample, Perinatal Birth Group and Gender

	<i>n</i>	<i>M</i>	<i>SD</i>	Min-Max
APPROACH				
Logical Analysis				
Sample	155	50.74	9.89	22-67
Perinatal Birth Group:				
FT	36	52.26	10.49	27-67
HPT	24	48.42	11.62	22-60
MPT	46	52.87	9.32	29-65
NPT	25	46.40	9.05	24-60
SGA	24	50.75	7.55	37-62
Gender				
Male	72	49.13	10.69	24-67
Female	83	52.13	8.98	22-67
Positive Reappraisal				
Sample	155	52.99	8.58	31-68
Perinatal Birth Group				
FT	36	52.39	9.81	34-68
HPT	24	54.42	8.32	36-68
MPT	46	54.74	7.88	38-68

	<i>n</i>	<i>M</i>	<i>SD</i>	Min-Max
NPT	25	50.28	7.79	34-60
SGA	24	51.92	8.63	31-62
Gender:				
Male	72	51.76	8.52	34-68
Female	83	54.05	8.54	31-68
Seeking Guidance & Support				
Sample	155	50.37	8.42	29-69
Perinatal Birth Group				
FT	36	52.39	8.12	37-64
HPT	24	49.29	9.35	34-69
MPT	46	51.15	8.35	29-69
NPT	25	47.88	8.40	34-59
SGA	24	49.79	7.86	37-64
Gender:				
Male	72	47.78	8.51	29-64
Female	83	52.61	7.71	37-69
Problem Solving				
Sample	155	52.83	10.01	27-67
Perinatal Birth Group				
FT	36	51.67	10.41	31-67
HPT	24	52.33	10.52	27-67

	<i>n</i>	<i>M</i>	<i>SD</i>	Min-Max
MPT	46	56.59	8.11	34-67
NPT	25	48.76	10.96	27-60
SGA	24	52.08	9.59	34-64
Gender				
Male	72	52.00	10.30	27-67
Female	80	53.38	9.87	30-67
AVOIDANCE				
Cognitive Avoidance				
Sample	155	54.53	9.72	34-76
Perinatal Birth Group				
FT	36	54.31	11.28	34-74
HPT	24	53.96	8.30	39-69
MPT	46	54.37	9.52	43-76
NPT	25	52.76	9.22	37-69
SGA	24	57.58	9.53	37-74
Gender				
Male	72	52.82	9.28	34-76
Female	83	56.01	9.91	37-74
Acceptance or Resignation				
Sample	155	52.83	9.41	33-73

	<i>n</i>	<i>M</i>	<i>SD</i>	Min-Max
Perinatal Birth Group				
FT	36	54.36	10.24	37-71
HPT	24	49.63	9.71	33-68
MPT	46	52.41	8.51	33-72
NPT	25	53.16	10.35	33-70
SGA	24	54.21	8.28	35-68
Gender				
Male	72	51.51	9.92	33-70
Female	83	53.98	8.85	33-73
Seeking Alternative Rewards				
Sample	154	53.79	9.04	37-76
Perinatal Birth Group				
FT	36	52.36	9.16	37-71
HPT	23	51.57	8.34	37-67
MPT	46	53.22	8.63	37-76
NPT	25	56.56	9.52	39-71
SGA	24	56.29	9.27	37-71
Gender				
Male	72	53.57	8.39	37-76
Female	82	53.99	9.63	37-71

	<i>n</i>	<i>M</i>	<i>SD</i>	Min-Max
Emotional Discharge				
Sample	155	57.13	10.15	39-88
Perinatal Birth Group				
FT	36	58.36	10.46	39-79
HPT	24	54.94	8.34	39-69
MPT	46	57.93	10.85	39-88
NPT	25	55.24	11.16	39-82
SGA	24	57.88	9.00	39-72
Gender				
Male	72	54.94	10.38	39-88
Female	83	59.02	9.62	39-82

Gender coping style mean scores between groups were highest in females for emotional discharge ($M = 59.02$, $SD = 9.62$) and lowest for males in seeking guidance and support ($M = 47.78$, $SD = 8.51$), (Table 15.). Male participants highest mean score was in emotional discharge ($M = 54.49$, $SD = 10.38$) while female participants lowest mean score was in logical analysis ($M = 52.13$, $SD = 8.98$).

Overall, the highest perinatal birth with gender group coping style mean score was in the female NPT for emotional discharge ($M = 61.44$, $SD = 12.03$) (Appendix K). In contrast the lowest mean score for perinatal birth group with gender coping style scores was in the male NPT for seeking guidance and support ($M = 44.63$, $SD = 8.58$).

Summary

In summary, the sample participants have the highest coping style scores for emotional discharge and the lowest scores for seeking guidance and support. Perinatal birth group coping scores were highest for emotional discharge in the FT participants and lowest for logical analysis in the NPT participants. Overall the MPT had the highest total approach scores followed by the FT, HPT, SGA and NPT groups. Avoidance coping total scores were highest in the SGA, followed by the FT, MPT, NPT and HPT perinatal birth groups.

Gender coping scores between groups were highest in females for emotional discharge and lowest for males in seeking guidance and support. Total approach and avoidance scores were highest for females. Both the highest and lowest coping scores in the perinatal birth group with gender were for the NPT born. NPT highest coping score was for females in emotional discharge and lowest score for males in seeking guidance and support.

Emotional Intelligence

Emotional intelligence (EQi) was measured using the Bar-on Total EQi score. The total sample EQI score mean of 98.78 ($SD = 15.59$) meets the standardized score mean of 100 ($SD = 15$), (Table 16). Perinatal birth group differences in means ranged from 96.42 ($SD = 15.43$) for the HPT and 101.08 ($SD = 14.78$) for the NPT born participants. Gender means (SD) and ranges were: 97.88 ($SD = 15.73$), 65 – 123 for males and 99.51 ($SD = 15.53$), 62 – 132 for females. In the perinatal birth group with gender, MPT males, had the highest EQi mean score ($M = 101.95$, $SD = 14.80$). SGA males, had the lowest mean score and widest SD range ($M = 88.00$, $SD = 23.58$).

Table 16

EQi Scores for Sample, Perinatal Birth Group and Gender

<i>n</i>	<i>M</i>	<i>SD</i>	Min, Max	
Sample	158	98.75	15.59	62-132
Perinatal Birth Group				
FT	38	98.37	17.23	62-132
HPT	24	96.42	15.43	68-120
MPT	47	99.17	14.99	65-129
NPT	25	101.08	14.78	73-124
SGA	24	98.46	15.96	62-123
Gender				
Male	73	97.88	15.73	65-123
Female	85	99.51	15.53	62-132

Table 16). Total EQi mean scores for perinatal birth groups with gender controlled were the same as the perinatal birth group EQi means.

Summary

In summary, total EQi scores were within the standard mean and standard deviation for the total sample. The NPT born 23 year olds had the highest EQi scores for perinatal birth groups. NPT females EQi scores were higher than male NPT EQi

Table 17

EQi Total Scores for Perinatal Birth Group with Gender

	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
n	17	21	10	14	26	21	16	9	4	20
<i>M</i>	97.47	99.10	100.60	93.43	96.92	101.95	100.63	101.89	88.00	100.55
<i>SD</i>	15.52	18.85	17.36	13.77	15.07	14.80	14.81	15.54	23.58	13.89
Analysis Two-way ANOVA: $F(4,148) = .977, p = .422$										

scores. Lower EQi scores were found for males when compared to females. Males had the widest standard deviation when compared across all groupings.

Emotional Health

Emotional health scores, as represented by the mean total problem t - score of the Adult Self Report (ASR) for the entire sample, ($M = 48.39$, $SD = 10.47$, Range 26 – 82) were within normal limits ($M = 50$, with wide varying ranges), (Table 18). Perinatal birth group ASR mean scores ranged from 46.48 ($SD = 9.76$) for the NPT participants to 50.40 ($SD = 10.88$) for the FT born. The gender group mean scores for ASR were similar (male: $M = 48.48$, $SD = 10.57$; female $M = 48.30$, $SD = 10.44$; Table 18). In the perinatal birth group with gender, female HPT participants, had the highest mean ASR scores (51.86, $SD = 12.92$; Table 19). In contrast, females in MPT group had the lowest mean ASR scores ($M = 45.19$, $SD = 9.59$). Perinatal birth group with gender controlled mean ASR scores were the

same as the perinatal birth groups.

Table 18

Emotional Health (ASR) Scores for Total Sample, Perinatal Birth and Gender

	<i>n</i>	<i>M</i>	<i>SD</i>	Min, Max
Sample	163	48.39	10.47	26-82
Perinatal Group				
FT	40	50.40	10.88	26-82
HPT	24	49.38	11.12	32-74
MPT	49	47.10	9.85	26-67
NPT	25	46.48	9.76	26-63
SGA	25	48.64	11.17	29-70
Gender				
Male	79	48.48	10.57	26-82
Female	84	48.30	10.44	29-74

Categories of risk for emotional health were also derived according to the ASR t-score DSM - IV categories (Table 20). In the total sample there were 14 (9%) participants who were in the borderline clinical range (60 - 63) and 13 (8%) participants who were in the clinical problem range (≥ 64 ($n = 27$, 17%). All

Table 19

Emotional Health (ASR) Scores for Perinatal Birth Group with Gender

	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
<i>N</i>	18	22	10	14	28	21	17	8	6	19
<i>M</i>	51.06	49.86	45.90	51.86	48.54	45.19	46.41	46.63	50.67	48.00
<i>SD</i>	11.75	10.36	7.22	12.92	9.96	9.59	10.93	7.33	14.28	10.39

perinatal birth groups had participants at borderline clinical range while the male ($n=13$, 6%) and female ($n=14$, 9%) participants had a near equal number at borderline clinical range.

The FT group had the highest frequency ($n=4$) of scores in the clinical problem range. The MPT group had the highest frequency ($n=4$) of scores in the clinical borderline range. The female FT and female HPT groups had the highest frequencies ($n=3$) of clinical problem scores.

In contrast, both females and males in the NPT group and males in the HPT group had no clinical problem scores. The highest frequencies ($n=3$) of borderline clinical range occurred in males from both of the MPT and NPT groups.

The decision to consider any scores above normal (≥ 60) as clinical problem scores resulted in recoding of the variable measuring ASR frequency categories. The

Table 20

Normal, Borderline, & Clinical Emotional Health (ASR) Mean Scores for Total Sample and Perinatal Birth Group, Gender and Perinatal Birth Group with Gender

	Sample	FT	HPT	MPT	NPT	SGA	M	F		
	<i>n %</i>									
Normal <59	135 (83)	33 (20)	19 (12)	41(25)	22(14)	20(12)	66 (41)	69 (43)		
Border 60-63	14 (9)	3 (2)	2 (1)	4 (3)	3 (2)	2(1)	9 (6)	5 (3)		
Clinical => 64	13 (8)	4 (3)	3 (2)	3 (2)	0	3(2)	4 (3)	9 (6)		
Analysis	$X^2 = (8,162) = 4.064, p = .851$				$X^2 (2,162) = 3.036, p = .219$					
	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
	<i>n (%)</i>									
Normal	15(19)	18(22)	10(13)	9(11)	23(29)	18(22)	14(18)	8(10)	4(5)	16(19)
Border	2(11)	1(5)	0	2(14)	3(11)	1(5)	3(18)	0	1(17)	1(5)
Clinical	1(6)	3(14)	0	3(21)	2(7)	1(5)	0	0	1(17)	2(11)

normal, borderline clinical problem and the clinical problem range scores were coded as non-clinical (< 59) and clinical problem (60 - ≥64) scores, Table 21.

There were 27 clinical problems representing 16% of the total sample participants. The FT and MPT had the highest frequencies (both $n = 7$) of clinical problem scores in

contrast to the NPT group with the lowest frequencies ($n = 3$) of clinical problem scores. Males ($n = 13$) and females ($n = 14$) were similar in frequencies for clinical problems. The female HPT and unlike previous findings, the males in the MPT groups, from the perinatal with gender groups, had the highest frequencies ($n = 5$) of clinical problem scores. No clinical problem scores occurred in males from the HPT

Table 21

Non-Clinical and Clinical Problem Scores for the Sample, Perinatal

Birth Group, Gender and Perinatal Group with Gender.

	Sample	FT	HPT	MPT	NPT	SGA	M	F		
	<i>n %</i>									
Non-Clinical	135(83)	33(20)	19(12)	41(25)	22(14)	20(14)	66(41)	69(42)		
Clinical	27(17)	7(4)	5(3)	7(4)	3(2)	5(3)	13(8)	14(9)		
$X^2 = (4,162) = 1.062, p = .900$ $X^2 = (1,162) = .005, p = 1.00$										
	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
	<i>n (%)</i>									
Non-Clinical	15(19)	18(22)	10(13)	9(11)	23(29)	18(22)	14(18)	8(10)	4(5)	16(19)
Clinical	3(4)	4(5)	0	5(6)	5(6)	2(2)	3(4)	0	2(3)	3(4)

and in females from the NPT groups. These frequencies, now with the exclusion of the males in the NPT, were otherwise constant with the previous counts.

Summary

The mean emotional health (ASR) scores for the total sample, perinatal groups, gender and perinatal group with gender were all within normal range (< 59). Total clinical problems comprised 16% of the sample (27/162). The highest frequencies of clinical problems were in the FT and MPT perinatal birth groups ($n = 7$) and were also evenly distributed between genders. Female HPT and male MPT had the highest frequencies of clinical problems in the perinatal with gender group.

The Trier Social Stress Test Salivary Cortisol Samples

The Trier Social Stress Test (TSST) salivary samples are collected at 8 intervals: Two samples prior to the task and 20, 30, 45, 60, 75 and 90 after completion. The TSST eight salivary cortisol samples have been winsorized and delta, peak, slope and AUCg were calculated. Values that were not within the recommended range of a normal distribution (ie., skewness < 2 and kurtosis < 7 ; Tabachnick & Fidell, 2012) underwent a natural log transformation. These transformed variables were then used in all analyses. Descriptive statistics and bar graphs representing the cortisol response follow.

The sample distribution of TSST salivary cortisol levels has an asymmetrical distribution, as expected, with a longer tail to the right and shorter tail to the left. Quicker increases in cortisol values, in response to the acute TSST stressor, were followed by slower incremental decreases in cortisol values during the recovery phase

which is physiologically expected.

The cortisol values of delta, peak and slope for the perinatal birth groups, demonstrates the widest means in all values for the NPT perinatal birth group. In descending order of the highest means for delta, peak and slope, next is the MPT, FT, SGA and lastly the HPT perinatal birth group. (See Table 22 & Figure 18 - 22). The HPT and SGA participants have the lowest delta value response. In contrast the MPT and NPT participants have the higher delta value.

The overall pattern of cortisol high and low values are consistent across the perinatal birth groups for delta (baseline), peak (response) and slope (recovery). In other words if one perinatal birth group values were high for delta they were also high for peak and slope.

Figure 18

Cortisol Delta Values for Perinatal Birth Group

Table 22. *Cortisol Values for Total Sample*

	N Statistic	Minimum Statistic	Maximum Statistic	Mean Statistic	Std. Deviation Statistic	Skewness Statistic	Std. Error	Kurtosis Statistic	Std. Error
Delta	149	-1.104	2.540	.64510	.74660	.310	.199	-.460	.395
Peak	149	-2.736	.883	-1.21014	.78078	.365	.199	-.265	.395
Slope	148	-.01472	.03387	.00821	.00959	.362	.199	-.483	.395
AUCg	142	1.997	4.722	3.17294	.52793	.384	.203	.340	.404
Valid N (Pairwise)	As above								

Table 23. *Cortisol Values by Perinatal Full Term*

	N Statistic	Minimum Statistic	Maximum Statistic	Mean Statistic	Std. Deviation Statistic	Skewness Statistic	Skewness Std. Error	Kurtosis Statistic	Kurtosis Std. Error
Delta	33	-1.104	1.992	.62199	.83044	.006	.409	-.730	.798
Peak	33	-2.736	.883	-1.26036	.82005	.678	.409	.724	.798
Slope	33	-.01472	.02656	.00801	.01076	.114	.409	-.700	.798
AUCg	33	1.997	4.722	3.17385	.58376	.503	.409	1.009	.798
Valid N (Pairwise)	33								

Table 24. *Cortisol Values by Perinatal Healthy Preterm*

	N Statistic	Minimum Statistic	Maximum Statistic	Mean Statistic	Std. Deviation Statistic	Skewness Statistic	Std. Error	Kurtosis Statistic	Std. Error
Delta	20	-.389	1.368	.40093	.46267	.036	.512	-.444	.992
Peak	20	-2.519	.512	-1.44036	.70056	.983	.512	1.820	.992
Slope	20	-.00519	.01824	.00513	.00605	.213	.512	-.305	.992
AUCg	19	2.036	4.700	3.06985	.55919	1.201	.524	3.503	1.014
Valid N (Pairwise)	20 & 19								

Table 25. *Cortisol Values by Perinatal Medical Preterm*

	N Statistic	Minimum Statistic	Maximum Statistic	Mean Statistic	Std. Deviation Statistic	Skewness Statistic	Std. Error	Kurtosis Statistic	Std. Error
Delta	47	-.768	2.540	.74067	.76901	.221	.347	.591	.681
Peak	47	-2.535	.014	-1.13901	.68635	-.258	.347	-.883	.681
Slope	47	-.01924	.03387	.00954	.00981	.209	.347	-.239	.681
AUCg	45	2.159	4.240	3.2165	.49371	.023	.354	-.233	.695
Valid N (Pairwise)	47 & 45								

Table 26. *Cortisol Values by Neurological Preterm*

	N Statistic	Minimum Statistic	Maximum Statistic	Mean Statistic	Std. Deviation Statistic	Skewness Statistic	Std. Error	Kurtosis Statistic	Std. Error
Delta	25	-.456	2.319	.80237	.77234	.110	.464	-1.034	.902
Peak	25	-2.526	.812	-53.533	.92529	-.198	.464	-.759	.902
Slope	24	-.00608	.03092	.01007	.01020	.265	.472	-.959	.918
AUCg	23	2.235	4.301	3.31604	.62245	-.283	.481	-.845	.935
Valid N (Pairwise)	As above								

Table 27. *Cortisol Values by Small for Gestational Age Preterm*

	N Statistic	Minimum Statistic	Maximum Statistic	Mean Statistic	Std. Deviation Statistic	Skewness Statistic	Std. Error	Kurtosis Statistic	Std. Error
Delta	24	-.297	2.341	.52955	.73026	.919	.472	.043	.902
Peak	24	-2.397	.792	-1.33087	.15894	1.125	.472	1.051	.902
Slope	24	-.00283	.02601	.00662	.00904	.882	.472	-.460	.918
AUCg	22	2.549	3.852	3.02191	.06988	.636	.401	.465	.935

Valid N (Pairwise) 24 & 22

Table 28. *Cortisol Values for Males*

	N Statistic	Minimum Statistic	Maximum Statistic	Mean Statistic	Std. Deviation Statistic	Skewness Statistic	Std. Error	Kurtosis Statistic	Std. Error
Delta	72	-.788	2.540	.78668	.78486	.198	.283	-.760	.559
Peak	72	-2.526	.358	-1.03552	.72346	-.290	.283	-.851	.559
Slope	72	-.00696	.03387	.01020	.01016	.267	.283	-.826	.559
AUCg	70	2.036	4.301	3.31035	.49641	-.130	.287	-.114	.566
Valid N (Pairwise)	72 & 70								

Table 29. *Cortisol Values for Females*

	N Statistic	Minimum Statistic	Maximum Statistic	Mean Statistic	Std. Deviation Statistic	Skewness Statistic	Std. Error	Kurtosis Statistic	Std. Error
Delta	77	-1.104	2.540	.051271	.68794	.329	.274	-.100	.541
Peak	77	-2.736	.883	-1.37342	.80144	.980	.274	1.086	.541
Slope	76	-.01472	.02656	.00633	.00867	.316	.276	-.208	.545
AUCg	72	1.997	4.722	3.03936	.52660	.972	.283	2.026	.559
Valid N (Pairwise)	As above								

Figure 19

Cortisol Peak Values for Perinatal Birth Group

Figure 20

Cortisol Slope Values for Perinatal Birth Group

Figure 21

Cortisol AUCg Values for Perinatal Birth Group

In contrast the values for the perinatal birth AUCg (total concentration across the TSST) follow a different pattern. The highest AUCg values were in the NPT, followed by the MPT, and then FT group.. The differences in the order between the AUCg and other cortisol values, show the HPT values next and lastly the SGA group.

Gender

Gender TSST cortisol values show higher means of cortisol responses across delta, peak, slope and AUCg for males than for female participants (Tables 28 & 29), Figures 22 - 25).

Figure 22

Cortisol Delta Values for Gender

Figure 23

Cortisol Peak Values for Gender

Figure 24

Cortisol Slope Values for Gender

Figure 25

Cortisol AUCg Values for Gender

Perinatal Birth Group with Gender

In the perinatal with gender birth group, males from the NPT group have the highest delta value while females in the SGA have the lowest (Figure 26). Peak values are also highest for males in the NPT group and instead lowest for the females in the MPT (Figure 27). Slope values are again highest for NPT males and lowest for NPT females (Figure 28). AUCg values are highest in the perinatal with gender group for NPT males and lowest for SGA females (Figure 29).

Summary

The NPT born 23 year olds have the highest mean cortisol values for delta, peak and slope while the HPT born have the lowest. AUCg concentration were highest in the NPT and lowest in the SGA. All female cortisol values were lower than males. Males in the NPT have the highest cortisol values in delta, peak, slope and AUCg from the perinatal birth groups with gender. Females born SGA were lowest in delta and AUCg values. Peak values were lower in females born MPT while the lowest values for slope were in females from the NPT born for the perinatal birth group with gender.

Figure 26

Cortisol Delta Values for Perinatal Birth Group with Gender

Figure 27

Cortisol Peak Values for Perinatal Birth Group with Gender

Figure 28

Cortisol Slope Values for Perinatal Birth Group with Gender

Figure 29

Cortisol AUCg Values for Perinatal Birth Group with Gender

Summary

Daily hassles stress score were highest for the SGA and lowest for the HPT groups. Females reported more daily hassles stress than males although both have wide ranges in scores. The highest score, for the total sample, were in emotional coping and the lowest score were in seeking guidance and support coping styles. The MPT group had the most approach coping styles while the SGA group had the lowest for the perinatal birth groups. Females overall have the highest scores for both approach and avoidance coping styles. EQi scores were all within normal range with the highest score for the NPT and lowest score for the HPT groups. EQi scores with the highest frequencies of clinical problems were in the FT and MPT perinatal birth groups.

Delta, peak and slope cortisol values were highest in the NPT and lowest in the HPT born. Cortisol total concentration levels, measured as AUCg, were highest in the NPT and lowest in the SGA perinatal birth groups. Females have the lower cortisol values across delta, peak, slope and AUCg. NPT males continue to have the highest values in delta, peak, slope and AUCg means from the perinatal birth group with gender. Delta and AUCg values were lowest in females born SGA, peak values were lower in females born MPT, and the lowest values for slope were in females from the NPT for the perinatal birth group with gender.

Hypotheses Testing

The three hypotheses are statistically analyzed according to the three study aims.

1. Compare effect of prematurity on stress, coping, emotional intelligence and emotional health.
2. Compare the salivary cortisol response between premature and full-term born infants in the social stress paradigm of the TSST.
3. Examine the relationship between effect of emotional health and on the stress recovery period of the TSST measured in salivary cortisol.

Hypothesis One

Hypothesis 1: Higher self - reported stress scores, higher use of avoidance coping styles, lower emotional intelligence scores and more emotional health disorders will be found for the adults at age 23 years born prematurely compared to the full-term born adults. Hypothesis 1 will be significant if DHS-R total stress scores are higher, EQi total scores are lower, ASR scores are higher, and CRI-A avoidance t - scores are higher, for the premature groups.

Pearson's Correlations were determined for all variables of hypothesis one. The correlations were not statistically significant $r = \geq .80$ were found (Table 30).

Stress

An ANCOVA, with α set at $p = 0.05$, was used to determine any perinatal birth group differences in DHS-R scores, after controlling for the effects of SES. Levene's test was not statistically significant. The ANCOVA determined no statistically significant effect of SES on perinatal birth group and DHS-R mean scores, $F(4, 150) = 1.447, p = .221$, with an intercept of $p = < .001$. See Tables 31, 32, 33, and 34 for further details.

Table 30

Correlation Matrix for Hypothesis One

Variables	(1)	(2)	(3)	(4)	(5)	(6)
(1) Perinatal Birth Group	-					
(2) Daily Hassles Stress	-.003	-				
(3) Emotional Intelligence (EQi)	.039	-.424**	-			
(4) Emotional Health (ASR)	-.014	.610**	-.481**	-		
(5) Logical Analysis	-.088	.061	.201*	-.131	-	
(6) Positive Appraisal	-.061	.020	.219**	-.107	.638**	-
(7) Seeking Guidance and Support	-.111	.044	.230**	-.079	.449**	.386**
(8) Problem Solving	-.020	-.167*	.312**	-.192*	.612**	.561**
(9) Cognitive Avoidance	.065	.408**	-.336**	.330**	.147	.048
(10) Acceptance or Resignation	.021	.382**	-.233**	.253**	.064	.008
(11) Seeking Alternative Rewards	.182*	-.015	.114	-.171*	.411**	.373**
(12) Emotional Discharge	-.022	.481**	-.300**	.291**	.132	.074

Variables	(7)	(8)	(9)	(10)	(11)	(12)
(8) Problem Solving	.363**	-				
(9) Cognitive Avoidance	.035	-.012	-			
(10) Acceptance or Resignation	.219**	-.103	-.434**	-		
(11) Seeking Alternative Rewards	.211**	.362**	.065	.067	-	
(12) Emotional Discharge	.197*	.075	.529**	.390**	.151	-

** $p = 0.01$ (2-tailed)

Table 31

Descriptive Statistics: Perinatal Birth Group for Daily Hassles Stress

Daily Hassles Stress (DHS-R)	Perinatal Birth Group			
	<i>n</i>	<i>M</i>	<i>SE</i>	95% CI
Full Term	40	35.30	3.78	[27.66, 42.94]
Healthy Preterm	24	26.04	3.47	[18.86, 33.22]
Medical Preterm	49	28.84	2.79	[23.22, 34.45]
Neurological Preterm	25	28.20	4.43	[19.07, 37.33]
Small for Gestational Age	25	36.60	4.73	[26.84, 46.36]

Table 32

Analyses of Variance Perinatal Birth Group for Daily Hassles Stress

Daily Hassles Stress (DHS)	Perinatal Birth Group				
	SS	df	MS	F	p
Between	2537.18	4	643.29	1.384	.242
Within	72416.05	158	458.33		
Total	74953.23	162			

Table 33

Descriptive Statistics: Perinatal Birth Group and DHS-R & SES as Covariate

Daily Hassles Stress	Perinatal Birth Group with Covariate SES			
	n	M	SE	95% CI
Full Term	36	35.46	5.59	[28.39, 42.54]
Healthy Preterm	24	25.23	4.41	[16.53, 33.94]
Medical Preterm	47	28.69	3.13	[22.51, 34.88]
Neurological Preterm	24	29.40	4.38	[20.74, 38.06]
Small for Gestational Age	25	36.91	4.30	[28.42, 45.40]

Table 34

ANCOVA: Perinatal Birth Group and DHS-R with Covariance SES

Perinatal Birth Group with Covariate SES					
Daily Hassles Stress	SS	df	MS	F	p
Corrected Model	3690.82	5	738.16	1.603	.163
Intercept	24061.34	1	24061.34	52.235	< .001
Socioeconomic Status	1394.37	1	1394.37	3.027	.080
Perinatal Birth Group	2665.50	4	666.37	1.447	.221
Error	69094.79	150	460.63		
Total	224131.00	156			
Corrected Total	72785.61	155			

An ANCOVA, with α of $p = 0.05$, was used to determine any perinatal birth group with gender differences in DHS-R scores, after controlling for the effects of SES. Levene's test was not statistically significant. The ANCOVA determined a statistically significant relationship of SES on DHS-R scores: $F(1, 145) = 4.435$, $p = .037$, partial $\eta^2 = .030$, indicating a small effect, with an intercept of $p = .001$. There were no statistically significant effects, as determined by ANCOVA, on perinatal birth group with gender mean DHS-R scores after controlling for SES: $F(4, 145) = 1.013$, $p = .403$, partial $\eta^2 = .027$, indicating a small effect, with an intercept of $p = .001$. See Tables 35 - 42 for further details.

Table 35

Descriptive Statistics: Gender and DHS-R

Daily Hassles Stress	Gender				
	<i>n</i>	<i>M</i>	<i>SD</i>	<i>SE</i>	95% CI
Male	79	29.82	23.27	2.62	[24.61, 35.03]
Female	84	32.31	19.79	2.16	[28.01, 36.60]

Table 36

ANOVA of Gender on DHS-R at Age 23 Years

Daily Hassles Stress	Gender				
	SS	<i>df</i>	<i>MS</i>	<i>F</i>	<i>p</i>
Between	251.756	1	251.756	.543	.462
Within	74701.471	161	463.984		
Total	74953.227	162			

Table 37

Descriptive Statistics: Gender and DHS-R with Covariate of SES

Daily Hassles Stress	Gender with Covariate SES		
	<i>n</i>	<i>M</i>	<i>SD</i>
Male	75	29.88	23.49
Female	81	32.32	19.91

Table 38.

Analysis of Gender and DHS-R with Covariate of SES

Daily Hassles Stress	Gender with Covariate SES				
	<i>SS</i>	<i>df</i>	<i>MS</i>	<i>F</i>	<i>p</i>
Corrected Model	1418.77	2	709.39	1.521	.222
Intercept	23224.69	1	23224.69	49.790	< .001
Socioeconomic Status	1186.74	1	1186.74	2.544	.113
Gender	393.45	1	393.45	.843	.360
Error	71366.84	153	446.45		
Total	224131.00	156			
Corrected Total	72785.61	155			

Table 39

Descriptive Statistics: Perinatal Birth Group with Gender for DHS-R

Perinatal Birth Group by Gender			
Daily Hassles Stress	<i>n</i>	<i>M</i>	<i>SD</i>
Full Term			
Male	18	37.72	24.62
Female	22	33.32	23.65
Healthy Preterm			
Male	10	17.50	12.25
Female	14	32.14	17.66
Medical Preterm			
Male	28	28.57	21.51
Female	21	29.19	17.11
Neurological Preterm			
Male	17	27.47	26.09
Female	8	29.75	10.90
Small for Gestational Age			
Male	6	39.17	27.75
Female	19	35.79	22.99

Table 40

Analysis of Variance Perinatal Birth Group with Gender

Perinatal Birth Group with Gender					
Daily Hassles Stress	SS	<i>df</i>	<i>MS</i>	<i>F</i>	<i>p</i>
Corrected Model	4064.81	9	451.65	.975	.463
Intercept	127140.27	1	127140.27	274.410	< .001
Perinatal Birth Group	2805.11	4	701.28	1.514	.201
Gender	125.52	1	125.52	.271	.603
Perinatal Birth Group by Gender	1470.96	4	357.74	.794	.531
Error	70888.42	153	463.32		
Total	232652.00	163			
Corrected Total	74953.23	162			

Table 41

Descriptive Statistics: Perinatal Birth Group with Gender & DHS-R with Covariate SES

Perinatal Birth Group by Gender with Covariate SES			
Daily Hassles Stress	<i>n</i>	<i>M</i>	<i>SD</i>
Full Term			
Male	17	36.94	25.51
Female	19	33.53	24.66
Healthy Preterm			
Male	10	17.50	12.25
Female	14	32.14	17.66
Medical Preterm			
Male	26	28.38	22.22
Female	21	29.19	17.11
Neurological Preterm			
Male	16	29.06	26.08
Female	8	29.75	10.90
Small for Gestational Age			
Male	6	39.17	27.75
Female	19	35.79	22.99

Table 42

Analysis Perinatal Birth Group with Gender & DHS-R with Covariate SES

Perinatal Birth by Gender with Covariate SES					
Daily Hassles Stress	SS	df	MS	F	p
Corrected Model	5763.85	10	576.39	1.247	.266
Intercept	24979.86	1	24979.86	54.043	< .001
Socioeconomic Status	2049.96	1	2049.96	4.435	.037 *
Perinatal Birth Group	3075.90	4	768.98	1.664	.162
Gender	317.44	1	317.44	.687	.409
Perinatal Birth Group					
by Gender	1873.05	4	468.26	1.013	.403
Error	67021.76	145	462.22		
Total	224131.00	156			
Corrected Total	72785.61	155			

Coping

Coping styles were divided into separate analyses of approach coping and avoidance coping. The approach coping variables consist of: Logical analysis, positive reappraisal, seeking guidance and support and problem solving. The avoidance coping variables consist of: Cognitive avoidance, acceptance or resignation, seeking alternative rewards and emotional discharge.

Perinatal Birth Groups

A one-way MANOVA with an α of $p = 0.05$, and was used to determine if there were any effects of prematurity on approach coping styles. The IV was perinatal birth group and the DV's were logical analysis, positive appraisal, seeking guidance and support, and problem solving. All Levene's tests for homogeneity of variances, Box's and Tukey's tests were not statistically significant.

No statistically significant differences were found at the macro level, though it approached significance: Wilk's $\Lambda = .165$, [$F(16, 600) = 1.618$, $p = .059$, partial $\eta^2 = .041$ with an intercept of $>.001$. Table 43 shows the means, *SD*, *SE* and 95% CI results. ANOVA's determined statistically significant differences across perinatal birth groups for logical analysis, ($p = .048$) and problem solving ($p = .002$) problem solving, (Table 44).

Significant differences across perinatal birth groups were found in pairwise comparisons for logical analysis, positive appraisal and problems solving. Logical analysis was statistically significant for FT and NPT, $p = .016$ and MPT and NPT, $p = .008$. Positive Appraisal was statistically significant for MPT and NPT, $p = .037$. Problem Solving was statistically significant for FT and MPT, $p = .025$ and MPT and NPT, $p = .002$. See Table 45 for further information. Post-Hoc Tukey Tests were statistically significant and confirmed only problem solving between MPT and NPT, $p = .013$.

Table 43

Descriptive Statistics for Approach Coping for Prenatal Birth Groups

	<i>n</i>	<i>M</i>	<i>SD</i>	<i>SE</i>	95% CI
Logical Analysis					
FT	36	52.56	10.49	1.62	[49.36, 55.75]
HPT	24	48.42	11.62	1.98	[44.50, 52.33]
MPT	46	52.87	9.32	1.43	[50.04, 55.70]
NPT	25	46.40	9.05	1.94	[42.56, 50.24]
SGA	24	50.75	7.55	1.98	[46.83, 54.67]
Positive Appraisal					
FT	36	52.39	8.32	1.42	[49.58, 55.20]
HPT	24	54.42	8.32	1.74	[50.96, 57.86]
MPT	46	54.74	7.88	1.26	[52.35, 57.23]
NPT	25	50.28	7.79	1.71	[46.91, 53.65]
SGA	24	51.92	8.63	1.74	[48.48, 55.36]
Seeking Guidance and Support					
FT	36	52.19	8.12	1.40	[48.43, 54.96]
HPT	24	49.29	9.37	1.71	[45.91, 52.68]
MPT	46	51.15	8.35	1.24	[48.71, 53.60]
NPT	25	47.88	8.40	1.68	[44.56, 51.20]
SGA	24	49.79	7.86	1.71	[46.41, 53.18]

	<i>n</i>	<i>M</i>	<i>SD</i>	<i>SE</i>	95% CI
Problem Solving					
FT	36	51.67	10.41	1.63	[48.50, 54.88]
HPT	24	52.33	10.52	1.99	[48.39, 56.27]
MPT	46	56.59	8.11	1.44	[53.74, 59.43]
NPT	25	48.76	10.98	1.95	[44.90, 52.62]
SGA	24	52.08	9.59	1.99	[48.14, 56.02]

Table 44

Analysis of Variance of Perinatal Group for Logical Analysis and Problem Solving at Age 23

	SS	<i>df</i>	MS	<i>F</i>	p	Partial η^2
Logical Analysis	927.72	4, 150	231.93	2.459	.048*	.062
Problem Solving	1131.42	4, 150	282.85	2.965	.002*	.073

Table 45

Pairwise Differences Between Perinatal Birth Groups in Logical Analysis, Problem Solving and Positive Appraisal.

	Mean			
	Difference	SE	p	95% CI
Logical Analysis				
FT and NPT	6.156	2.53	.016	[1.160, 11.151]
MPT and NPT	6.470	2.41	.008	[1.702, 11.237]
Positive Appraisal				
MPT and NPT	4.459	2.12	.037	[-.270, 8.648]
Problem Solving				
FT and MPT	4.920	2.17	.025	[-.626, 9.215]
MPT and NPT	7.827	2.43	.002	[3.032, 12.622]
¹ Post-hoc	7.83	2.43	.013*	[1.13, 14.53]

¹ Tukey Multiple Comparisons

Approach coping styles of logical analysis ($p = .048$) and problem solving ($p = .002$) mean differences were statistically significant across perinatal birth groups. Logical analysis scores were statistically significantly lower in the NPT and highest in the FT ($p = .016$) while MPT scores were higher than NPT ($p = .008$) perinatal birth groups. The positive appraisal scores were the lowest in the NPT and highest in the MPT ($p = .037$) perinatal birth group. Problem solving mean scores were the highest

in the MPT and differed from the lower scores in the FT ($p = .025$), while the NPT scores were the lowest compared to the highest MPT ($p = .002$). Differences between MPT and NPT perinatal birth groups in problem solving were confirmed by Tukey's multiple comparisons ($p = .013$).

A MANCOVA, with an α of $p = 0.05$, and was used to determine if there were any effects of perinatal birth group with SES controlled across the DV's of approach coping styles. Box's and Levene's test were not statistically significant. No statistically significant differences were found in the perinatal birth group, after controlling for the effects of SES, on logical analysis, positive appraisal, seeking guidance and support, and problem solving coping.

A MANCOVA, with an α of $p = 0.05$, and was used to determine if there were any effects of perinatal birth group with SES controlled across the DV's of avoidance coping styles. Box's and Levene's test were not statistically significant.

Table 46

Descriptive Statistics: Avoidance Coping for Prenatal Birth Groups

	<i>n</i>	<i>M</i>	<i>SD</i>
Cognitive Avoidance			
FT	36	54.31	11.28
HPT	23	54.39	8.21
MPT	46	54.37	9.52
NPT	25	52.76	9.22
SGA	24	57.58	9.53

	<i>n</i>	<i>M</i>	<i>SD</i>
Acceptance or Resignation			
FT	36	54.36	10.24
HPT	23	49.79	9.91
MPT	46	52.41	8.15
NPT	25	53.16	10.35
SGA	24	54.21	8.28
Seeking Alternative Rewards			
FT	36	52.36	9.16
HPT	23	51.57	8.34
MPT	46	53.22	8.63
NPT	25	56.56	9.52
SGA	24	56.29	9.27
Avoidance Coping continued			
Emotional Discharge			
FT	36	58.36	10.46
HPT	23	55.30	8.33
MPT	46	57.93	10.85
NPT	25	55.24	11.16
SGA	24	57.88	9.00

No statistically significant differences were determined by MANCOVA for SES effects on avoidance coping: Wilk's $\Lambda = .943$ [$F(4, 145) = 2.183$], $p = .074$, partial $\eta^2 = .057$, with an intercept of $p = < .001$. No statistically significant effects for perinatal birth group with SES controlled were determined:

Wilk's $\Lambda = .885$ [$F(16, 444) = 1.131$], $p = .323$, partial $\eta^2 = .030$, with an intercept of $p = < .001$.

Gender

A one-way MANOVA with an α of $p = 0.05$, and was used to determine if there were any effects of gender on approach coping styles. The IV was gender and the DV's were logical analysis, positive appraisal, seeking guidance and support and problem solving. All Levene and Box's tests were not statistically significant.

Table 47

Gender and Approach Coping

	<i>n</i>	<i>M</i>	<i>SD</i>
Logical Analysis			
Male	72	49.13	10.70
Female	83	52.13	8.98
Positive Appraisal			
Male	72	51.76	8.52
Female	83	54.05	8.54

	<i>n</i>	<i>M</i>	<i>SD</i>
Seeking Guidance and Support			
Male	72	47.78	8.51
Female	83	52.61	7.71
Problem Solving			
Male	72	52.00	10.99
Female	83	53.54	9.77

Significant effects of gender on approach coping were found at the macro level: Wilk's $\Lambda = .913$ [$F(4, 150) = 3.556$], $p = .008$, partial $\eta^2 = .087$, a medium effect size with an intercept of $p = < .001$. ANOVAs determined a statistically significant effect of gender for the approach coping style of seeking guidance and support: $F(1, 153) = 13.780$, $p = < .001$, partial $\eta^2 = .083$ with a medium effect size and intercept of $p = > .001$. See Tables 47 and 48 for further details.

A MANCOVA, with α of $p = 0.05$, was used to determine any gender differences in approach coping style scores, after controlling for the effects of SES. Levene and Box's tests were not statistically significant. The MANCOVA determined statistically significant effect of SES on approach coping scores, Wilk's $\Lambda = .913$ [$F(4, 149) = 3.528$], $p = .009$, partial $\eta^2 = .087$, a medium effect size with an intercept of $p = < .001$. Statistically significant effects were also determined Table 48

Gender Analyses of Variance for Approach Coping Style of Seeking Guidance and Support

	Gender				
	SS	df	MS	F	p
Between	901.932	1	901.932	13.780	< .001*
Within	10014.107	153	65.452		
Total	10916.039	154			

for gender with SES controlled on approach coping scores:

Wilk's $\Lambda = .920$ [$F(4, 149) = 3.249$], $p = .014$], partial $\eta^2 = .080$, a medium effect size with an intercept of $p = < .001$.

Mid-level ANCOVAs determined statistically significant effects of SES on logical analysis, $F(1, 152) = 13.289$, $p = < .001$, partial $\eta^2 = .080$ with a medium effect size and intercept of $p = > .001$. Statistically significant effects of gender with SES controlled were found for seeking guidance and support, $F(1, 152) = 12.698$, $p = < .001$, partial $\eta^2 = .077$ with a medium effect size and intercept of $p = > .001$.

A one-way MANOVA with an α of $p = 0.05$, and was used to determine if there were any effects of gender on avoidance coping styles. The IV was gender and the DV's were cognitive avoidance, acceptance or resignation, seeking alternative rewards and emotional discharge. All Levene and Box's tests were not statistically significant. See Table 49 for descriptive details.

Table 49

Gender and Avoidance Coping

	<i>n</i>	<i>M</i>	<i>SD</i>
Cognitive Avoidance			
Male	72	52.82	9.28
Female	82	56.16	9.88
Acceptance or Resignation			
Male	72	51.51	9.92
Female	82	54.06	8.87
Seeking Alternative Rewards			
Male	72	53.57	8.39
Female	82	53.99	9.04
Emotional Discharge			
Male	72	54.94	10.38
Female	83	59.17	10.15

No statistically significant effects of gender on avoidance coping styles were found at the macro level: Wilk's $\Lambda = .950$ [$F(4, 149) = 1.978$], $p = .101$, partial $\eta^2 = .050$, with an intercept of $p = < .001$. ANOVAs determined a statistically significant effect of gender for two avoidance coping styles: Cognitive avoidance, $F(1, 152) = 4.634$, $p = .033$, partial $\eta^2 = .03$ with a small effect size and intercept of $p = > .001$ and emotional discharge $F(1, 152) = 6.902$, $p = .009$, partial $\eta^2 = .043$ with a small effect size and intercept of $p = > .001$.

A MANCOVA, with α of $p = 0.05$, was used to determine any gender differences in avoidance coping style scores, after controlling for the effects of SES. Levene's Test was significant for seeking alternative rewards at $p = .027$. Box's test was not statistically significant. The MANCOVA determined a statistically significant effect of SES on avoidance coping scores:

Wilk's $\Lambda = .938$ [$F(4, 148) = 2.444$], $p = .049$], partial $\eta^2 = .062$, a medium effect size with an intercept of $p = < .001$. No statistically significant effects were determined for gender on avoidance coping scores with SES controlled:

Wilk's $\Lambda = .938$ [$F(4, 148) = 2.425$], $p = .051$], partial $\eta^2 = .062$, a medium effect size with an intercept of $p = < .001$.

Perinatal Birth Group with Gender

A two-way MANOVA with an α of $p = 0.05$, and was used to determine if there were any effects of perinatal birth group with gender on approach coping styles. The IV was gender and the DV's were approach coping styles. Levene's test was significant at $p = .021$ for logical analysis. Box's test was not statistically significant.

Perinatal birth term with gender was not statistically significant at the macro level for effects on approach coping styles: Wilk's $\Lambda = .915$ [$F(20, 469) = .633$], $p = .888$], partial $\eta^2 = .022$, with an intercept of $p = < .001$. ANOVAS were not statistically significant for approach coping styles. See Table 50 for descriptive details. Tukey's Post-hoc multiple comparisons was statistically significant for MPT and NPT, $p = .015$

Perinatal Birth Group with Gender Approach Coping Means and Standard Deviations

	<i>n</i>	<i>M</i>	<i>SD</i>
Logical Analysis			
Full Term			
Male	17	48.76	13.07
Female	19	55.95	6.05
Healthy Preterm			
Male	10	47.10	12.21
Female	14	49.36	11.55
Medical Preterm			
Male	25	51.48	9.94
Female	21	54.52	8.47
Neurological Preterm			
Male	16	45.94	9.03
Female	9	47.22	9.58
Small for Gestational Age			
Male	4	53.75	2.50
Female	20	50.15	8.11
Positive Appraisal			
Full Term			
Male	17	49.29	10.55

	<i>n</i>	<i>M</i>	<i>SD</i>
Female	19	55.16	8.42
Healthy Preterm			
Male	10	52.60	10.22
Female	14	55.71	6.76
Medical Preterm			
Male	25	54.16	6.76
Female	21	55.43	9.13
Neurological Preterm			
Male	16	49.31	7.23
Female	9	52.00	8.87
Small for Gestational Age			
Male	4	55.00	6.98
Female	20	51.30	8.95
Seeking Guidance and Support			
Full Term			
Male	17	50.53	7.94
Female	19	53.68	8.20
Healthy Preterm			
Male	10	45.30	10.07
Female	14	52.14	8.01

	<i>n</i>	<i>M</i>	<i>SD</i>
Medical Preterm			
Male	25	48.36	8.22
Female	21	54.48	7.37
Neurological Preterm			
Male	16	44.63	8.58
Female	9	53.67	3.87
Small for Gestational Age			
Male	4	51.25	5.38
Female	20	49.50	8.35
Problem Solving			
Full Term			
Male	17	49.88	11.21
Female	19	53.26	9.67
Healthy Preterm			
Male	10	50.80	10.80
Female	14	53.43	10.59
Medical Preterm			
Male	25	56.16	8.13
Female	21	57.10	8.26
Neurological Preterm			
Male	16	47.81	10.69

	<i>n</i>	<i>M</i>	<i>SD</i>
Female	9	50.44	11.93
Small for Gestational Age			
Male	4	54.75	10.56
Female	20	51.55	9.59

A MANCOVA, with α of $p = 0.05$, was used to determine any perinatal birth group with gender differences in approach coping style scores, after controlling for the effects of SES. Levene and Box's tests were not statistically significant. The MANCOVA determined a statistically significant effect of SES ($p = .003$) and gender ($p = .050$) on approach coping scores as previously noted. No statistically significant effects were determined for perinatal birth group with gender on approach coping scores with SES controlled: Wilk's $\Lambda = .899$ [$F(20, 465) = .761$], $p = .761$, partial $\eta^2 = .026$, with an intercept of $p = < .001$.

A MANCOVA, with α of $p = 0.05$, was used to determine any perinatal birth group with gender differences in avoidance coping style scores, after controlling for the effects of SES. Levene's tests was statistically significant at $p = .019$. Box's test was not statistically significant. No statistically significant effects were determined for perinatal birth group with gender on avoidance coping scores with SES controlled: Wilk's $\Lambda = .914$ [$F(16, 428) = .797$], $p = .689$, partial $\eta^2 = .022$, with an intercept of $p = < .001$. See Table 51.

Table 51

Perinatal Birth Group with Gender Avoidance Coping Means and Standard Deviations

	<i>n</i>	<i>M</i>	<i>SD</i>
Cognitive Avoidance			
Full Term			
Male	17	51.24	10.32
Female	19	57.05	11.66
Healthy Preterm			
Male	10	52.40	7.47
Female	13	55.43	9.20
Medical Preterm			
Male	25	53.48	9.88
Female	21	54.43	9.20
Neurological Preterm			
Male	16	51.94	9.20
Female	9	54.22	9.62
Small for Gestational Age			
Male	4	60.00	2.31
Female	20	57.10	10.38

	<i>n</i>	<i>M</i>	<i>SD</i>
Acceptance or Resignation			
Full Term			
Male	17	52.94	8.70
Female	19	55.63	11.53
Healthy Preterm			
Male	10	45.70	11.73
Female	13	52.85	7.27
Medical Preterm			
Male	25	51.48	8.18
Female	21	52.85	7.27
Neurological Preterm			
Male	16	52.94	12.23
Female	9	52.56	12.23
Small for Gestational Age			
Male	4	54.50	9.00
Female	20	55.15	8.38

	<i>n</i>	<i>M</i>	<i>SD</i>
Seeking Alternative Rewards			
Full Term			
Male	17	51.71	7.66
Female	19	52.95	10.50
Healthy Preterm			
Male	10	48.10	7.26
Female	13	54.23	9.16
Medical Preterm			
Male	25	54.08	8.33
Female	21	52.19	9.06
Neurological Preterm			
Male	16	57.63	8.67
Female	9	54.67	11.16
Small for Gestational Age			
Male	4	55.75	6.85
Female	20	56.40	9.83

	<i>n</i>	<i>M</i>	<i>SD</i>
Emotional Discharge			
Full Term			
Male	17	55.76	10.18
Female	19	60.68	10.42
Healthy Preterm			
Male	10	51.60	9.68
Female	13	58.15	6.07
Medical Preterm			
Male	25	57.32	11.70
Female	21	58.67	9.97
Neurological Preterm			
Male	16	51.75	9.26
Female	9	61.44	12.03
Small for Gestational Age			
Male	4	57.75	6.19
Female	20	57.90	9.59

Summary

Significant differences were found between the prematurely born for approach coping style scores. The MPT ($M = 53$) and NPT ($M = 46$) differed in logical analysis scores.

Emotional Intelligence

A one-way ANCOVA, with α of $p = 0.05$, was used to determine any perinatal birth group difference in EQi scores, after controlling for the effects of SES. Levene's test was not statistically significant. The ANCOVA determined a statistically significant effect of SES on EQi: ($F(1, 151) = 9.749, p = .002$, partial $\eta^2 = .061$, indicating a medium effect with an intercept of $p = .001$. There were no statistically significant effects, as determined by ANCOVA, on perinatal birth group EQi scores after controlling for SES: $F(4, 151) = .266, p = .900$, partial $\eta^2 = .007$, with an intercept of $p = .001$. See Tables 52, 53, 54 and 55 for further details.

A one-way ANCOVA, with α of $p = 0.05$, was used to determine any gender difference in EQi scores, after controlling for the effects of SES. Levene's test was not statistically significant. The ANCOVA determined a statistically significant relationship of SES on EQi: $F(1, 154) = 9.726, p = .002$, partial $\eta^2 = .059$, indicating a medium effect with an intercept of $p = .001$. There were no statistically significant effects, as determined by ANCOVA, of gender on EQi scores after controlling for SES: $F(4, 154) = .105, p = .747$, partial $\eta^2 = .001$, with an intercept of $p = < .001$. See Tables 56, 57, 58 and 59 for further details.

Descriptive Statistics: Perinatal Birth Group for Emotional Intelligence

Emotional Intelligence EQi Scores	Perinatal Birth Group		
	<i>n</i>	<i>M</i>	<i>SD</i>
Full Term	38	98.37	17.23
Healthy Preterm	24	96.42	15.43
Medical Preterm	47	99.17	15.00
Neurological Preterm	25	101.08	14.77
Small for Gestational Age	24	98.46	15.96

Table 53

Perinatal Birth Group Analyses of Variance for Emotional Intelligence

Emotional Intelligence EQi Scores	Perinatal Birth Group				
	<i>SS</i>	<i>df</i>	<i>MS</i>	<i>F</i>	<i>p</i>
Between	282.261	4	70.565	.285	.887
Within	37897.112	153	247.694		
Total	38179.373	157			

Table 54

Descriptive statistics: Perinatal Birth Group and Emotional Intelligence with Covariate of Socioeconomic Status

Perinatal Birth Group with Covariate SES			
Emotional Intelligence			
EQi Scores	<i>n</i>	<i>M</i>	<i>SD</i>
Full Term	37	98.22	17.44
Healthy Preterm	24	96.42	15.43
Medical Preterm	47	99.17	15.00
Neurological Preterm	25	101.08	14.77
Small for Gestational Age	24	98.46	15.96

Table 55

ANCOVA for Perinatal Birth Group and Emotional Intelligence with Covariate of Socioeconomic Status

Perinatal Birth Group with Covariate SES					
EQI Scores	SS	df	MS	F	p
Corrected Model	2583.45	5	516.69	2.194	.058
Intercept	119321.60	1	119321.60	506.563	< .001
Socioeconomic Status	2296.32	1	2296.32	9.749	.002*
Perinatal Birth Group	250.27	4	62.57	.266	.900
Error	35568.22	151	235.55		
Total	1568209.00	157			
Corrected Total	38151.67	156			

Table 56

Descriptive Statistics: Gender for Emotional Intelligence

EQI Scores	n	M	SD	SE	95% CI
Male	73	97.88	15.73	1.84	[94.21, 101.55]
Female	85	99.51	15.53	1.69	[96.16, 102.86]

Table 57

Gender Analyses of Variance for Emotional Intelligence

EQi Scores	SS	df	MS	F	p
Between	104.236	1	104.236	.427	.514
Within	38075.137	156	244.071		
Total	38179.373	157			

Table 58

Gender for Emotional Intelligence with Covariate Socioeconomic Status

EQI Scores	n	M	SD
Male	73	97.88	15.73
Female	84	99.45	15.62

Table 59

ANCOVA Gender for Emotional Intelligence with Covariate of

Socioeconomic Status

EQi Scores	SS	do	MS	F	p
Corrected Model	2357.54	2	1178.77	5.072	.007
Intercept	120393.57	1	120393.56	517.979	<. 001
Socioeconomic Status	2260.57	1	2260.57	9.725	.002*
Gender	24.36	1	24.36	.105	.747
Error	35794.13	154	232.43		
Total	1568209.00	157			
Corrected Total	38151.67	156			

Table 60

Descriptive Statistics: Perinatal Birth Group with Gender for Emotional Intelligence

EQI Scores	<i>n</i>	<i>M</i>	<i>SD</i>
Full Term			
Male	17	94.47	15.52
Female	21	99.10	18.85
Healthy Preterm			
Male	10	100.60	17.36
Female	14	93.43	13.77
Medical Preterm			
Male	26	96.92	15.07
Female	21	101.95	14.80
Neurological Preterm			
Male	16	100.63	14.81
Female	9	101.89	15.54
Small for Gestational Age			
Male	4	88.00	23.58
Female	20	100.55	13.89

A two-way ANCOVA, with α of $p = 0.05$, was used to determine any perinatal birth group with gender differences in EQi scores, after controlling for the effects of socioeconomic status. Levene's test was not statistically significant. The ANCOVA

determined a statistically significant relationship of SES on EQi scores:

($F(1, 146) = 10.614, p = .001$, partial $\eta^2 = .068$, indicating a medium effect with an intercept of $p = < .001$. There were no statistically significant effects, as determined by ANCOVA, on perinatal birth group with gender on mean EQi scores after controlling for SES: $F(4, 146) = 1.351, p = .254$, partial $\eta^2 = .036$, with an intercept of $p = < .001$. See Tables 61 and 62 for further details.

Table 61

Descriptive Statistics: Perinatal Birth Group with Gender for Emotional Intelligence with Covariate SES

EQi Scores	<i>n</i>	<i>M</i>	<i>SD</i>
Full Term			
Male	17	97.47	15.52
Female	20	98.85	19.30
Healthy Preterm			
Male	10	100.60	17.36
Female	14	93.43	13.77
Medical Preterm			
Male	26	96.92	15.07
Female	21	101.95	14.80
Neurological Preterm			
Male	16	100.63	14.81
Female	9	101.08	14.77

EQi Scores	<i>n</i>	<i>M</i>	<i>SD</i>
Small for Gestational Age			
Male	4	88.00	23.58
Female	20	100.55	13.89

Table 62

ANCOVA for Perinatal Birth Group with Gender for Emotional Intelligence with Covariate of Socioeconomic Status

EQI Scores	SS	<i>df</i>	<i>MS</i>	<i>F</i>	<i>p</i>
Corrected Model	3921.09	10	392.11	1.672	.092
Intercept	107283.00	1	107283.00	457.583	< .001
Socioeconomic Status	2488.43	1	2488.43	10.614	.001*
Perinatal Birth Group	510.03	4	127.51	.554	.704
Gender	84.15	1	84.15	.359	.550
Perinatal Birth Group by Gender	1266.89	4	316.72	1.351	.254
Error	34230.57	146	234.46		
Total	1568209.00	157			
Corrected Total	38151.67	156			

Emotional Health

A one-way ANCOVA, with α of $p = 0.05$ was used to determine any perinatal birth group differences in ASR scores, after controlling for the effects of SES.

Levene's test was not statistically significant. The ANCOVA determined statistically significant effects of SES on ASR, $F(1, 150) = 4.380, p = .038$, . partial $\eta^2 = .028$, indicating a small effect with an intercept of $p = < .001$. There were no statistically significant effects, as determined by ANCOVA, on perinatal birth group mean ASR scores after controlling for SES: $F(4, 150) = .801, p = .526$, partial $\eta^2 = .0321$, with an intercept of $p = < .001$. See Tables 63, 64, 65 and 66 for further details.

Table 63

Descriptive Statistics: Perinatal Birth Group for Emotional Health

ASR-R Scores	Perinatal Birth Group				
	<i>n</i>	<i>M</i>	<i>SD</i>	<i>SE</i>	95% [CI]
Full Term	40	50.40	10.88	1.720	[46.92, 53.88]
Healthy Preterm	24	49.38	11.12	2.270	[44.68, 54.07]
Medical Preterm	49	47.10	9.85	1.406	[44.27, 49.93]
Neurological Preterm	25	46.48	9.76	1.953	[42.45, 50.51]
Small for Gestational Age	25	48.64	11.17	2.334	[44.03, 53.25]

Table 64

Perinatal Birth Group Analyses of Variance for Emotional Health

ASR Scores	SS	df	MS	F	p
Between	358.94	4	89.73	.815	.517
Within	17393.72	158	110.09		
Total	17752.65	162			

Table 65

Perinatal Birth Group for Emotional Health with Covariate of SES

ASR Score	n	M	SD
Full Term	36	50.61	11.43
Healthy Preterm	24	49.38	11.12
Medical Preterm	47	46.98	9.86
Neurological Preterm	24	47.13	9.42
Small for Gestational Age	25	48.64	11.17

Table 66

ANCOVA for Perinatal Birth Group for Emotional Health with Covariate of SES

Perinatal Birth Group for Emotional Health with Covariate SES					
ASR Score	SS	df	MS	F	p
Corrected Model	813.36	5	162.67	1.484	.198
Intercept	42962.48	1	42962.48	392.005	< .001
Socioeconomic Status	480.01	1	480.01	4.380	.038*
Perinatal Birth Group	351.17	4	87.79	.801	
Error	26439.54	150	109.60		
Total	383816.00	156			
Corrected Total	17252.90	155			

Gender

An ANCOVA was used to determine any gender differences in ASR scores, after controlling for the effects of SES. The ANCOVA determined no effects on gender and ASR scores when SES was controlled: $F(1, 153) = .004$, $p = .950$, partial $\eta^2 = < .001$, with an intercept of $p = < .001$. SES was found to have an effect on ASR, $F(1, 153) = 4.167$, $p = .043$, partial $\eta^2 = .043$ a small effect with an intercept of $p = < .001$. See Table 67 for descriptive statistics.

Table 67

Descriptive Statistics: Gender and ASR

	<i>n</i>	<i>M</i>	<i>SD</i>	<i>SE</i>	Min-Max	95% CI
Male	79	48.48	10.57	1.19	26-82	[46.11, 50.85]
Females	84	48.30	10.44	1.14	29-74	[56.03, 50.56]

Perinatal Birth Group with Gender

An ANCOVA was used to determine any differences between perinatal birth groups with gender and ASR scores, controlling for the effects of SES. No statistically significant results were found between perinatal birth groups with gender on ARS scores when SES was controlled: $F(4, 145) = .987, p = .416$, with an intercept of $p = < .001$. There was no effect on perinatal birth group or gender when SES was controlled. See Table 68.

Table 68

Descriptive Statistics: Perinatal Birth Group with Gender and ASR

	<i>n</i>	<i>M</i>	<i>SD</i>
Full Term			
Male	18	51.06	11.75
Female	22	49.86	10.36

	<i>n</i>	<i>M</i>	<i>SD</i>
Healthy Preterm			
Male	10	45.90	7.22
Female	14	51.86	12.92
Medical Preterm			
Male	28	48.54	9.96
Female	21	45.19	9.59
Neurological Preterm			
Male	17	46.41	10.93
Female	8	46.63	7.33
Small for Gestational Age			
Male	19	50.67	14.28
Female	25	48.00	10.39

An alternative analysis to test the effects of both daily stress and SES on the social stress response measures in cortisol during the TSST for vulnerable perinatal birth groups (born with medical diagnoses) was conducted. Theoretically, prematurely born age 23 year olds with more medical diagnoses and invasive interventions would be at higher risk for resetting or dysfunction of the HPA cortisol response. Combining DHS and SES allows for both daily and long term stress risk effects.

Hypothesis One: Subset Analysis

A subset analysis was conducted to examine stress (DHS-R) and SES scores between the prematurely born at high risk (medical diagnoses) and full term age 23 year olds. The perinatal birth groups of FT, NPT and MPT were selected to met a new IV criteria of: 1. FT with high stress and low SES. 2. FT with low stress and high SES. 3. Preterm (MPT and NPT) with high stress and low SES. 4. Preterm (MPT and NPT) with low stress and high SES. Stress scores were defined as low = < 25 and high = > 30. SES scores were defined as low within the 8 – 29 range and as high in the 40 – 66 range based on Hollingshead five categories of SES (Hollingshead, 1975). The variables' low range SES scores were derived from the lower (8 – 19) and lower- middle (20 -29) Hollingshead category scores. The middle SES category (30 -39) scores were excluded. The variables' high SES range of scores combined the upper-middle (40 - 54) and upper (55 - 66) Hollingshead category scores. The DVs were slope and AUCg cortisol values. See Table 69

Table 69

Perinatal Risk Group Frequencies

	<i>n</i>	%
FT High Stress and Low SES	6	12.8%
FT Low Stress and High SES	11	23.4%
PT High Stress and Low SES	6	12.8%
PT Low Stress and High SES	24	51.1%

A one – way MANOVA, with an α of $p = 0.05$, was used to determine any differences between Perinatal Risk groups across the cortisol values of slope and AUCg. The IV was the four perinatal risk groups and the DV's were slope and AUCg cortisol values. Box's and Levene's tests were not statistically significant. Tukey Post hoc homogeneous subsets were not statistically significant.

At the macro level statistically significant interactions, with exact statistics, were determined for perinatal risk groups across the cortisol values of slope and AUCg. Wilk's $\Lambda = .620$, [$F(6, 76) = 3.420, p = .005$], partial , $\eta^2 = .213$ with a small to medium effect size. The intercepts were $p = .003$ for slope and $p = < .001$ for AUCg.

ANOVAs were used to determine any differences between perinatal birth risk groups and the cortisol values of delta and peak (Table 70). ANOVA's determined statistically significant differences of perinatal risk groups across slope and AUCg cortisol values: with a large

Table 70

Descriptive Statistics: Perinatal Risk Group, Slope and AUCg

	<i>M</i>	<i>SD</i>
Slope		
FT High DHS and Low SES	-.0025	.0096
FT Low DHS and High SES	.0106	.0092
PT High DHS and Low SES	.0032	.0066
PT Low DHS and High SES	.0081	.0081

	<i>M</i>	<i>SD</i>
AUCg		
FT High DHS and Low SES	3.2678	.5745
FT Low DHS and High SES	3.5003	.6907
PT High DHS and Low SES	2.5192	.3655
PT Low DHS and High SES	3.2395	.4929

effect and intercept of $p = .003$. AUCg, , $F(3, 39) = 4.257, p = .032$], partial $\eta^2 = .247$, with a large effect and intercept of $p = < .001$. See Table 71 for further details.

Between-subjects comparisons were confirmed by Tukey's Post-hoc statistically significant results (Table 72). FT high DHS and Low SES were statistically significant from FT Low DHS and High SES ($p = .037$) on cortisol slope. Also PT High DHS and Low SES differed from PT Low DHS and High SES ($p = .006$) for Table 71

Analysis of Variance for Perinatal Risk Group, Slope and AUCg

	<i>SS</i>	<i>df</i>	<i>MS</i>	<i>F</i>	<i>p</i>
Intercept					
Slope	.001	1	.001	10.326	.003
AUCg	301.046	1	301.046	1053.049	< .001

	<i>SS</i>	<i>df</i>	<i>MS</i>	<i>F</i>	<i>p</i>
Perinatal Risk Group					
Slope	.001	3	.001	3.244	.032*
AUCg	3.651	3	1.217	4.257	.011*
Error					
Slope	.003	39	6.926		
AUCg	11.149	39	.286		
Total					
Slope	.005	43			
AUCg	454.259	43			

Low DHS and High SES and FT High DHS and Low SES ($p = .014$), were determined for slope values. FT Low DHS and High SES was statistically significant between PT High DHS and Low SES ($p = .007$) for AUCg values. Low DHS and High SES and FT High DHS and Low SES ($p = .014$), were determined for slope values. FT Low DHS and High SES was statistically significant between PT High DHS and Low SES ($p = .007$) for AUCg values.

Table 72

Perinatal Risk Group Tukey Multiple Comparisons for Slope and AUCg

	Mean	SE	<i>p</i>	95% CI
Difference				
Slope				
FT High DHS and Low SES				
FT Low DHS and High SES	-.013	.005	.037*	[-.025, -.001]
PT Low DHS and High SES				
FT High Stress and Low SES	.011	.004	.014*	[.002, .019]
AUCg				
FT Low DHS and High SES				
PT High DHS and Low SES	.981	.282	.007*	[.411, 1.551]
PT Low DHS and High SES				
PT High DHs and Low SES	.720	.245	.006*	[.255, 1.216]

Summary

Hypothesis one was partially supported. There were no statistically significant findings for the analyses of perinatal group on age 23 year outcomes of stress, emotional intelligence and emotional health (Appendix--). Statistically significant perinatal birth group differences were found for the approach coping style of logical analysis where MPT and NPT ($p = .013$) differed with MPT scores higher ($M = 53$) than NPTscores ($M = 46$). After controlling for SES, perinatal birth group was no

longer significant for logical analysis approach coping. Statistically significant gender differences were found for approach coping styles of seeking guidance and support ($p = < .009$) and when controlling for SES ($p = < .001$). No statistically significant differences were found between perinatal birth group with gender for approach coping style.

For avoidance coping styles, no statistically significant results were determined. However, for the avoidance coping styles of cognitive avoidance ($p = .033$) and emotional discharge ($p = .009$) gender effects were found for females who had higher scores than males. No statistically significant results were determined for avoidance coping styles in the perinatal birth group with gender analysis. There was an SES effect on the approach coping style of logical analysis. A SES effect for avoidance coping styles for gender was also found.

A subset analysis compared premature (Medical) risk groups and the FT group who reported high and low stress at age 23 with high and low SES. Statistically significant differences between FTL, FTH, PTL and PTH birth groups in cortisol slope and AUCg concentration values were found. Specifically, DHS-R and SES between the high risk MPT and NPT was statistically significant, $p = .005$. Additionally the AUCg total concentration values between the PTL DHS and high SES group differed significantly from the PTH DHS and low SES group, $p = .006$.

Hypothesis Two

Adults at age 23 who were born prematurely will have a prolonged stress recovery period for the TSST. Hypothesis 2 will be significant if the AUCg (total level) mean is higher during the stress recovery period (slope) for TSST in the prematurely.

The sample Hobel Total (HT) risk scores and perinatal birth groups were categorized into three groups of : Full term (FT), preterm with HT low risk (low neonatal acuity; PHL) and preterm with HT high risk (high neonatal acuity; PHH). PHL and PHH risk scores were determined by the frequency distribution of the preterm Hobel Total scores in the $\leq 25^{\text{th}}$ and $\geq 25^{\text{th}}$ percentiles of the combined preterm born infants. Scores of ≤ 115 defined PHL and scores of ≥ 188 defined PHH. See Table 73.

Table 73

Perinatal Birth Groups and Full Term, Premature Hobel Low and Premature Hobel High Risk Groups

					Sample	Hobel Group %tiles		
Risk	<i>n</i>	<i>M</i>	<i>SD</i>	Min, Max	Interquartile	Low	Moderate	High
					Range	25%	50%	25%
Sample Perinatal Birth Groups by Hobel Total Risk Score								
FT	41	13	12.39	0-52	23	≤1	2-24	25-52
HPT	24	112.90	34.17	51-187	52	≤82	83-133	134-187
MPT	49	143.53	32.75	81-202	46	≤126	127-172	173-202
NPT	26	167.10	29.35	86-220	40	≤148	149-188	189-220
SGA	26	137.71	38.28	34-201	42	≤118	118-159	160-201

Risk	<i>n</i>	<i>M</i>	<i>SD</i>	Min, Max	Sample	Group %tiles		
					Interquartile	Low	Moderate	High
					Range	25%	50%	25%
<hr/>								
Perinatal Birth Groups of Full Term and Prematurely Born by Hobel Total Risk Score								
FT	41	13	12.39	0-52	23	≤1	2-24	25-52
PT	125	141.34	37.43	34-220	56	≤115	116-187	188-220
Full Term, Premature Hobel Low and Premature Hobel High Risk Groups								
FT	41	13	12.39	0-52	23	≤1	2-24	25-52
PT LHT	32	91.27	19.49	34-115	29	≤80	81-110	111-115
PT HHT	12	199.21	8.99	189-220	10	≤195	196-216	217-220

Pearson Correlations, two-tailed, were run to determine any multicollinearity in the IV of Perinatal Hobel score risk groups (FT, PHL and PHH) and DVs of slope and AUCg (Table 74). No significant multicollinearity was found.

Table 74

Correlations for Perinatal Hobel Total Risk Group, Slope and AUCg

Variables	(1)	(2)	(3)
(1) Slope	-		
(2) AUCg	.299*	-	
(3) Perinatal Hobel	-.070	-.082	-
Total Risk Group			

* $p = 0.05$ (2-tailed)

A one-way MANOVA, with an α of $p = 0.05$, and was used to determine if there were any effects of perinatal Hobel risk groups on slope and AUCg cortisol values. The IV was the Hobel groups. Box's test was not significant. Levene's test was statistically significant for slope, $p = .04$ and not statistically significant for AUCg. Tukey's Post-hoc homogeneous subsets were not statistically significant.

Table 75

Perinatal Hobel Total Risk Birth Group, Slope and AUCg Values of Means, Standard Deviation and CI

Variable	<i>n</i>	<i>M</i>	<i>SD</i>	<i>SE</i>	95% CI
Slope					
FT	33	.008	.010	.002	[.005, .011]
PT LHR	26	.005	.007	.002	[.001, .009]
PT HHT	11	.007	.008	.003	[.002, .013]
AUCg					
FT	33	3.17	.584	.096	[2.983, 3.365]
PT LHR	26	3.13	.554	.108	[2.913, 3.344]
PT HHT	11	3.14	.414	.166	[2.712, 3.375]

No statistically significant interactions of perinatal Hobel risk groups were determined by MANOVA across the cortisol values of slope and AUCg.

Wilk's $\Lambda = .969[F(4, 132) = 529, p = .715]$, partial $\eta^2 = .016$. Intercept $p = < .001$.

One-way ANOVAs determined no statistically significant effects between the Hobel risk groups of FT, PHL and PHH s in slope and AUCg cortisol values:

Slope, $F(2, 67) = .815, p = .447$ and AUCg, $F(2, 67) = .235, p = .791$. Additional details can be found in Table 75.

A MANCOVA, with an α of $p = 0.05$, was used to determine any effect of perinatal Hobel risk groups with SES controlled, across slope and AUCg cortisol values. Box and Levene's tests were not statistically significant.

No statistically significant differences were found in the perinatal Hobel risk groups, after controlling for the effects of SES, on slope and AUCg cortisol value.

SES, Wilk's $\Lambda = .973[F(2, 62) = .870, p = .424]$, partial $\eta^2 = .027$ with an intercept of $< .001$. Perinatal Hobel risk group, Wilk's $\Lambda = .973[F(4, 124) = .435, p = .783]$, partial $\eta^2 = .014$. Intercepts for both were $< .001$.

No statistically significant results were found in the tests of between subjects effects for perinatal Hobel risk group controlling for SES on slope or AUCg cortisol values. SES: Slope, $F(1,63) = .943, p = .335$, partial $\eta^2 = .015$ and AUCg, $F(1,63) = 1.289, p = .261$, partial $\eta^2 = .020$. Perinatal Hobel risk group: Slope $F(2,63) = .346, p = .709$, partial $\eta^2 = .011$ and AUCg, $F(2,63) = 524, p = .595$, partial $\eta^2 = .016$.

Summary

Hypothesis two was not supported. MANOVAs determined no statistically significant differences between perinatal Hobel risk groups across slope and AUCg cortisol values. MANCOVAs determined no statistically significant differences in perinatal Hobel risk groups when SES was controlled, across slope and AUCg

cortisol values.

Hypothesis Three

The stress recovery period for adults at age 23 years with emotional health problems will be prolonged compared to adults without emotional health problems (as determined by ASR t-scores) when prematurity is controlled.

Pearson Correlations determined no r values $\geq .80$ in the variables of emotional health (ASR) and the cortisol values of slope and AUCg. See Table 76.

Table 76

Correlation Matrix for Emotional Health, Slope and AUCg

Variables	(1)	(2)	(3)
(1) Emotional Health (ASR)	-		
(2) Slope	-.278**	-	
(3) AUCg	-.066	.425**	-

** $p = 0.01$ (2-tailed)

A one – way MANOVA, with an α of $p = 0.05$, was used to determine any differences between two ASR clinical category scores and the cortisol values of slope and AUCg. The IV was ASR (no clinical problems or yes clinical problems) and the DV's were slope and AUCg. Box's and Levene's tests were not statistically significant.

At the macro level statistically significant interactions, with exact statistics, were determined for ASR across the cortisol values of slope and AUCg.

Wilk's $\Lambda = .921$, [$F(2, 139) = 5.929$, $p = .003$], partial $\eta^2 = .079$ with a medium effect size. Intercept $p = < .001$. ANOVA's determined the statistically significant

Table 77

Sample Descriptive Statistics for Effects of ASR on TSST Cortisol Values of Slope and AUCg

Variable	<i>n</i>	<i>M</i>	<i>SD</i>	<i>SE</i>	95% CI
Slope					
No Clinical Problems	119	.009	.009	.002	.008, .011
Clinical Problems	23	.002	.008	.002	-.002, .006
AUCg					
No Clinical Problems	119	3.188	.508	.120	3.093, 3.284
Clinical Problems	23	3.093	.627	.120	2.875, 3.311

Table 78

Analysis of Variance for Effects of ASR on TSST Cortisol Values of Slope and AUCg

Variables	<i>SS</i>	<i>df</i>	<i>MS</i>	<i>F</i>	<i>p</i>	Partial η^2
Slope	.001	1	.001	11.436	.001	.076
AUCg	.174	140	.174	.612	.432	.004

differences of ASR across slope and AUCg cortisol values were between slope:

$F(1, 140) = 11.436, p = .001$, partial $\eta^2 = .076$, with a medium effect. See Tables 77 and 78 for further details.

determined for ASR clinical categories across slope and perinatal birth groups:

Wilk's $\Lambda = .922$, [$F(2, 145) = 6.168, p = .003$], partial $\eta^2 = .078$ with a medium effect size. Intercept $p = < .001$. ANOVA determined perinatal birth groups and ASR categories were not significant, $F(1, 146) = .222, p = .638$ with an intercept of $p = < .001$. Statistically significant results were determined by ANOVA for ASR clinical categories and slope: $F(1, 146) = 12.237, p = .001$, partial $\eta^2 = .077$ with a medium effect and an intercept of $p = < .001$. See Table 79 for further details.

Table 79

Perinatal Birth Group and ASR Clinical Categories Effects on Slope

	<i>n</i>	<i>M</i>	<i>SD</i>
Perinatal Group			
Non-Clinical	125	2.93	1.345
Clinical Problems	23	2.78	1.445
Slope			
Non-Clinical	123	.0094	.0095
Clinical Problems	23	.0020	.0078

Table 80

Analysis of Variance of ASR Clinical Categories for Perinatal Group and Slope

	SS	df	MS	F	p
Intercept					
Perinatal Birth Group	633.492	1	633.492	342.219	< .001
Slope	.003	1	.003	29.417	< .001
ASR Clinical Categories					
Perinatal Birth Group	.411	1	.411	.222	.638
Slope	.001	1	.001	12.237	.001*
Error					
Perinatal Birth Group	270.265	146	1.851		
Slope	.012	146	8.541		

In summary hypothesis three is fully supported. Statistically significant results were determined between slope values for AUCg clinical categories whether full term or prematurely born.

Summary

In Chapter 5, the significant findings with research considerations related to the three hypotheses will be discussed. Limitations of the study will be reviewed along with research and clinical implications of the findings.

CHAPTER FIVE

In this chapter, the study results are reviewed for each hypothesis and considered in relation to the concepts of DOHaD theory. Implications for theory development, nursing research, clinical practice and policy considerations are presented.

Aims and Hypotheses

Aim 1: Compare effect of prematurity on stress, coping, emotional intelligence, and emotional health. In this aim, the 23-year outcome variables were examined in relation to the five perinatal birth groups. The outcomes were DHS-R summed scores ranging from 0 - 159 for severity of stress, the CRI-A mean scores ranging from 20 - 80 + with M 50 (*SD* 10) for avoidance coping, the Bar-On EQI total mean score of 100, (*SD* 15) for emotional intelligence effectiveness and the ASR mean score of ≥ 60 for risk to emotional health.

Age 23 is a critical time period in the lifespan due to concurrent physiological completion of brain growth and further developmental growth. It is a time when *cognitive and social abilities increase* (Arnett, 2013, Epstien, 1986). Central to this is *the ability to deal with life's stresses and challenges* (WHO, 2015) and maintain a good adjustment across the stress response (Ficchione, 2015; Luther, Cicchetti & Becker, 2000). A primary feature of stress responses and the role of the HPA Axis, is the bidirectional impact of adaptation, to both the psychosocial and biological processes (McEwen & Gianaros, 2010). McEwen & Gianaros (2010) view, the role of the brain in stress and adaptation, as central to socioeconomic, health and disease status.

Hypothesis 1. Higher self-reported stress scores, higher use of avoidance coping styles, lower emotional intelligence scores and more emotional health disorders will be

found for the adults at age 23 years born prematurely compared to the term-born adults. Hypothesis one will be significant if DHS–R total scores are higher, CRI-A avoidance t-scores are higher, EQi total scores are lower, and ASR scores are higher for the premature groups. This hypothesis was partially supported.

ANOVAs and MANOVA results showed statistical significance for differences between the prematurely and full term born groups in stress, coping, emotional intelligence and emotional health. Statistically significant differences ($p = .013$) were found for the approach coping style of problem solving between MPT ($M = 57$, the highest of average scores) and NPT ($M = 49$, the lowest of average scores). Yet, with a mean score of 50 ($SD = 10$), the approach coping style scores were within the normative range. Additionally, the statistically significant differences for gender in seeking guidance and support and cognitive avoidance were supported in previous research with healthy young adults who were not born prematurely (Holahan, Moos, Holahan, Brennan & Schutte, 2005; Man, Dugan, & Rector, 2012; Matud, 2004; Tamres, Janicki and Helgeson, 2002).

Given this is a period in life, when young adults are beginning to assume more responsibility, they are being confronted with new types of stress and learning how to cope effectively (Arnett, 2000, 2013 & 2017; Ingels, Glennie & Lauff, 2012). Lasting coping styles may not have a cumulative effect at this point in time for both the full term and prematurely born (Lovallo, 2005; Somerfield & McCrae, 2000). Previous and future daily hassles stress which accumulates over time (allostasis) may take decades to be expressed as an emotional health disorder (Shonkoff, Boyce & McEwen, 2009). Either there were less emotional or clinical problems within this sample; confounding variables, less chronically repeated daily stress or stress measurements were introduced too early to capture the

expressions of adult onset clinical problems. It is also possible that young adults under-report their stress. Saigal (2014), reported that former preterm infants perceived themselves to be doing better than their parents reported. It is also possible that there was a social desirability bias in the CRI-A responses. But, the self-perception of the young adults in this sample represents their state and is important that it is recognized.

In a subset analysis, stress and SES scores between the prematurely born at high risk (neonatal risk) and the full term age 23 year olds, were examined and found as statistically significant for differences between the full term and prematurely born groups in cortisol slope values and within the prematurely born groups in AUCg values. The full term born group overall have quicker cortisol responses to a social stressor as indicated by the differences in slope change compared to the preterm group with neonatal medical and neurological diagnoses when daily stress and socioeconomic status effects are added.

The study also found when the full term group who reported low stress and high socioeconomic status was compared to a group born prematurely with high daily stress and low SES, there were significant differences in the total cortisol concentration levels (AUCg) during the social stressor.

The frequent and prolonged stress often occurring in lower SES and coupled with lack of emotional parental support, has been correlated with future physical and emotional problems (McEwen & Seeman, 1999; Li, Zang, Tian, Liu, Yin & Xi, 2014). This combination of frequent and prolonged stress associated with lower SES and often with unsupportive and/or abusive relationships with parents, is the definition of “toxic stress” (Gardner, Qualter & Whitely, 2011; Shonkoff, Boyce & McEwen, 2009). In toxic stress the prolonged cortisol HPA Axis response alters the brains development (Thompson, 2015).

Add to this combination an already altered cortisol response to stress, such as being born prematurely and developing medical and neurological diagnoses, then risk for psychopathy, cardiovascular and metabolic problems increases (Saigal, 2014).

Chrousos & Gold, 1992, found research evidence for HPA Axis hyperactivity and hypoactivity dysfunction interacting with many bodily systems and affecting a variety of pathophysiological states. Again the identified pathophysiological states included psychiatric, endocrine and inflammatory disorders.

This study findings supports the DOHaD theory that HPA axis dysregulation occurs during the neonatal period with neonatal illness and intensive care environments and that this dysregulation continues to be activated when the living context is represented by SES and stress in this analysis (Gardner, Shonoff, Siegel, Dobbins, Earls, McGuinn, Pascoe & Wood, 2011).

The circumstances of a low SES and the related social and neighborhood issues may not be changeable, yet research has shown that parental or adult supportive and emotional relationships with their children, in low SES environments, can be protective from these adverse effects (Gardner, et al., 2011; Noble, Norman & Farah, 2004; Li, Zang, Tian, Yin & Xi 2014; Parent, Garai, Forehand, Roland, Potts, Haker, Champion, et al., 2011).

Gunner & Donzeel, 2002, found when parents provided supportive, attentive and sensitive care to children experiencing stress, fear and anger, the child's cortisol levels decreased. When parents were not attentive or responsive to a child experiencing stress, fear or anger responses, the child's cortisol levels increased (Gunner & Donzell, 2002). In the same situations with the children, when the responding person was a babysitter, the same cortisol responses were found (Gunner & Donzell, 2002).

Other findings, which are descriptive in nature, confirm what is already known about the characteristics of current young adults (Arnett, 2000, 2013 & 2017). The novel results are that the present study reveals similar results for the vulnerable prematurely born groups. Overall the sample participants as a group, had average to above average stress scores. Additionally, the full term and SGA preterm group had above average stress. Self-reported stress scores prior to this time period are unknown and future stress scores would be useful to compare stress scores overtime for the cumulative effect leading to the concept of allostatic load. What is not known, is if the average daily stress scores for all age 23 year olds in this study, are representative of sustained or passing acute response patterns at this stage of development. Therefore, what influence current stress has on, and if and when, the timing of any long-term bidirectional health effects may be expressed is unknown (Dahl, 2001; Shonkoff, Boyce & McEwen, 2009).

According to Lazarus, (1999) stress-mediating processes include a coping response which requires first the appraisal and second the effectiveness of coping. Appraisals according to Lazarus, are how the situation is cognitively evaluated and effect of the resultant emotional responses to that evaluation (Lazarus, 1999).

Approach coping styles, can also be thought of as problem-focused and addressing stress directly, has four methods or strategies, to deal with the situation (Folkman & Moskowitz, 2004): 1. Logical analysis attends to the situation, uses past experiences and thinks about actions and consequences. 2. Positive appraisal accepts the situation and thinks about it favorably. 3. Seeking guidance and support involves utilization of others for help in dealing with the situation. 4. Problem solving utilizes giving thought to actions and consequences, utilization of information and resources and taking action. Approach

coping mean scores were average for all perinatal birth groups except “somewhat above average” for the group that was seriously ill with medical problems (MPT group). This finding serves as a protective coping style for the at risk MPT born age 23 year old.

In avoidance coping styles, which can also be thought of as emotional coping to avoid stressful feelings, there are again four methods to deal with the situation (Folkman & Moskowitz, 2004): 1. Cognitive avoidance minimizes or denies the situation or consequences. 2. Acceptance or resignation views the situation as unalterable. 3. Seeking alternative rewards directs involvement elsewhere for satisfaction. 4. Emotional discharge involves openly venting feelings of anger and frustration and engaging in behaviors (often risky) to temporarily reduce tension.

Cognitive avoidance, seeking alternative rewards and emotional discharge were “somewhat above average” for all perinatal birth groups with the highest score for the SGA preterm group. Descriptively the “somewhat above average” use of avoidance coping during young adulthood found in this study agrees with the other recent findings (Arnett, 2000 & 2003).

Campbell-Sills, Cohan and Stein, 2006, assessed 132 university undergraduates using a battery of tests related to: Coping in stressful situations, resilience, personality, childhood trauma and symptomatology. Coping styles were found to have more impact on resiliency than personality. Task oriented coping was related to resiliency while emotionally oriented coping was related to less resiliency.

A number of researcher have found baseline avoidance coping styles, especially cognitive avoidance and emotional discharge, linked to the generation of more stress and the development of depressive symptoms decades later (Holahan, Moos, Holahan, Brennan &

Man, Dugan & Rector, 2012). Given this knowledge, the recent increase of young adults use of risky behavioral choices in dealing with stress and anger responses (avoidance coping by emotional discharge) could predict an increase in later life stress and depressive symptoms.

In coping research, there is no established gold standard for measurement and there are many theoretical approaches with related measures, making comparisons of results across studies difficult (Folkman & Moskosi, 2004; Skinner & Zimmer-Gembeck, 2007). Importantly, the interpretation of the effectiveness of a coping style needs to be made in the context of the situation where used and not solely according to the type of style (Lazarus, 1999; Skinner & Zimmer-Gembeck, 2007).

Roesch, Weiner & Vaughn, 2002, reviewed treatment research studies on cognitive approaches to stress and coping, which focused on psychological and physical adjustment. Positive appraisal of a situation as challenging was associated with better psychological and physical adjustment. Treatment interventions that altered negative assessments of the situation resulted in improved adjustment. When relaxation and social support was included in treatment the efficacy improved. Two approach coping styles stand out from this study: Positive appraisal and seeking guidance & support.

Blalock & Joiner, 2000, evaluated 179 men and women using the CRI-A, Negative Life Events Questionnaire and the Beck Depression and Anxiety Inventory, at three time points. Females who reported high negative life events and high cognitive avoidance coping also reported more depressive symptoms.

In the literature on emerging young adults, there is evidence of delayed developmental (demographic) milestones. That is, the current generation of young adults,

are delayed in taking full financial responsibilities, marriage and parenthood to name a few. This has been found for the current sample as well (Arnett, 2013; Moos & Holahan, 2003). It follows, then, that responding to stress with an established coping pattern is not fully developed at age 23 years. Further investigation after this age period would add to our knowledge of whether the coping styles develop, and whether there are any protective factors or risks to long-term health consequences.

The age 23 participants sample overall EQi score mean of 98.75 ($SD = 15.59$) are 1.25 points below mean, < 1 SD above and within standardized range results (Bar-On, 2002 & 2006; Di Fabio & Saklofske, 2014; De Weerd & Rossi, 2012). The EQi mean scores ranged from 62 (lower than standard scores) to 132 (higher than standard scores) or from 27% below standard to 15% above standard scores (Bar-On, 2002 & 2006; Di Fabio & Saklofske, 2014; De Weerd & Rossi, 2012). The essence of emotional intelligence as a young adult at aged 23 years and measured by the EQi is the ability to be aware of, understand, control and express emotions (Bar-On & Parker, 2000). The sample participants average standard EQi rating suggests the group is *potentially effective in emotional and social functioning* (Bar-On & Parker, 2000, p 366). The lower 27% have the *possibility* for the existence of emotional health problems while the upper 15% are more likely to function *effectively emotionally* (Bar-On & Parker, 2000, p 366).

EQi mean scores are known to increase with age and this is thought to be due to increased cognitive abilities as well as life experiences (Bar-On & Parker, 2000). The highest EQi mean scores across the lifespan are found in 40 to 50 year old respondents (Bar-On & Parker, 2000, p 366). In addition to increased cognitive abilities and life experiences, adults have been found to utilize more emotional regulation strategies and

may also react less intensely to emotions than emerging adults. (Schiebe & Carstensen, 2009).

Importantly, at age 23, no statistically significant differences existed between the prematurely and full term born for emotional intelligence which indicates both groups fairing equally as well at present. EQi scores were found to be associated with SES in this study. Statistically significant differences for SES were found for EQi when SES effects were controlled in both the gender and the perinatal birth with gender groups, ($F(1, 146) = 10.614$, $p = .001$, partial $\eta^2 = .068$, indicating a medium effect with an intercept of $p = < .001$).

Emotional health as represented by ASR clinical problem scores did not statistically differ across perinatal birth groups. Socioeconomic effects were determined to be significant for ASR scores in general, $F(1, 150) = 4.380$, $p = .038$, partial $\eta^2 = .028$, indicating a small effect with an intercept of $p = < .001$. Slightly higher mean ASR scores were found for full term, healthy preterm and small for gestational age born at 23 years old, in general even after controlling for SES. The medical preterm and small for gestational age consistently have the slightly lower mean ASR scores.

In summary, young adulthood remains a critical time period for maturity of coping, emotional intelligence, and clinical problem behaviors. This may be more crucial for the prematurely born who may have HPA dysregulation due to neonatal and perinatal stress especially when coupled with repeatedly high stress levels and lower socioeconomic status. In this sample, there was partial evidence of this between the prematurely born with high stress and low SES and the prematurely born with low stress and high SES statistically significant differences ($p = .006$). ANOVA's determined statistically significant differences of perinatal risk groups across slope and AUCg cortisol values: Slope, $F(3, 39) = 3.244$,

$p = .032]$, partial $\eta^2 = 2.00$, with a large effect and intercept of $p = .003$. AUCg with $F(3, 39) = 4.257, p = .032]$, partial $\eta^2 = .247$, with a large effect and intercept of $p = < .001$.

Aim 2: Compare the salivary cortisol response between premature and full - term born infants in the social stress paradigm of the TSST. Descriptive statistics including line graphs by groups will be examined first. Analysis of Variance (ANOVA) will be used to determine differences in salivary cortisol responses as measured by AUCg and at 6 sample times from the TSST paradigm at 15, 30, 45, 60, 75 and 90 minutes.

This study found support for the second aim and fills a gap in research on the differences between all premature and full-term born in cortisol responses to the social stress paradigm of TSST. The finding of differences in cortisol response helps in identifying vulnerable individuals of all birth groups. What is known is premature born infants have a delayed cortisol response in general (Bruehl, Wolf, & Covit, 2009; Sullivan, et al., 2008).

Lee, Fried, Thayer and Kuzawa, 2014, studied the cortisol levels of 1,403 Cuban young adults (ages 21-23) to test if diurnal (daily) cortisol levels would vary depending on preterm status. Standardized cortisol measures, protocol and methods were used. Physical examination findings as well as extensive questionnaires focused on individual, household and community variables. The researchers found statically significant results in male born preterm young adults for lower morning cortisol values that declined slower throughout the day (Lee, Fried, Thayer & Kuzawa, 2014).

Unlike other reported research, this aim addressed the notion that prematurely born infants underdeveloped neuroendocrine systems, coupled with frequent activations by extensive neonatal intensive care interventions, contributions to the dysregulated HPA Axis

responses. The design differed from other studies by using a neonatal acuity score (Hobel) as the neonatal risk level indicator, rather than birth weight or gestational age in combination or alone (Wust, Entringer, Federenko, Schlotz and Hellhammer, 2005). The Hobel Total acuity scores were composed of 35 medical factors including birth weight and gestational age (Strobino, 2006). The Hobel Total is the only birth scoring measure that includes medical problems after birth and which is highly correlated with length of hospitalization stays (Strobino, 2006). However, the Hobel Total acuity score relationship to future disease formation has not been determined (Strobino, 2006). This hypothesis supported DOHaD theory of the long-term effects of being born prematurely on biology and the impact of stress.

Hypothesis 2. Adults at age 23 who were born prematurely will have a prolonged stress recovery period for the TSST. Hypothesis 2 will be significant if the AUCg mean is higher during the stress recovery period of the TSST in the prematurely born age 23 year old adult.

The null hypothesis was accepted as proved by MANOVAs and MANCOVAs, which determined no statistically significant results for differences between Hobel Total high and low risk perinatal groups across TSST slope and AUCg cortisol values. Additionally no statistically significant results were found when SES was controlled. The Levene's test was statistically significant for slope, which indicated Type I errors, were not guaranteed.

The assumption that Hobel Total risk scores which indicate medical acuity, longer hospital stays and more intrusive intentions, as an indirect measure of alterations to HPA Axis regulation at a critical time period, underlies this aim. Buske-Kirschbaum, Krieger, Wilkes, Raugh, Weiss and Hellhammer, (2007), found no difference in cortisol patterns

between children who were prematurely or full term born in a small sample of 18 children per group aged 8-14 who participated in the child version of the TSST. When cortisol responses to the TSST were compared to diurnal cortisol responses (over the day measurements without a stress challenge) higher cortisol level differences were found in the first cortisol measurement after awakening (Cortisol awakening response or CAW) for the prematurely born. Recent research of age 54-76 year olds ($N = 466$) found TSST cortisol responses were positively associated with the CAW cortisol response (Kidd, Carvalho & Steptoe, 2014).

Dickerson and Kemeny (2004) conducted a rigorous meta-analysis of 208 laboratory studies of stress inducing tests and cortisol responses. The psychological interpretation of the stress inducing test, as an “uncontrollable social threat”, is necessary to induce a physiological response. The participants assessment of uncontrollable social threat were not paired with their cortisol responses in this study. Overall, research evidence demonstrates cortisol responses do increase in response to the TSST stress challenge and in this study did increase from baseline or delta measurements prior to testing. In addition, additional data was collected during the TSST protocol to confirm the stress challenge. Heart rate, blood pressure, salivary alpha amylase, and self-reported visual analog scale, and Adverse Symptoms Checklist (completed 30 minutes prior to the TSST, 30 minutes after the start of the TSST, and 75 minutes after the start of the TSST).

This study may lend support to previous findings of no differences in slope and AUCg between the prematurely born and full term born on acute stress cortisol responses. However, the prior studies did not examine the neonatal acuity among the prematurely born. The neonatal medical acuity is not clinically questionable yet may not be the direct casual

link to altered HPA Axis functioning or dysregulation and requires a mediating factor not captured in this analysis. Additionally, the temporal distance of 23 years from the birth Hobel Total risk score measure and the cortisol slope and AUCg values measurements has been likely impacted by many multifaceted events overtime. The analyses did incorporate SES, which has been found to be a significant factor in HPA regulation. More proximal cumulative effects such as caregiving and other lifetime social factors were not modeled here, although the data has been collected.

Winchester, Sullivan, Roberts and Granger, 2015, addressing a gap in the research, studied the relationship between prematurity, medical complication and HPA Axis function at age 23. One hundred forty-nine young adults ($n = 34$ full-term and $n = 115$ preterm born) from the longitudinal study were divided into three groups of preterm with neonatal illness. Overall sample results demonstrated marked difference between multiple cortisol levels obtained over the day in the medically preterm and the neurological preterm born when compared to other perinatal birth groups. The moderating effects of birth weight and SES on the prematurely born were examined. Significant effect for birth weight moderating the HPA Axis activity over time in the prematurely born were determined. Marked differences in cortisol again were found for the influence of SES on cortisol in the medically and preterm born 23 year olds. Within the medically and neurological preterm born young adults, cortisol values were found to vary between participants, with low cortisol responders and high cortisol responders, representing both HPA Axis dysfunctional regulation patterns. The pattern differences in the HPA Axis activity were predicted by birth weight and neonatal illness experienced at birth by the preterm born young adults.

Finally the Hobel Total risk scores relationship to future disease formation and/or HPA

Axis dysregulation has not been established and warrants further study given the scores capture weight differences, more comorbid conditions, length of hospital stay, after birth conditions, and related morbidity in normal weight infants for comparisons (Strobino, 2006).

Aim 3. Examine the relationship between the effect of emotional health and on the stress recovery period of the TSST measured in salivary cortisol. Three levels of emotional health (normal > 59, borderline 60 – 63, and clinical $\geq$ 64) as measured by the ASR were examined for the salivary cortisol samples. Descriptive statistics including graph lines by groups were used followed by ANCOVA where the effects of prematurity and gender were controlled to understand whether cortisol AUCg and slopes differ due to emotional health.

As previously defined, a 2-group (clinical problems/no clinical problems) format was used for the emotional health ASR scores. Nineteen percent of the total sample had clinical emotional health problems. Of note is the EQi score predicted a possible 27% of the sample with emotional problems. National prevalence rates in 2014 for any mental illness, excluding substance use, for ages 18 -25 year olds is 18% for the United States (NIMH, 2014), so this sample closely reflected this prevalence. Worldwide, while age of onset of mental illness often occurs during childhood and adolescence, the majority of mood disorder onsets follow “*a roughly linear increase through late middle age*” and then declines (Kessler, Amminger, Aguiar-Gaxiola, Alonso, Lee & Uston, 2007).

This study uniquely focuses on 23 year olds within the age range of 18 -25 year olds when mood disorders as emotional health clinical problem are beginning to be diagnosed. United States prevalence rates increase through ages 26-43 years and begin to decline at around age 50 (NIMH, 2014). The National Survey on Drug Use and Health, 2008, found 58.7% of

adults with a serious mental health issue were not involved in treatment (SAMHSA, 2014). The American Psychological Association in 2016 found 4% of young adults reported not seeking mental health care despite identifying a need for the care (APA, 2017).

Hypothesis 3. The stress recovery period for adults at age 23 - years with emotional health problems will be prolonged compared to adults without emotional health problems (as determined by ASR t-scores) when prematurity is controlled.

The alternative hypothesis was accepted as the MANOVA revealed statistically significant results for differences between ASR non-clinical and clinical problem scores across TSST slope values: Wilk's $\Lambda = .921$, [$F(2, 139) = 5.929$, $p = .003$], partial $\eta^2 = .079$ with a medium effect size and intercept $p = < .001$.

This study not only supports previous research examining differences in slope cortisol values of emotional health clinical problems yet adds new knowledge by the validation of self-report emotional health measures to salivary cortisol differences.

Conclusions

Most importantly, this study supports that the HPA Axis response to a social stressor is a biomarker of premature birth and of psychological problems. The prematurely born who suffered with neonatal medical illnesses have differences in cortisol slope changes and AUCg total cortisol concentration 23 years after birth. This is notable when confronted with increased stress and especially when from a lower socioeconomic background. The finding of differences between the acute stress response indicated by cortisol slope values and their relationship to the diurnal cortisol awakening response is worth exploring as biomarker measure for acute stress responses. Additionally females with higher avoidance coping use, coupled with prematurity, high stress and low socioeconomic status may be at even more risk

for emotional problems (psychological clinical problems).

Secondly, all participants with psychological problems demonstrated differences in cortisol slope changes when compared to those without psychological problems regardless of perinatal group. The self reported psychological problems were confirmed by related differences in acute stress response cortisol values.

Implications for Research

Research designs for further study of the HPA Axis role in prematurity will need to focus on time points of daily stress responses, over time as a longitudinal measure of cumulative stress effects, through the next few decades when all types of disorders develop. Although there is more research confirmation for medical disease consequences in response to HPA dysregulation, the findings for cortisol differences in all type of psychological problems warrants further research. Abnormal cortisol result findings have contributed to evidence for the HPA Axis role in psychological problems.

Vreeberg, Hoogendijk, Van Pelt, DeRijk, et al., 2009, conducted one of the largest studies to examine cortisol in depression and anxiety. A cohort study of 1588 Netherlands consisted of 3 groups: None, current or remitted psychiatric disorder. Diurnal cortisol measures were analyzed. Significant results for cortisol awakening levels were found for both current and people with remitted depression. The results were considered to indicate an increased biological vulnerability to depression.

Dexamethasone suppression tests have been used to test for HPA Axis hyperactivity. An HPA Axis that does not respond to dexamethasone is considered hyperactive (reduced sensitivity), (Pariante & Miller, 2001). In an extensive review of research literature examining HPA Axis function and many illnesses, Pariante & Miller, (2001), found

evidence of an impaired negative feedback loop system with depressive illnesses. The circulating cortisol levels were high and the HPA Axis was not responding to this feedback. HPA Axis and neuroendocrine dysregulation was interpreted as the link to depression.

Clearly, there is HPA dysregulation in those participants with psychological problems and the long term consequence of medical problems coexisting with this potentially exists at a psychobiological interface. Participants on medications for psychological problems would need to be identified to examine the effects of medication on cortisol levels. The current sample with clinical problems maybe too small and if further continual study occurs then additional participants would be added.

Further longitudinal follow up of stress levels and possibly the addition of other established stress inducing testing with cortisol samples, would identify any changes within and between current participants at a different life time point. This information could be used to address any cortisol differences in perinatal birth groups in their third decade as well as participants with new onset psychological problems.

Finally, secondary data can be used to identify the onset of childhood and psychiatric illnesses for the entire sample up until this time. Identifying the characteristics of participants who develop psychological problems by birth illness status, gender, stress levels, coping styles, socioeconomic status and coexisting medical problems may identify mediating and moderating relationships.

Clinically, the prematurely born who had medical and neurological problems during the neonatal period are at higher risk for the later effects of high stress and lower socioeconomic status as indicated by alterations in their HPA Axis function to acute stress. Knowing, cortisol levels are slower to respond and more concentrated in the prematurely born risk

group identified, preventative actions and psychological interventions related to stress would be of benefit. Prevention consisting of education as to what is known about their cortisol responses, motivational interviewing, identifying potential protective and risk factors within their environment, identifying effective self-regulation responses to stress, stress reduction methods, promoting self-discerned limited exposure to stress when applicable and identifying toxic stress would benefit them (Epel, Blackburn, Dhabbar, Adler, Morrow & Cawthon, 2004; Everly & Benson, 1989; Farb, Anderson, Mayberg, Bean, McKeon & Segal, 2010; Gardner, Qualter & Whitely, 2011; Goldin & Gross, 2010; McEwen, 2008; Siela & Wieseke, 2012; Vyas, Mitra, Rao, & Chattarji et al., 2002). Toxic stress is defined as exposure to excessive or prolonged stress inducing the physiological response, which occurs in the absence of a stable responsive relationship, that provides a protective buffer (Gardner, Qualter & Whitely, 2011). Clinical treatment intervention, in addition to the above would include mindfulness based stress reduction and cognitive behavioral programs (Garber, Clarke, Weersing, Beardslee, Brent, Gladstone, Debar, et al., 2009).

Implications for Theory Development

Many methodological criticisms of earlier epidemiological studies using DOHaD theories concerned the use of body weight and gestational age measurements, statistical effects, controlling for known confounding variables, and the need for longitudinal follow-up to identify trajectories have been and are being addressed (Sloan & Overland, 2012; Erickson, 2006; Godfrey, 2006; Sullivan, et al., 2008). This study is one of a few recent research studies reporting on the prematurely born HPA axis effects evidence at young adulthood who were followed from birth. Most studies have been cross sectional and this study is one of a few that have been prospective since birth. Measures were

confirmed and methods standardized adding to the rigor of investigations guided by DOHaD theory.

The underlying concepts of allostasis, allostatic load, HPA axis dysregulation and the effect of early life stress on young adults were both tested and supported. The application of the DOHaD theoretical approach resulted in finding cortisol differences at age 23 among the prematurely born. Prematurely born with medical and neurological illnesses exposed to more early stress showed different changes in their stress cortisol responses. The cortisol differences between 23 year olds with and without psychological clinical problems also confirmed the HPA Axis role.

The expansion of DOHaD theory to incorporate SES has taken decades of research to obtain prospective findings. Results from DOHaD theory addressing other factors than the biological induced and post diagnostic illness conditions are now being published. The theory has moved into the realm of concretely providing evidence of the reciprocity of psychological, social and environmental issues. The implication for DOHaD theory from this study demonstrates the continued need to integrate the physiological, psychological, SES, parenting and other environmental factors. Addressing postnatal and early life stress experiences effects across the lifespan will add to our theoretical knowledge of the protective and risk effects beyond this age time point.

Implications for Nursing Research

Nursing stress research has focused primarily on the Selye's and Lazarus's model consistent with elements of this study. Incorporating DOHaD theory conceptualizations of the HPA Axis role as stress inducing (through appraisals) and as a target of stress which ultimately effects aspects of health is congruent with nursing values (Shonkoff, Boyce &

McEwen, 2009; Rice, 2012).

In a unique study, a prospective longitudinal care study, conducted by Lester, Hawes, Abar, Sullivan, Miller, Bigsby, Laptook, et al., (2014), vulnerable premature infants with medical and neurological illnesses received neonatal intensive care in a single-family room. Significant progress in weight gain, decreased medical complications and less medical interventions occurred. The number of medical procedures, as well as stress and pain were mediated by parental involvement. Lester, et al., 2014, study addressed “toxic” stress effects with the prematurely born infants. Reducing overstimulation from multiple care givers and infants in one room, life sustaining equipment, and excessive activity were decreased from the environment. Response to stress were decreased in infants whose underdeveloped systems responded with spasticity. Parental involvement and attention was shown to decrease overactive stress response and physiological responses. Not only was the offending stress removed the protective parental attention was added. More studies documenting interventions blocking stress with supportive parental relationships are needed.

Given that the prematurely born infant who developed neonatal medical illness is at greater risk for the detrimental effects of stress, studies targeting prevention at the earliest age are needed. Studies targeting children with known SES adversity which include protective interventions will identify effective interventions earlier in their life’s course (Gardner, et al., 2011; Gunnar & Donzella, 2002). Prospective research throughout adolescence, young adulthood and the remainder of the life span to track the development of psychological clinical risk, problems and associated protective mechanisms will expand our knowledge (Shonkoff, Boyce & McEwen, 2009; Sullivan et al., 2008; Winchester, Sullivan, Roberts & Granger, 2016)

Implications for Clinical Practice

Nursing is in a unique position of providing caregiving across the lifespan, can incorporate supportive client specific guidance on dealing with minor and major stress, and be active in interrupting unhealthy behaviors. Early intervention in stress reduction can prevent the development of adulthood diseases as we have discussed (Shonoff, Boyce & McEwen, 2009). Opportunities to intervene abound starting with infancy and the earlier the intervention the better. The Hobel acuity score as an encompassing measure of many neonatal health issues would benefit from further testing in assessing DOHaD theory concepts as it relates to HPA function. We have seen opportunities to decrease stress in acute neonatal care and need to look at other age related aspects of our care. In addition to prematurely born infants with increased medical risk, “toxic” stress for any infant or child requires supportive intervention and further research of its nuances of developmental effectiveness.

Children from low socioeconomic circumstances show heightened stress responses, experience more conflict, are more likely to be subjected to punitive parental response and have fewer positive or rewarding experiences (Shonoff, Boyce & McEwen, 2009). The prematurely born with medical and neurological problems, exposed to toxic stress, at a biological critical time period, from a lower socioeconomic status is at a higher risk for increased adulthood disease development. Preventative interventions are preferred at an early age.

Age 23 year olds are still learning how to cope with developmental stress and have not yet fully solidified their coping styles. Age 23 is also a critical biological and developmental time period and the recommendations for childhood interventions are still applicable.

Implications for Policy

Supporting, implementing, and translating to nursing care the policy recommendations of the American Academy of Pediatrics regarding childhood adversity and toxic stress would benefit lifelong psychological and physical health (Gardner, Qualter, & Whitely, 2011). The recommendations incorporate the biological, behavioral and social sciences into an framework for promoting health and disease prevention. (Gardner, Qualter, & Whitely 2011). Multiple personal experiences, environmental and genetic dispositions are taken into consideration. (Gardner, Qualter, & Whitely 2011). The American Academy of Pediatrics policy recommends (Gardner, Qualter, & Whitely, 2011): First, psychosocial problems are no longer viewed as different in causes and consequences then any other physical illness. Second, both physical and mental health training should be incorporated in health care. Third, protecting children from stress and adversity will help minimize a variety of social problems. Fourth, focusing on reducing adversity and stress as a priority will help decrease future psychological and physical disease. Fifth, screening for toxic stress in childhood and promoting positive parenting techniques will support positive health behaviors.

Summary

Although little is known about the psychobiological processes of adults at age 23 who were born prematurely, there is agreement, that the origins of adulthood disease involve biological and developmental factors including social, educational and environmental, effects from early on in life. Shonkoff, Boyce And McEwen, 2009, identify two contributing causes to future adult disease which are applicable to both physical and psychological problems: 1. Cumulative damage from stress defined as allostatic load. 2. The effect of adversity during a sensitive biological growth period, which is now defined

as biological embedding. This study's primary purposes were to: 1. To assess the role of stress, coping, emotional intelligence and emotional health in the prematurely born. 2. Examine the HPA axis cortisol response of the prematurely born age 23 year old to induced stress. 3. Examine the relationship between self-reported psychological problems and induced stress HPA Axis responses.

The study confirmed this time period as one of above average stress, confirmed previous findings related to maturity of coping styles of age 23 year old young adults, and identified a small percentage with psychological clinical problems at present. The prematurely born are comparable to those born full term born in demographics, coping, EQi and emotional disorders, with all perinatal groups primarily in an average range.

Difference between perinatal groups at age 23 appear in cortisol responses to induced social stress that may signal risk for later adulthood disorders. The prematurely born with medical and neurological problems when coupled with varying extremes of stress and socio-economic levels have differences in both cortisol changes and the total cortisol concentration. This perinatal risk group requires close follow-up, preventive strategies and timely interventions. Differences in cortisol responses between self reported psychological problems are congruent with the differing patterns of acute stress cortisol responses. The findings support the theoretical assumptions, previous research and the evidence-based recommendations from the Academy of Pediatricians.

Future research to identify later adulthood stress, coping, emotional health and cortisol responses in relationship to psychological problem development and coexisting medical problems is warranted. Education and interventions to reduce stress earlier in life requires emphasis of the many factors converging with biology to create health problems. At age 23

the participants were still transitioning into young adulthood and would benefit from stress reduction and coping skills to reduce prolonged stress. Nursing is in a unique position to incorporate stress identification and stress reduction into care and educate about the social, mind, body and environmental impacts of stress on health. Nurses can advocate for public programs to provide interventions for children, adolescents and young adults in adverse circumstances.

Appendix A Measurements

Concept	Measurement	Psychometrics	Score Range	Variable
I. DEMOGRAPHIC: Birth, Age 23 & Hollingshead				
Age 23	Education Employed Minority Status		Low $\geq$ 12 years High < 12 years Yes or No Low: White High: Non-white	<u>Education</u> High School GED Prep Transitional Vocational Community College Graduate Other
Perinatal Birth Group & Neonatal Risk Variables	Birth weight grams Gestational Age in weeks (GA) Hobel neonatal score Hobel birth total score Oxygen duration in hours Number of days Hospitalized	1151-3420 grams 28 – 40 weeks Starts at 0 Starts at 0 None - > 24 hours None –days	None or present IVH grades I-IV	<u>Perinatal Birth Group (Total 5 groups)</u> FT = Full Term: 40 weeks, 3420 gms & healthy HPT = Healthy Preterm: < 37 wks, 1518 gms & no complications SGA = Small for gestational age < 37 wks, < 10%ile weight for GA MPT = Medical Preterm: < 37wks, 1268 gms & Dx.:

	Diagnoses (Dx.): Intraventricular Hemorrhage (IVH) Bronchopulmonary Dysplasia (BPD) Necrotizing Enterocolitis (NEC) Sepsis Meningitis Hydrocephalus			BPD, sepsis, NEC & IVH: I, II NPT = Neurological Preterm: < 37wks, 1149 gms & Dx.: Meningitis, Hydrocephalus, IVH: III, IV
Social Status @ Birth & Age 23	Hollingshead Four Factor Index of Social Status	Interrater: r range = .86-.91 Intermeasure r range = .81-.88	Weighed scores range 8-66 with 5 categories: Lower 8-19 Lower-middle 20-29 Middle 30-39 Upper Middle 40-54 Upper 55-66	SES = Social Economic Status Education Sex/gender Marital status
II. STRESS: DHS-R				
Everyday stress & magnitude	Daily Hassles Scale-Revised	Internal consistency: $r = .93-.97$ over 2 years	Weighed scores range 0-159: Above average	53 items <u>DHS: Scores 0 – 159</u>

		Mean day to day: $r = .77$ Mean monthly: $r = .82$	>30 Average 25-30 Below Average < 25	
III. COPING: CRI-A				
Coping Responses: Types & Strategies	Coping Response Inventory- Adult Form	Internal consistency: .61-.74 males .58-.71 females	t -scores range: 20-80, M50 (SD10) Below average ≤ 34 Average 35-65 Above average ≥ 66 <u>4 Coping Responses:</u> Cognitive Approach Cognitive Avoidance Behavioral Approach Behavioral	48 items <u>2 Types= approach & avoidance</u> <u>Approach = scores 20-80 (M50, SD10)</u> Logical analysis Positive reappraisal Seeking guidance & support Problem solving <u>Avoidance = scores 20-80 (M50, SD 10)</u> Cognitive avoidance Acceptance or Resignation Seeking alternative Rewards Emotional

			Avoidance	Discharge <u>2 coping strategies:</u> Cognitive Behavioral
IV. EMOTIONAL INTELLIGENCE: Bar-On EQi				
Emotional & social behaviors dealing with daily demands	Bar-On EQi	<p>Internal consistencies: r range = 69-.96</p> <p>All composite Scales to Total EQi score: r range = .67-.93</p> <p>Test-retest reliability at 6 months: range .72-.80</p>	<p>Raw score Standardized M100 (SD 15)</p> <p><u>5 composite scores:</u> Intrapersonal Interpersonal Adaptability Stress Management General Mood Self-motivation</p> <p>Overall EQi score: M 100 (SD 15)</p> <p>Higher the score</p>	<p>133 items</p> <p>15 subscales related to 5 composite scores</p> <p>EQi total score: M100 (SD 15)</p>

			higher the potential for effective emotional & social functioning	
V. EMOTIONAL HEALTH: ASR				
Assess emotional health	Adult Self-Report	<p>Internal Consistency: r range = .78-.85</p> <p>Reliability: r range = .80-.90 after 1 week r = .69</p>	<p>t scores of clinical risk: >60 for risk to emotional health</p> <p>DSM – V oriented Dx: Normal <59 Borderline 60-63 Clinical $\geq$ 64</p> <p>Depression Anxiety Somatic Avoidant personality AD/H Antisocial</p>	<p>Scores: < 59 - $\geq$ 64</p> <p>Depression Anxiety Somatic Avoidant Personality AD/H Antisocial</p>

VI. STRESS REACTIVITY: TSST				
<p>Induce psychological stress: Threat of social Evaluation & perception of uncontrollability</p> <p>HPA Axis activation</p>	The Trier Social Stress Test	<p>Biological Parameters responsive to TSST through blood samples, saliva & physical measurements: Endocrine immunological others</p>	<p><u>Cortisol levels:</u> Baseline the Immediately after 15, 30, 45, 60, 75 & 90 minutes after test</p> <p><u>Usual cortisol response:</u> Increase baseline to peak by 50-300% over baseline</p> <p><u>Usual psychological response:</u> Self-report negative mood changes & moderate increase in anxiety ratings</p>	<p>TSST</p> <p><u>3 parameters:</u> Endocrine = cortisol Immunological Others</p>

			Heart rate & BP	
VII. HPA AXIS BIOMARKER CORTISOL: Salivary samples				
Cortisol confounder Exclusion criteria	Urine Pregnancy Screening Test	Sensitivity: 20 mIU/ml	<p>Detects human Chorionic Gonadatropin (hCG) & reacts with rabbit anti-G.</p> <p>Positive reaction = Pregnancy</p>	<p><u>OSOM hCG Combo:</u> Positive Negative</p>
	Urine Toxicology	<p>Sensitivity & specificity within, between tests and testers, confirmed by gas chromatography- mass spectrometry (GC/MS)</p>	<p><u>Test and control area:</u> Negative = line in both test & control area</p> <p>Positive = if Line occurs only in control area</p>	<p><u>Innovacon 8 panel:</u> Positive Negative</p>

		<p>Drug cut off points for abuse According to Federal Substance Abuse & Mental Health Administration (SAMHSA)</p>	<p><u>Drugs of Abuse:</u> Cocaine Amphetamine Meth- Amphetamine Marijuana Opiates Phencyclidine Barbiturates Benzodiazepines</p>	
Salivary cortisol levels (Immunoassay)	Expanded Range High Sensitivity Salivary Cortisol Enzyme Immunoassay	<p>Duplicate assays of each 25 uL sample of saliva each test</p> <p>Sensitivity Minimum cortisol Amount from 0 = < .003 ug/dL</p> <p>Correlation between saliva & serum: $r = .91$, $p = .0001$</p> <p>Sensitivity 100% & Specificity</p>	<p>Range: .003 – 3.0 ug/dL</p> <p>6 sample times from TSST paradigm at 15, 30, 45, 60, 75, & 90 minutes</p>	Salimetrics: ERHS-EIA

		<p>95% between Cushing Syndrome & Obesity</p> <p>Built in hydrogen (ph) alert if levels artificially high or low: (≤ 3.5 or ≥ 9.0)</p>		
Stress reactivity & recovery period total cortisol output	<p>Area under the curve with respect to ground</p> <p>Total TSST Cortisol Response</p>	<p>Total distance area under plotted curve of cortisol measures starting from zero</p> <p>Total</p>	<p>Differences between groups in response as measured by AUCg at 6 sample times of TSST recovery period</p> <p>If less of a slope and/or limited changes in fluctuations along with higher cortisol levels = blunted response/ HPA dysregulation</p>	<p>AUCg Slope</p> <p>Total</p>

Appendix B

*Power Analyses*ANOVA: $N = 166$, 5 Groups Protocol

F tests - ANOVA: Fixed effects, omnibus, one-way

Analysis: Sensitivity: Compute required effect size

Input:	α err prob	=	0.05
	Power ($1-\beta$ err prob)	=	0.8
	Total sample size	=	166
	Number of groups	=	5
Output:	Noncentrality parameter λ	=	12.2916735
	Critical F	=	2.4278100
	Numerator df	=	4
	Denominator df	=	161
	Effect size f	=	0.2721144

ANOVA: $N = 166$, 5 Groups Distribution Plot

Appendix B

continued

ANOVA: $N = 142$, 5 Groups Protocol

F tests - ANOVA: Fixed effects, omnibus, one-way

Analysis: Sensitivity: Compute required effect size

Input:	α err prob	=	0.05
	Power ($1-\beta$ err prob)	=	0.8
	Total sample size	=	142
	Number of groups	=	5
Output:	Noncentrality parameter λ	=	12.3550884
	Critical F	=	2.4377490
	Numerator df	=	4
	Denominator df	=	137
	Effect size f	=	0.2949706

ANOVA: $N = 142$, 5 Groups Distribution Plot

Appendix B

continued

ANCOVA: $N = 170$, 5 Groups & 2 Covariates Protocol

F tests - ANCOVA: Fixed effects, main effects and interactions

Analysis: Sensitivity: Compute required effect size

Input:	α err prob	=	0.05
	Power ($1-\beta$ err prob)	=	0.8
	Total sample size	=	170
	Numerator df	=	7
	Number of groups	=	5
	Number of covariates	=	2
Output:	Noncentrality parameter λ	=	14.9775903
	Critical F	=	2.0661634
	Denominator df	=	163
	Effect size f	=	0.2968223

ANCOVA: $N = 170$, 5 Groups and 2 Covariates Distribution Plot

Appendix B

continued

ANCOVA: $N = 166$, 5 Groups and 1 Covariate Protocol

F tests - ANCOVA: Fixed effects, main effects and interactions

Analysis: Sensitivity: Compute required effect size

Input:	α err prob	=	0.05
	Power ($1-\beta$ err prob)	=	0.80
	Total sample size	=	166
	Numerator df	=	6
	Number of groups	=	5
	Number of covariates	=	1
Output:	Noncentrality parameter λ	=	14.1674669
	Critical F	=	2.1556612
	Denominator df	=	160
	Effect size f	=	0.2921407

ANCOVA: $N = 166$, 5 Groups and 1 Covariate Distribution Plot

Appendix B

continued

ANCOVA, $N = 166$, 5 Groups and 2 Covariates Protocol

F tests - ANCOVA: Fixed effects, main effects and interactions

Analysis: Sensitivity: Compute required effect size

Input:	α err prob	=	0.05
	Power ($1-\beta$ err prob)	=	0.8
	Total sample size	=	166
	Numerator df	=	7
	Number of groups	=	5
	Number of covariates	=	2
Output:	Noncentrality parameter λ	=	14.9935698
	Critical F	=	2.0676042
	Denominator df	=	159
	Effect size f	=	0.3005374

ANCOVA: $N = 166$, 5 Groups and 2 Covariates Distribution Plot

Appendix B

continued

ANCOVA, $N = 166$, 5 Groups and 3 Covariates Protocol

F tests - ANCOVA: Fixed effects, main effects and interactions

Analysis: Sensitivity: Compute required effect size

Input:	α err prob	=	0.05
	Power ($1-\beta$ err prob)	=	0.80
	Total sample size	=	166
	Numerator df	=	8
	Number of groups	=	5
	Number of covariates	=	3
Output:	Noncentrality parameter λ	=	15.7686374
	Critical F	=	1.9974370
	Denominator df	=	158
	Effect size f	=	0.3082074

ANCOVA: $N = 166$, 5 Groups and 3 Covariates Distribution Plot

Appendix B

continued

ANCOVA, $N = 142$, 5 Groups and 1 Covariate Protocol

F tests - ANCOVA: Fixed effects, main effects and interactions

Analysis:	Sensitivity: Compute required effect size		
Input:	α err prob	=	0.05
	Power ($1-\beta$ err prob)	=	0.80
	Total sample size	=	142
	Numerator df	=	6
	Number of groups	=	5
	Number of covariates	=	1
Output:	Noncentrality parameter λ	=	14.2648115
	Critical F	=	2.1658823
	Denominator df	=	136
	Effect size f	=	0.3169486

ANCOVA, $N = 142$, 5 Groups and 1 Covariate Distribution Plot

Appendix B

continued

ANCOVA, $N = 142$, 5 Groups and 2 Covariates Protocol

F tests - ANCOVA: Fixed effects, main effects and interactions

Analysis:	Sensitivity: Compute required effect size		
Input:	α err prob	=	0.05
	Power ($1-\beta$ err prob)	=	0.8
	Total sample size	=	142
	Numerator df	=	7
	Number of groups	=	5
	Number of covariates	=	2
Output:	Noncentrality parameter λ	=	15.1096354
	Critical F	=	2.0780690
	Denominator df	=	135
	Effect size f	=	0.3261991

ANCOVA, $N = 142$, 5 Groups and 2 Covariates Distribution Plot

Appendix B

continued

ANCOVA, $N = 142$, 5 Groups and 3 Covariates Protocol

F tests - ANCOVA: Fixed effects, main effects and interactions

Analysis:	Sensitivity: Compute required effect size		
Input:	α err prob	=	0.05
	Power ($1-\beta$ err prob)	=	0.80
	Total sample size	=	142
	Numerator df	=	8
	Number of groups	=	5
	Number of covariates	=	3
Output:	Noncentrality parameter λ	=	15.9043340
	Critical F	=	2.0081589
	Denominator df	=	134
	Effect size f	=	0.3346675

ANCOVA, $N = 142$, 5 Groups and 3 Covariates Distribution Plot

Appendix C

Institutional Review Board

THE
UNIVERSITY
OF RHODE ISLAND
DIVISION OF RESEARCH
AND ECONOMIC
DEVELOPMENT

OFFICE OF RESEARCH INTEGRITY

70 Lower College Road, Suite 2, Kingston, RI 02881 USA

p: 401.874.4328 f: 401.874.4814 web.uri.edu/researchecondev/office-of-research-integrity

FWA: 00003132

IRB: 00000599

DATE: May 17, 2016

TO: Mary Sullivan, RN, PhD

FROM: University of Rhode Island IRB

STUDY TITLE: Stress, Coping, Emotional Intelligence, Emotional Health & Cortisol in
Preterm Infants at Age 23

IRB REFERENCE #: 910108-1

LOCAL REFERENCE #: HU1516-180

SUBMISSION TYPE: New Project

ACTION: DETERMINATION OF NOT HUMAN SUBJECT RESEARCH

EFFECTIVE DATE: May 17, 2016

Thank you for your submission of New Project materials for this research study. The University of Rhode Island IRB has determined this project does not meet the definition of human subject research under the purview of federal regulation 45 CFR 46 regarding human subject research at this time. Therefore, your project does not require Institutional Review Board (IRB) oversight. Any changes in focus of this project will require further review of the IRB.

If you have any questions, please contact us by email at researchintegrity@etal.uri.edu. Please include your study title and reference number in all correspondence with this office.

Matthew Delmonico, Ph.D., MPH
IRB Chair

Appendix D

Correlations and Descriptive Statistics (N = 166): Perinatal Birth Group & Age 23 Years

Variables	1	2	3	4	5	6	7	8	9	10	11
1. IQ Score age 17	-										
2. Birth Weight	.046	-									
3. Hobel Neonatal Score	-.039	-.850**	-								
4. Birth – SES Score	.411**	-.086	.127	-							
5. SES Age 23	.459**	.049	.019	.545**	-						
6. Total Hassles	-.107	.061	-.106	-.097	-.119	-					
7. Logical Analysis	.394**	.072	-.106	.262**	.296**	.016	-				
8. Positive Appraisal	.178*	-.020	-.086	.084	.144	.020	.638**	-			
9 Seek Guide & Support	.037	.118	-.107	.051	.121	.044	.449**	.386**	-		
10. Problem Solving	.237**	-.057	.057	.103	.133	-.167*	.612**	.561**	.363**	-	

Appendix D

continued

Variables	1	2	3	4	5	6	7	8	9	10	11
11. Cognitive Avoidance	.038	-.034	-.081	-.105	-.082	.408**	.148	.048	.035	-.012	-
12. Acceptance or Resignation	-.001	.034	-.015	-.076	-.065	.382**	.064	.008	.219**	-.103	.434**
13. Seek Alter Rewards	.045	-.118	.036	.062	.130	-.015	.411**	.373**	.211**	.362	.065
14. Emotional Discharge	-.070	-.017	-.034	-.189*	-.162*	.481**	.132	.074	.197*	.075	.529**
15. Total EQi	.299**	-.004	.053	.168*	.247**	-.423**	.201*	.219**	.230**	.312**	-.336**
16. ASR – Total Problems (Emotional Health)	-.137	.064	-.096	-.118	-.164*	.717**	-.178*	-.124	-.173*	-.313**	.451**
Variables	1	2	3	4	5	6	7	8	9	10	11
<i>N</i>	149	166	166	166	159	163	155	155	155	155	155
<i>M</i>	100.33	1784.35	64.82	39.19	40.58	31.10	50.74	52.99	50.37	52.83	54.53
<i>SD</i>	12.90	976.14	45.56	14.6	13.61	21.51	9.89	8.58	8.42	10.01	9.72

Appendix D

continued

Variables	12	13	14	15	16
12. Acceptance or resignation	-				
13. Seek Alter Rewards	.067	-			
14. Emotional Discharge	.390**	.151	-		
15. Total EQi	-.233**	.114	-.370**	-	
16. ASR – Total Problems (Emotional Health)	.323**	-.109	.437**	-.653**	-
Variables	12	13	14	15	16
<i>N</i>	155	154	155	158	163
<i>M</i>	52.83	53.79	57.13	98.75	48.39
<i>SD</i>	9.41	9.04	10.15	15.60	10.47

* = Correlation Significant at $p = 0.05$ (Two - tail); ** = Correlation Significant at $p = 0.01$ (Two - tail)

Appendix E

Levene's Test for Homogeneity of Variance (Pairwise)

	Perinatal Birth Group	Gender	Perinatal Birth X Gender
	$F(df1, df2) = \text{Levene's Statistic}, p$		
Aim I. Dependent Variables Stress (DHS), Coping (CRI), Emotional Intelligence (EI) and Emotional Health (EH):			
Hassles	$F(4,147) = 1.233, p = .299$	$F(1,150) = 2.264, p = .135$	$F(9, 153) = 1.762, p = .080$
Logical Analysis	$F(4,147) = 1.025, p = .397$	$F(1,150) = 3.032, p = .084$	$F(9,145) = 2.271, p = .021^*$
Positive Appraisal	$F(4,147) = .928, p = .449$	$F(1,150) = .027, p = .871$	$F(9,145) = 1.107, p = .362$
Seek Guidance & Support	$F(4,147) = .302, p = .876$	$F(1,150) = .417, p = .520$	$F(9,145) = 1.263, p = .262$
Problem Solving	$F(4,147) = 1.379, p = .261$	$F(1,150) = .006, p = .938$	$F(9,145) = .661, p = .743$
Cognitive Avoidance	$F(4,147) = 1.327, p = .305$	$F(1,150) = .744, p = .390$	$F(9,145) = 1.311, p = .236$
Accept or Resignation	$F(4,147) = 1.192, p = .317$	$F(1,150) = 1.135, p = .288$	$F(9,145) = 2.131, p = .030^*$

Appendix E

continued

	Perinatal Birth Group	Gender	Perinatal Birth X Gender
Seek Alternative Rewards	$F(4,147) = .374, p = .850$	$F(1,150) = 3.269, p = .073$	$F(9,144) = .828, p = .591$
Emotional Discharge	$F(4,147) = .713, p = .584$	$F(1,150) = .461, p = .498$	$F(9,145) = .894, p = .532$
Total EQI	$F(4,147) = .447, p = .774$	$F(1,150) = .026, p = .873$	$F(9,148) = .488, p = .881$
ASR	$F(4,147) = .310, p = .871$	$F(1,150) = .331, p = .566$	$F(9,153) = .561, p = .561$

Aim II. Salivary Cortisol TSST Stress Response and Recovery

DELTA change from 2 nd baseline to peak	$F(4,137) = 1.699, p = .154$	$F(1,140) = 2287, p = .133$	$F(9,139) = 1.494, p = .156$
PEAK of cortisol after TSST	$F(4,137) = 1.140, p = .341$	$F(1,140) = .787, p = .337$	$F(9,139) = .743, p = .669$
SLOPE – Average cortisol			

Appendix E

continued

change/minutes to reach peak $F(4,137) = 1.721, p = .149$ $F(1,140) = 3.146, p = .078$ $F(9,138) = 1.661, p = .104$

	Perinatal Birth Group	Gender	Perinatal Birth X Gender
AUCg Total	$F(4,137) = .1.830, p = .126$	$F(1,140) = .049, p = .825$	$F(9,132) = 1.759, p = .082$
Aim III. Emotional Health (ASR) and HPA Axis Response (Salivary Cortisol TSST Stress Response and Recovery)			
ASR	$F(4,137) = .160, p = .958$	$F(1,140) = .000, p = .995$	$F(4, 137) = 1.936, p = .108$
* Levene's Test of Perinatal Birth Group with Covariate Gender			
Hassles	$F(4,158) = 1.793, p = .133$		
Logical Analysis	$F(4,150) = .942, p = .441$		

Appendix E

continued

Levene's Test of Perinatal Birth Group with Covariate Gender

Positive Appraisal	$F(4,150) = .992, p = .414$
Seeking Guidance and Support	$F(4,150) = .385, p = .819$
Problem Solving	$F(4,150) = 1.347, p = .255$
Cognitive Avoidance	$F(4,150) = 1.047, p = .385$
Acceptance or resignation	$F(4,150) = 1.258, p = .289$
Seek Alternative Rewards	$F(4,149) = .343, p = .849$
Emotional Discharge	$F(4,150) = 1.065, p = .376$
Total EQI	$F(4,153) = .251, p = .909$
ASR	$F(4,158) = .172, p = .952$
Delta	$F(4,144) = 1.375, p = .245$

Appendix E

continued

Levene's Test of Perinatal Birth Group with Covariate Gender

Peak	$F(4,144) = .708, p = .588$
Slope	$F(4,143) = 1.395, p = .239$
AUC _g	$F(4,137) = 1.935, p = .108$

Appendix F

Perinatal Group by Gender: Neonatal Demographic and Medical Diagnoses Data, N=166

Perinatal Group by Gender										
Variables	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
	<i>M (SD)</i>									
	<u>Analysis: ANOVA</u>									
Birth wgt.gms.	3323	3409.83	1509	1442.86	1285.54	1276.42	1247.06	1018.33	1380	106.25
	(78.79)	(69.71)	(105.72)	(89.35)	(63.18)	(772.95)	(81.08)	(111.44)	(136.48)	(74.74)
	<u>$F(4, 156) = 1.622, p = .171$</u>									
GA weeks	39.72	39.83	31.20	30.93	29.64	29.05	28.82	28.50	33.83	31.45
	(.669)	(1.03)	(1.75)	(1.73)	(2.35)	(2.29)	(1.98)	(2.62)	(2.99)	(2.67)
	<u>$F(4, 156) = 1.317, p = .266$</u>									

Appendix F

continued

Variables	Perinatal Group by Gender									
	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
	<i>M (SD)</i>									
<u>Analysis: ANOVA</u>										
Hobel NN	12.67	13.26	106.10	117.75	141.96	145.62	163	174.83	129.83	140.08
	(10.07)	(14.16)	(38.69)	(31.12)	(30.27)	(36.46)	(29.14)	(29.84)	(25.47)	(41.62)
<i>F(4,156) = .276, p = .893; Levene's F(9, 156) = 6.976), p = .000</i>										
Hobel Total	2.33	.91	54.30	59.96	90.84	88.43	110.12	116.50	72.33	77.98
	(4.58)	(2.45)	(19.97)	(24.98)	(24.46)	(27.44)	(24.99)	(16.75)	(36.71)	(33.29)
<i>F(4, 156) = .231, p = .921</i>										

Appendix F

continued

Variables	Perinatal Group by Gender									
	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
	<i>M (SD)</i>									
<u>Analysis: ANOVA</u>										
Hobel Total	2.33	.91	54.30	59.96	90.84	88.43	110.12	116.50	72.33	77.98
	(4.58)	(2.45)	(19.97)	(24.98)	(24.46)	(27.44)	(24.99)	(16.75)	(36.71)	(33.29)
<u><i>F</i>(4, 156) = .276, <i>p</i> = .893</u>										
O ₂ Duration	0	0	13.86	13.62	609.39	256.09	606.12	680	386	218.70
in hours			(15.03)	(21.26)	(759.19)	(339)	(750.79)	(484.03)	(861.85)	(477.95)
<u><i>F</i>(4,156 = 1.180, <i>p</i> = .372 / Levene's: <i>F</i> (9,156) = 16.589, <i>p</i> = .000*</u>										

Appendix F

continued

Variables	Perinatal Group by Gender									
	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
	<i>M (SD)</i>									
<u>Analysis: ANOVA</u>										
Number of days	3.17	2.91	31.70	34.00	53.36	51.62	62.29	70.56	39.33	55.90
	(0.51)	(0.29)	(7.96)	(11.31)	(23.87)	(24.26)	(24.49)	(15.90)	(30.56)	(31.42)
<u><i>F</i>(4, 165) = .842, <i>p</i> = .500</u>										

Appendix F

continued

Perinatal Group by Gender										
Neonatal Medical	FT		HPT		MPT		NPT		SGA	
Diagnoses	M	F	M	F	M	F	M	F	M	F
N (%)										
Analysis										
IVH	0	0	1	1	1	1	9	8	0	3
$X^2(16,166) = 72.077^a, p = .001$ (20 cells 80% >5)										
Grade 1	0	0	1 (10)	0	0	0	2 (11.8)	3 (33)	0	1(5)
Grade II	0	0	0	1 (7)	1 (3.6)	1(4.8)	1 (5.9)	4 (44)	0	0
Grade III	0	0	0	0	0	0	5 (29)	1 (11)	0	1 (5)
Grade IV	0	0	0	0	0	0	1 (6)	0	0	1 (5)

Appendix F

continued

Perinatal Group by Gender										
Neonatal Medical	FT		HPT		MPT		NPT		SGA	
Diagnoses	M	F	M	F	M	F	M	F	M	F
N (%)										
Analysis										
BPD	0	0	0	0	8 (28.6)	3 (14.3)	5 (29.4)	3 (33)	1 (17)	1 (5)
$X^2(4,166) = 21.974^a, p = .001$ (3 cells 30% >5)										
NEC	0	0	0	0	3 (11)	4 (19)	1 (5.9)	1 (11)	0	3 (15)
$X^2(4,166) = 9.432^a, p = .051$ (5 cells 50% >5)										
Sepsis	0	0	0	0	2 (7)	3 (14.3)	5 (29.4)	1 (11)	2 (33)	1 (5)
$X^2(4,166) = 13.792^a, p = .008$ (5 cells 50% >5)										

Appendix F

continued

Perinatal Group by Gender										
Neonatal Medical Diagnoses	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
<i>N (%)</i>										
Analysis										
Meningitis	0	0	0	0	0	0	3 (17.3)	0	1 (16.7)	0
$X^2(4,166) = 12.257^a, p = .016$ (5 cells 50% >5)										
Hydrocephalus	0	0	0	0	0	0	3 (17.6)	0	0	0
$X^2(4,166) = 16.451^a, p = .002$ (5 cells 50% >5)										

Appendix G

Perinatal Group Age 23 by Gender Demographic Data N = 166

Variable	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
$N(%)$ Analysis										
Race: $X^2(16) = 15.302, p = .503$										
African	1 (1.3)	3 (3.6)	1 (1.3)	1 (1.2)	3 (4)	2 (2.4)	2 (2.7)	0	0	1 (1.2)
White	15 (20)	17 (22.7)	9 (12)	10 (12)	22 (29.3)	18 (21.7)	14 (18.7)	8 (9.6)	6 (8)	19 (22.9)
Native	1 (1.3)	0	0	1 (1.2)	0	0	0	0	0	0
Asian	0	0	0	0	1 (1.3)	0	0	0	0	0
> 1 race	0	0	0	2 (2.4)	0	0	0	1 (1.2)	0	0

Appendix G

continued

Variable	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
$N(\%)$ Analysis										
Educational Level										
High School: $\chi^2(8) = 2.417, p = .965$										
GED	0	2 (2.4)	1 (1.5)	1 (1.2)	1 (1.5)	1 (1.2)	1 (1.5)	0	0	0
Diploma	13 (19.1)	16 (19.3)	8 (11.8)	13 (15.7)	21 (30.9)	19 (22.9)	13 (19.1)	9 (10.8)	5 (7.4)	20 (24.1)
Prep School	1 (1.4)	0	1 (1.4)	0	0	0	0	0	0	0
$\chi^2(4) = 3.471, p = .482$										
Vocational	0	0	1 (1.4)	1 (1.2)	3 (4.1)	2 (2.4)	0	2 (2.4)	0	1 (1.2)
$\chi^2(4) = 4.568, p = .335$										

Appendix G

continued

Variable	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
$\chi^2(4) = 2.201, p = .699$										
Community	0	1 (1.2)	0	0	1 (1.4)	1 (1.2)	0	1 (1.2)	0	2 (2.4)
$\chi^2(4) = 6.157, p = .188$										
College	7 (9.5)	8 (9.6)	2 (2.7)	7 (8.4)	9 (12.2)	15 (18.1)	2 (2.7)	3 (3.6)	2 (2.7)	9 (10.8)
$\chi^2(4) = 5.029, p = .284$										
Graduate	1 (1.4)	1 (1.2)	0	1 (1.2)	0	0	0	0	0	0
$\chi^2(4) = 2.377, p = .667$										
Transitional	0	0	0	0	1 (1.4)	0	0	0	0	0

Appendix G

continued

Variable	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
N (%) Analysis										
Employed	11 (14.7)	14 (16.7)	5 (6.7)	6 (7.1)	21 (28)	16 (19)	14 (18.7)	7 (8.3)	5 (6.7)	13 (15.5)
X ² (1,4) = 7.0322, p = .334										
Occupational Category: X ² (12) = 21.430 p = .044 (5 cells < 5)										
Unskill/skill	8 (10.7)	12 (14.3)	7 (9.3)	7 (8.3)	13 (17.3)	6 (7.1)	11 (14.7)	5 (6)	3 (4)	5 (6)
Semiprof	4 (5.3)	4 (4.8)	0	1 (1.2)	11 (14.7)	6 (7.1)	1 (1.3)	3 (3.6)	2 (2.7)	5 (6)
Prof/exec	3 (4)	0	0	1 (1.2)	0	1 (1.2)	1 (1.3)	0	0	0
Student	2 (2.7)	4 (4.8)	3 (4)	5 (6)	2 (2.7)	8 (9.5)	3 (4)	1 (1.2)	1 (1.3)	10 (11.9)
Marital Status: X ² (8) = 8.675, p = .370										
Single	12 (16)	14 (16.9)	7 (9.3)	6 (7.2)	21 (28)	16 (19.3)	14 (18.7)	6 (7.2)	6 (8)	18 (21.7)

Appendix G

continued

Variable	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M N (%) Analysis	F	M	F	M	F
Cohab or										
married	5 (6.7)	6 (7.2)	3 (4)	2 (2.4)	4 (5.3)	5 (6)	2 (2.7)	3 (3.6)	0	1 (1.2)
Sep/div/wid	0	0	0	0	1 (1.3)	0	0	0	0	0
IQ Score Categories at Age 17: $\chi^2(16) = 10.417, p = .844$										
≥ 130	0	0	0	0	0	0	0	0	0	0
120 – 129	1 (1.5)	1 (1.2)	2 (3)	0	1 (1.5)	3 (3.7)	0	1 (1.2)	0	1 (1.2)
110 – 119	1 (1.5)	5 (6)	1 (1.5)	4 (4.9)	4 (6)	1 (1.2)	3 (4.5)	1 (1.2)	1 (1.5)	4 (4.9)
90 – 109	11 (16.4)	12 (14.6)	2 (3)	5 (6.1)	13 (19.4)	9 (11)	6 (9)	6 (7.3)	4 (6)	12 (14.6)
80 – 89	3 (4.5)	2 (2.4)	2 (3)	3 (3.7)	2 (3)	2 (2.4)	6 (9)	0	1(1.5)	2 (2.4)

Appendix G

continued

	FT		HPT		MPT		NPT		SGA	
	M	F	M	F	M	F	M	F	M	F
	<i>M (SD)</i> Analysis									
70 – 79	0	1 (1.2)	1 (1.5)	1 (1.2)	2 (3)	2 (2.4)	0	1 (1.2)	0	1 (1.2)
Age	23.6 (1)	23.3 (<1)	23 (<1)	23 (<1)	23.3 (<1)	22.9 (1)	23.4 (1.1)	23.4 (1.3)	22.8 (<1)	22.7(<1)
SES	45(17)	39.23 (12)	32 (15)	42 (13)	38 (13)	44(12)	38 (15)	44 (13)	40 (11)	42 (12)
	$F(4, 149) = 1.597, p = .178$									
IQ	101 (11)	101 (13)	101 (20)	98 (16)	101 (12)	103 (15)	97 (13)	100 (15)	98 (9)	101 (12)
	$F(4, 139) = .180, p = .948$									

Appendix H

Gender Neonatal Demographic and Medical Diagnoses Data, N=166

Variables	Sample	Male	Female
	$N = 166$	$n = 79 (47.5\%)$	$n = 87 (52.4\%)$
	$M (SD)$	$M (SD)$	$M (SD)$
<u>Analysis: One-way ANOVA (analysis by analysis)</u>			
Birth weight in grams	1784.35 (976)	1776.95 (910.89)	1791.07 (1037.06)
Range	640-4370	810 - 4255	640 - 4370
$F(1, 164) = .009, p = .926$			
Gestational age in weeks	32.46 (4.8)	32.28 (4.69)	32.63 (4.90)
Range	25-43	25 – 40	25 – 43
$F(1, 164) = .225, p = .636$			
Hobel neonatal score	65.82 (45.6)	68.79 (45.45)	61.21 (45.62)

Appendix H

continued

Variables	Sample	Male	Female
	$N=166$	$n = 79 (47.5\%)$	$n = 87 (52.4\%)$
	$M (SD)$	$M (SD)$	$M (SD)$
<u>Analysis: One-way ANOVA</u>			
Range	0-160	0 – 160 Appendix H	0 - 136
		<u>$F(1, 164) = .1147, p = .286$</u>	
Hobel birth total score	109.64 (64.6)	111.57 (62.63)	107.89 (66.6)
Range	0-220	0 – 220	0 - 211
		<u>$F(1, 164) = .134, p = .715$</u>	
O ₂ Duration in hours	276.41 (540.3)	377.49 (665.8)	184 (373.3)
Range	0-2568	0 – 2568	0 – 2064

Appendix H

continued

Variables	Sample	Male	Female
	$N=166$	$n = 79$ (47.5%)	$n = 87$ (52.4%)
	M (SD)	M (SD)	M (SD)
<u>Analysis: One-way ANOVA</u>			
<u>$F(1, 164) = 5.416, p = .021^*$</u>			
Number of days Hospitalized; Range	39.42 (30.3) 2-134	40.04 (29.70) 3 – 107	38.85 (30.98) 2 - 134
<u>$F(1, 164) = .063, p = .802$</u>			
SES raw score	39.19 (14.6)	38.37 (15.198)	39.93 (14.083)
Range	8-66	8 – 66	11 - 66
<u>$F(1, 164) = .474, p = .492$</u>			

Appendix H

continued

Neonatal Medical Diagnoses	Sample <i>N</i> (%)	Male <i>n</i> (%)	Female <i>n</i> (%)
<u>Analysis: Chi Sq.</u>			
IVH	24 (14.4)		
Grade 1	8(5)	3 (1.8)	5 (3)
Grade II	7 (4)	2 (1.2)	5 (3)
Grade III	7 (4)	5 (3)	2 (1.2)
Grade IV	2 (1)	1 (.6)	1 (.6)
		<u>$\chi^2(4) = 2.946, p = .567$</u>	
BPD	21 (13)	14 (8.4)	7 (4.2)
		<u>$\chi^2(1) = 3.508, p = .061$</u>	

Appendix H

continued

Neonatal Medical Diagnoses	Sample <i>N</i> (%)	Male <i>n</i> (%)	Female <i>n</i> (%)
<u>Analysis: Chi Sq.</u>			
NEC	12 (7)	4 (2.4)	8 (4.8)
<u>$\chi^2(1) = 1.054, p = .051$</u>			
Sepsis	14 (8)	9 (5.4)	5 (3)
<u>$\chi^2(1) = 1.709, p = .265$</u>			
Meningitis	4 (2)	4 (2.4)	0
<u>$\chi^2(1) = 4.514^a, p = .034$ (^a5 cells 50%<5)</u>			
Hydrocephalus	3 (2)	3 (1.8)	0
<u>$\chi^2(1) = 3.365, p = .106$</u>			

Appendix I

Summary of Perinatal Group at Age 23 Demographic Data, $N = 166$

Variable	FT	HPT	MPT	NPT	SGA	
	N (%=within birth group)					Analysis
Gender						$\chi^2(4) = 11.919, p = .018^*$
Male	18 (43.9)	10 (56.1)	28 (41.7)	17 (65.4)	6 (23.1)	
Female	23 (56.1)	14 (58.3)	21 (42.9)	9 (34.6)	20 (76.9)	
Race						$\chi^2(4) = 15.301, p = .503$
African American	4 (10.8)	2 (8.3)	5 (10.9)	2 (8)	1 (3.8)	
White	32 (86.5)	19 (79.2)	0	0	0	
Native American	1 (2.7)	1 (4.2)	0	0	0	
Asian	0	0	1 (2.2)	0	0	
More than 1 race	0	2 (8.3)	0	1 (4)	0	

Appendix I

continued

Variable	FT	HPT	MPT	NPT	SGA	
<i>N</i> (%=within birth group)						Analysis
Educational Level						
High School	29 (87.9)	21 (87.5)	40 (90.9)	22 (91.7)	25 (96.2)	
GED	2 (6.1)	2 (8.3)	2 (4.5)	1 (4.2)	0	
						HS & GED: $X^2(8) = 2.417, p = .965$
Prep School	1 (2.7)	1 (4.2)	0	0	0	$X^2(4) = 3.471, p = .482$
Vocational School	0	2 (8.3)	5 (10.6)	2 (8.3)	1 (3.8)	$X^2(4) = 5.568, p = .335$
Community College	1 (2.9)	0	2 (4.3)	1 (4.2)	2 (8)	$X^2(4) = 2.201, p = .699$
College Graduate	15 (41.7)	9 (37.5)	24 (51.1)	5 (20.8)	11 (42.3)	$X^2(4) = 6.157, p = .188$
Graduate School	2 (5.4)	1 (4.3)	0	0	0	$X^2(4) = 5.029, p = .284$

Appendix I

continued

Variable	FT	HPT	MPT	NPT	SGA	
	N (%=within birth group)					Analysis
Transitional Program	0	0	1 (2.1)	0	0	$\chi^2(4) = 2.377, p = .667$
Employed	25 (67.6)	13 (54.2)	37 (78.7)	21 (84)	18 (69.2)	$\chi^2(4) = 7.032, p = .134$
Occupational Category	$\chi^2(12) = 21.430^a, p = .044[(^a 5 \text{ cells } <); \text{Cramer's } V = .282$					
Unskilled to skilled	20 (54.1)	14 (58.3)	19 (40.4)	16 (64)	8 (30.8)	
Semiprofessional	8 (21.6)	1 (4.2)	17 (36.2)	4 (16)	7 (26.9)	
Professional/executive	3 (8.1)	1 (4.2)	1 (2.1)	1 (4)	0	
Student	6 (16.2)	8 (33.3)	10 (21.3)	4 (16)	11 (42.3)	
Marital Status						$\chi^2(8) = 8.675, p = .370$

Appendix I

continued

Variable	FT	HPT	MPT	NPT	SGA	
	N (%=within birth group)					Analysis
Single	26 (70.3)	19 (79.2)	37 (78.7)	20 (80)	24 (96)	
Cohabit/married	11 (29.7)	5 (20.8)	9 (19.1)	5 (20)	1 (4)	
Sep/divorce/widow	0	0	1 (2.1)	0	0	
IQ Score Categories at Age 17						$\chi^2(16)=10.417, p = .844$
130 and above	0	0	0	0	0	
120 – 129	2 (5.4)	2 (9.5)	4 (9.8)	1 (4.2)	1 (3.8)	
110 – 119	6 (16.2)	5 (23.8)	7 (17.1)	4 (16.7)	5 (19.2)	
90 – 109	23 (62.2)	7 (33.3)	22 (53.7)	12 (50)	16 (61.5)	
80 – 89	5 (13.5)	5 (23.8)	4 (9.8)	6 (25)	3 (11.5)	

Appendix I

continued

Variable	FT	HPT	MPT	NPT	SGA	
	N (%=within birth group)					Analysis
70 – 79	1 (2.7)	2 (9.5)	4 (9.8)	1 (4.2)	1 (3.8)	
Variable	FT	HPT	MPT	NPT	SGA	Analysis
	M (SD)					
Age	23.44 (1)	23 (0.7)	23 (0.9)	23.4 (1)	22.8 (0.8)	$F(4,161) 2.284, p = .063$
SES Raw Scores						$F(1, 164) = .474, p = .492$
	37 (23.3)	24 (15.3)	47 (29.6)	25 (15.7)	26 (16.4)	

Appendix I

continued

Variable	FT	HPT	MPT	NPT	SGA	Analysis
	M (SD)					
	42 (15)	37 (15)	41 (13)	40 (15)	42 (12)	
IQ Raw Scores						$F(1, 147) = .156, p = .693$
	37 (24.8)	21 (14.1)	41 (27.5)	24 (16.1)	26 (17.4)	
	101 (12)	99 (16)	102 (13)	98 (13)	100 (11)	

Appendix J

Gender Correlations

Variables	N	1	2	3	4	5	6	7	8	9	10
1. Gender	166	-									
2. Hassles (Stress)	163	.058	-								
3. Logical Analysis	155	.152	.016	-							
4. Positive Appraisal	155	.133	.020	.638**	-						
5. Seek Guide & Support	155	.287**	.044	.449**	.386**	-					
6. Problem Solving	155	.007	-.167*	.612**	.561**	.363**	-				
7. Cognitive Avoidance	155	.164*	.408**	.148	.048	.035	-.012	-			
8. Accept or Resign	155	.131	.382**	.064	.008	.219**	-.103	.434**	-		
9. Seek Alter Rewards	154	.023	-.015	.411**	.373**	.211**	.362**	.065	.067	-	
10. Emotional Discharge	155	.201*	.481**	.132	.074	.197*	.075	.529**	.390**	.152	-

* $p = .01$

** $p = .05$

Appendix J

Gender Correlations

Variables	N	1	2	3	4	5	6	7	8	9	10
11. Total EQi	158	.052	-.423**	.201*	.219**	.230**	.312**	-.336**	-.233**	.114	-.370**
12. ASR – Total Problems	163	-.099	.717**	-.178*	-.124	-.173*	-.313**	.451**	.323**	-.109	.437**

Variables	N	11	12
11. Total EQi	158	-	
12. ASR – Total Problems	163	-.653**	-

Appendix K

Summary of Age 23 Gender Demographic Data

Variable	M	F	Analysis
<i>N (%)</i>			
Race			$X^2 (4, 158) = 3.866, p = .424$
African American	7 (4)	7 (94)	
White	66 (42)	72 (46)	
Native American	1 (<1)	1 (<1)	
Asian	1 (<1)	0	
More than 1 race	0	3 (2)	
Educational Level			
High School			$X^2 (2, 151) = 2.068, p = .356$
GED	3 (2)	4 (3)	
Diploma	60 (40)	77 (51)	
Prep School	2 (1)	0	$X^2 (1, 158) = 2.299, p = .129$
Vocational School	4 (3)	6 (7)	$X^2 (1, 158) = .002, p = .654$
Community College	1 (< 1)	5 (3)	$X^2 (1, 154) = 2.270, p = .132$
College Graduate	22 (14)	42 (27)	$X^2 (1, 157) = 7.058, p = .008^*$
Graduate School	1 (< 6)	2 (1)	$X^2 (1, 157) = .234, p = .629$
Transitional Program	1 (< 6)	0	$X^2 (1, 158) = 1.142, p = .285$

Appendix K

continued

Variable	M N (%)	F N (%)	Analysis
Employed	56 (35)	58 (37)	$X^2 (1, 159) = .617, p = .432$
Occupational Category	$X^2 (1, 159) = 8.257^a, p = .041$ (^a 2 cells, 25 & <5)		
Unskilled to skilled	42 (26)	35 (22)	
Semiprofessional	18 (11)	19 (12)	
Professional/executive	4 (3)	2 (1)	
Student	11 (7)	28 (18)	
Marital Status	$X^2 (2, 158) = 1.174, p = .556$		
Single	60 (38)	66 (42)	
Cohabiting/ married	14 (9)	17 (11)	
Sep, div, widow	1 (< 1)	0	
IQ Score Categories at Age 17	$X^2 (49, 149) = 56.170, p = .224$		
130 and above	0	0	
120 – 129	4 (2)	6 (4)	
110 – 119	10 (6)	17 (10)	
90 – 109	36 (22)	44 (27)	
80 – 89	14 (8)	9 (5)	
70 - 79	3 (2)	6 (4)	

Appendix K

continued

Variable	M	F	Analysis
	M (SD)		
IQ	99.87 (12.68)	100.771 (13.14)	$F(1, 148) = .156, p = .693$
Age	23.33 (1.022)	23.07 (.998)	$F(1,165) = 2.751, p = .099$

Appendix L

Coping Styles (CRI -A) for Perinatal Group by Gender

	FT		HPT		MPT		NPT		SMG	
	M	F	M	F	M	F	M	F	M	F
<i>n</i>	17	19	10	14	25	21	16	9	4	20
Two – way ANOVA										

APPROACH

Logical analysis: $F(4, 145) = .886, p = .486$

<i>M</i>	48.76	55.95	47.10	49.36	51.48	54.52	45.94	47.22	53.75	50.15
<i>SD</i>	13.07	6.05	13.07	11.55	9.94	8.47	9.03	9.58	2.50	8.11

Positive Reappraisal: $F(4, 145) = .865, p = .487$

<i>M</i>	49.29	55.16	52.60	55.71	54.16	55.43	49.31	52.00	55.00	51.30
<i>SD</i>	10.55	8.42	10.22	6.75	6.80	9.13	7.24	8.87	6.98	8.95

Appendix L

continued.

	FT		HPT		MPT		NPT		SMG	
	M	F	M	F	M	F	M	F	M	F
Two – way ANOVA										
Seeking Guidance & Support: $F(4, 145) = 1.203, p = .312$										
<i>M</i>	50.53	53.68	45.30	52.14	48.36	54.48	44.63	53.67	51.25	49.50
<i>SD</i>	7.94	8.20	10.07	8.01	8.22	7.37	8.58	3.87	5.38	8.35
Problem Solving: $F(4, 145) = .314, p = .868$										
<i>M</i>	49.88	52.26	50.80	53.43	56.16	57.10	47.81	50.44	54.75	51.55
<i>SD</i>	11.21	9.67	10.80	10.59	8.13	8.26	10.69	11.93	10.56	9.58
AVOIDANCE										
Cognitive Avoidance: $F(4, 145) = .523, p = .719$										
<i>M</i>	51.24	57.05	52.40	55.07	53.48	55.43	51.94	54.22	60.00	57.10

Appendix L

continued.

	FT		HPT		MPT		NPT		SMG	
	M	F	M	F	M	F	M	F	M	F
	Two – way ANOVA									
<i>SD</i>	10.32	11.66	7.47	8.95	9.88	9.20	9.20	9.62	2.31	10.37
Acceptance or Resignation: $F(4, 145) = 435, p = .783$										
<i>SD</i>	52.94	55.63	45.70	52.43	51.48	53.52	52.94	53.56	54.50	54.15
<i>MD</i>	8.70	11.54	11.74	7.15	8.18	8.96	12.23	6.39	9.00	8.38
Seeking Alternative Rewards: $F(4, 144) = .978, p = .422$										
<i>M</i>	51.71	52.36	48.10	54.23	54.08	52.19	57.63	54.67	55.75	56.40
<i>SD</i>	7.66	10.50	7.26	8.38	8.33	9.05	8.67	11.16	6.85	9.83

Appendix L

continued.

	FT		HPT		MPT		NPT		SMG	
	M	F	M	F	M	F	M	F	M	F
Two – way ANOVA										
Emotional Discharge: $F(4, 145) = .843, p = .500$										
<i>M</i>	55.76	60.68	51.60	57.33	57.32	58.67	51.75	61.44	57.75	57.90
<i>SD</i>	10.18	10.42	9.68	6.60	11.70	9.97	9.26	12.03	6.19	9.59

Appendix M

ANOVAS and MANOVAS

Stress

A one-way ANOVA, excluding missing values case-by-case analysis with α of $p = 0.05$, was used to determine any mean differences between the IV of perinatal birth group and DV of DHS-R. Levene's test was not statistically significant. The ANOVA determined no statistically significant effects between perinatal birth groups in DHS-R scores, $F(4, 158) = 1.384, p = .242$. See Tables 31 and 32 for further details.

A one-way ANOVA, excluding missing values case-by-case analysis with α of $p = 0.05$, was used to determine any mean differences between the IV of gender and DV of DHS-R. Levene's test was not statistically significant. The ANOVA determined no statistically significant effects between genders in DHS-R scores, $F(1, 161) = .543, p = .462$. See Tables 35 and 36 for further details.

A two-way ANOVA, excluding missing values case-by-case analysis with α of $p = 0.05$, was used to determine any mean differences between the IV of perinatal birth group with gender and DV of DHS-R. Levene's test was not statistically significant. The ANOVA determined no statistically significant effects between perinatal birth group with gender in DHS-R scores, $F(4, 153) = .794, p = .531$ with an intercept of $p = > .001$. See Tables 35, 36, 37, 38 and 39 for further details.

An ANCOVA, with α of $p = 0.05$, was used to determine any gender differences in DHS-R scores, after controlling for the effects of SES. Levene's test was not statistically significant. The ANCOVA determined no statistically significant

Appendix M

continued

effect of SES on gender and DHS-R, $F(1, 153) = .843, p = .360$, with an intercept of $p = < .001$. See Tables 35, 36, 37, 38 and 39 for further details.

Coping

A one-way MANOVA with an α of $p = 0.05$, and was used to determine if there were any effects of prematurity on avoidance coping styles. The IV was perinatal birth group and the DV's were cognitive avoidance, positive appraisal, acceptance or resignation, seeking alternative rewards and emotional discharge. All Levene's, Box's and Tukey's tests were not statistically significant.

No statistically significant differences were found at the macro level: Wilk's $\Lambda = .891 [F(16, 447) = 1.079], p = .372$, partial $\eta^2 = .028$ with an intercept of $> .001$. ANOVA's determined no statistically significant differences across perinatal birth groups for cognitive avoidance, positive appraisal, seeking alternative rewards and emotional discharge. See Table 46 for means and standard deviations.

A two-way MANOVA with an α of $p = 0.05$, and was used to determine if there were any effects of perinatal birth group with gender on avoidance coping styles. The Levene's test was significant at $p = .034$ for acceptance or resignation. Box's tests were not statistically significant.

Perinatal birth term with gender was not statistically significant at the macro level for effects on approach coping styles: Wilk's $\Lambda = .920 [F(16, 431) = .774], p = .749$, partial $\eta^2 = .021$, with an intercept of $p = < .001$. ANOVAs were not statistically significant for all avoidance coping styles. See Table 51 for descriptive details.

Appendix M

continued

Tukey's Post-hoc multiple comparisons were not statistically significant.

Emotional Intelligence

A one-way ANOVA, excluding missing values case-by-case analysis with α of $p = 0.05$, were used to determine any mean differences between the IV of perinatal birth groups and DV of emotional intelligence (EQi). Levene's test was not statistically significant. The one-way ANOVA determined no statistically significant effects between perinatal birth groups across EQi scores, $F(4, 153) = .285$, $p = .887$. See Tables 52 and 53 for further details.

A one-way ANOVA, excluding missing values case-by-case analysis with α of $p = 0.05$, were used to determine any mean differences between the IV of gender and DV of EQi. Levene's test was not statistically significant. The one-way ANOVA determined no statistically significant effects between genders in EQi scores, $F(1, 156) = .427$, $p = .517$. See Tables 56 and 57 for further details.

A two-way ANOVA, excluding missing values case-by-case analysis with α of $p = 0.05$, was used to determine any mean differences between the IVs of perinatal birth group with gender and DV EQi. Levene's test was not statistically significant. The two-way ANOVA determined no statistically significant effects between perinatal birth groups with gender in EQi scores, $F(4, 148) = .977$, $p = .422$ with an intercept of $p = > .001$. See Table 60 for further details.

A one-way ANOVA, excluding missing values case-by-case analysis with α of $p = 0.05$, was used to determine any mean differences between the IV of

Appendix M

continued

perinatal birth groups and DV of emotional health (ASR) t-scores. Levene's test was not statistically significant. The one-way ANOVA determined no statistically significant effects between perinatal birth groups in ASR, $F(4, 158) = .815$, $p = .517$. See Tables 63 and 64 for further details.

A one-way ANOVA, with α of $p = 0.05$, was used to determine any mean differences between the IV of gender and DV of ASR. Levene's test was not statistically significant. The ANOVA determined no statistically significant effects between genders in ASR, : $F(1, 161) = .012$, $p = .911$.

A two-way ANOVA was used to determine any differences between perinatal birth groups with gender on ASR scores. No statistically significant differences were found between perinatal birth groups with gender on ASR scores, $F(4, 153) = .825$, $p = .511$, with an intercept of $p = < .001$. See Table 68 for descriptive.

Bibliography

Achenbach, T. (2003). *Manual for the adult self-report for ages 18-59*. Burlington, VT.: University of Vermont, Department of Psychiatry.

Achenbach, T. (2011). *ASEBA: Achenbach system of empirically based assessment*. Retrieved March 6, 2011 from <http://www.aseba.org/adults.html>

Achenbach, T., & Rescorla, L. (2003). *Manual for the ASEBA adult forms & profiles*. Burlington, VT: University of Vermont, Department of Psychiatry.

Achenbach, T. M., Rescorla, L. A., & Maruish, M. E. (2004). The Achenbach system of empirically based assessment (ASEBA) for ages 1.5 to 18 years. *Maruish ME. The use of psychological testing for treatment planning and outcomes assessment*, 2, 179-213.

Acuna, L. Garcia, A., & Bruner, C. (2012). The social readjustment rating scale of Holmes and Rahe in Mexico: a rescaling after 16 years. *Revista Mexicana De Psicología*, 29(1), 16-32. Retrieved February 22, 2015 from researchgate.net [PDF]

Adams, J., & Weakliem, D., (2011). August B. Hollingshead's "Four factor index of social status": from unpublished paper to citation classic. *Yale Journal of Sociology*, 8(Fall). Retrieved September 23, 2011 from http://elsinore.cis.yale.edu/sociology/yjs/yjs_fall_2011.pdf

Akerjordet, K., & Severinsson, E. (2007). Emotional intelligence: A review of the literature with specific focus on empirical and epistemological perspectives. *Journal of Clinical Nursing*, 1405-1416. doi: 10.1111/j.1365-2702.2006.01749.x

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

Aldwin, C. (2009). *Stress, coping and development: an integrative perspective* (2nd Ed.) New York, NY: The Guilford Press.

- Allen, M. C., Cristofalo, E. A., & Kim, C. (2011). Outcomes of preterm infants: morbidity replaces mortality. *Clinics in Perinatology*, 38(3), 441-454.
- Altemus, M. (2006). Sex differences in depression and anxiety disorders: Potential biological determinants. *Hormones and Behavior*, 50, 534-538.
- Almlund, M., Duckworth, A. L., Heckman, J. J., & Kautz, T. D. (2011). *Personality psychology and economics* (No. w16822). National Bureau of Economic Research.
- Alvarez, J., & Emory, E. (2006). Executive function and the frontal lobes: A meta-analytic Review. *Neuropsychology Review*, 16(1), 17-41.
doi: 10.1007/s11065-006-9002-x
- American Institute of Stress (AIS). (2016). Holmes – Rahe Stress Inventory. Retrieved January 22, 2016 from <http://www.stress.org/holmes-rahe-stressinventory/>
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, fourth edition, text revision, DSM-IV-TR*. American Psychiatric Association: Washington, DC.
- American Psychological Association (APA). (2017). Data on behavioral health in the United States: access to treatment. Retrieved March 31, 2017 from www.apa.org/helpcenter/data-behavioral-health.aspx
- Armstrong, A., Galligan, R., & Critchley, C. (2011). Emotional intelligence and psychological resilience to negative life events. *Personality and Individual Differences*, 51, 331-336. doi: 10.1016/j.paid.2011.03.025
- Anshel, M. H., Sutarso, T., & Sozen, D. (2012). Relationship between cognitive appraisal and coping style following acute stress among male and female Turkish athletes. *International Journal of Sport & Exercise Psychology*, 10(4), 290 – 304.
- Anxiety Disorders Association of America. (2007). Statistics and facts about anxiety disorders. Retrieved April 23, 2016 from <http://www.adaa.org/aboutadaa>

- American Academy of Pediatrics. (2012). Policy statement: Early childhood adversity, toxic stress, and the role of the pediatrician: Translating developmental Science into lifelong health. *Pediatrics*, 129(1), 224-231
- American Psychological Association (APA). (2010). Gender and Stress. Retrieved September 28, 2016 from <http://www.apa.org/news/press/releases/stress/2010/gender-stress.aspx>
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders*: DSM-5. Washington, D.C: American Psychiatric Association.
- Arnett, Jeffery, J. (2000). Emerging adulthood: a theory of development from the late teens through the twenties. *American Psychologist*, 55(5), 469-480.
doi: 10.1037//0003-066X.55.5.469
- Arnett, J. (2017). *Suffering, selfish, slackers? Myths and reality about emerging adults*. Boston College Resources, Spring, C21, 8-9.
- Arnett, J. (2013). *Adolescence and emerging adulthood: A cultural approach* (5th ed.). Upper Saddle River, NJ: Pearson Edu. Inc.
- Arnold, E., & Kverno, K. (2009). *Psychiatric-Mental Health Nursing*. Silver Spring, MD: American Nurses Credentialing Center.
- ASEBA. (2003). Adult Self - Report for Ages 18 – 59. University of Vermont: Burlington, VT.
- ASEBA. (2011). *Manual for the ASEBA adult forms & profiles*. University of Vermont: Burlington, VT.
- ASEBA (Archenbach System of Empirically Based Assessment). (2016). Adult (Ages 18-59) Assessments. Retrieved February 14, 2016 from: <http://www.aseba.org/adults.html#scales>

- Ashkanasy, N. M., Ashton-James, C. E., & Jordan, P. J. (2004). Performance impacts of appraisal and coping with stress in workplace settings: The role of affect and emotional intelligence. *Research in occupational stress and wellbeing*, 3, 1-43.
- Atzil, S., Hendler, T., & Feldman, R. (2011). Specifying the neurobiological basis of human attachment: brain, hormones, and behaviors in synchronous and intrusive mothers. *Neuropsychopharmacology*, 36, 2603-2615.
doi: 10.1038/npp.2011.172
- Baber, N., Muzaffer, R., Khan, M., & Imdad, S. (2010). Impact of socioeconomic factors on nutritional status in primary school children. *Journal of Ayub Medical College Abbottabad*, 22(4): 15-18. Retrieved March 23, 2015 from <http://www.ncbi.nlm.nih.gov/pubmed/22455252>
- Banerjee, A., Chitnis, U. B., Jadhav, S. L., Bhawalkar, J. S., & Chaudhury, S. (2009). Hypothesis testing, type I and type II errors. *Industrial Psychiatry Journal*, 18(2), 127–131. <http://doi.org/10.4103/0972-6748.62274>
- Banga, G., Barche, A., Singh, R., Sheehan, C., & Vasylyeva, T. (2015). Sex-related health disparities among preterm babies. *International Journal of Integrative Pediatrics and Environmental Medicine*, 1-6. doi: 10.6000/ijipem/2015/1
- Bannon, W. (2013). *The 7 steps of data analysis: a manual for conducting a quantitative research study*. New York, NY: StatsWhisperer Press.
- Barker, D. (1990). The fetal and infants origins of adult disease. *British Medical Journal*, 301(6761), 1111.
- Barker, D. (1995). Fetal origins of coronary heart disease. *British Medical Journal*, 311, 171-174.
- Barker, D. (2004). Developmental origins of adult health and disease. *Journal of Epidemiological Community Health*, 58, 114-115

- Barker, D. (2005). The developmental origins of insulin resistance. *Hormone Research*, 64(suppl 3), 2-7. doi: 10.1159/000089311
- Barker, D. (2007). The origins of the developmental origins theory. *Journal of Internal Medicine*, 261, 412-417. doi: 10.1111/j.1365-2796.2007.01809.x
- Barker, D., Eriksson, Forsen, T., & Osmond, C. (2002). Fetal origins of adult disease: strength of effects and biological basis. *International Journal of Epidemiology*, 31, 1235-1239.
- Barker, D., & Osmond, C. (1986). Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *The Lancet*, 1077-1082.
- Barker, D., & Thornburg, K. (2013). The obstetric origins of health for a lifetime. *Clinical Obstetrics and Gynecology*, 56(3), 511-519.
- Baron, R & Kenny, D. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, 51, 1173-1182
- Bar-On, R. (1997). The emotional quotient inventory (EQ-i): A test of emotional intelligence. *Toronto: Multi-Health Systems*, 28.
- Bar-On, R. (2002), Bar-On emotional quotient inventory: Users's manual. New York: Multi-health System, Inc.
- Bar-On, R. (2006). The Bar-On model of emotional-social intelligence (ESI). *Psicothema*, 18(supl.), 13-25. Retrieved February 24, 2015 from www.psicothema.com
- Bar-On, R., & Parker, J. (Ed.). (2000). *The handbook of emotional intelligence: theory, development, assessment, and application at home, school, and in the workplace*. San Francisco, CA: Jossey-Bass.

- Bar-On, R., Tranel, D., Denberg, N., & Bechara, A. (2003). Exploring the neurological substrate of emotional and social intelligence. *Brain*, 126, 1790-1800.
doi: 10.1093/brain/awg177
- Barreto, M., & Frazier, L. (2012). Coping with life events through possible selves. *Journal of Applied Social Psychology*, 42(7), 1785-1810.
- Behrman, R., & Butler, A. (Ed.). (2007). Premature birth: causes, consequences & prevention, *Institute of medicine (US) committee on understanding premature birth and assuring healthy outcomes*. Washington, DC: National Academies Press. Retrieved February 28, 2015 from National Center for Biotechnology Information (NCBI), www.ncbi.nlm.nih.gov. bookshelf ID: NBK11356
- Ben – Sholmo, Y., & Kuh, D. (2002). A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. *International Journal of Epidemiology*. 31, 285-293.
- Benson, P., Scales, P., Hawkins, J., Oesterle, S., & Hill, K. (2004). Executive summary: Successful young adult development. A report submitted to: The Bill & Melinda Gates Foundation. Retrieved February 2, 2016 from <https://docs.gatesfoundation.org/.../Succ>.
- Bibb, Sandra. (2007). Issues associated with secondary analysis of population health data. *Applied Nursing Research*, 20, 94-99. doi: 10.1016/j.apnr.2006.02.003
- Blalock, J., & Joiner, T. (2000). Interaction of cognitive avoidance coping and stress in predicting depression/anxiety. *Cognitive therapy and research*, 24(1), 47-65
- Brackett, M., Mayer, J., & Warner, R. (2004). Emotional intelligence and its relationship to everyday behavior. *Personality and Individual Differences*, 36, 1387-1402.
doi: 10.1016/S0191-8869(03)00236-8
- Brakewood, B., & Poldrack, R. (2013). The ethics of secondary data analysis: Considering the application of Belmont principles to the sharing of neuroimaging data. *NeuroImage*, 82, 671-676. doi: 10.1016/j.neurimage.2013.02.040

- Britannia.com (2012). Retrieved October 3, 2015 from <http://www.merriam-webster.com/dictionary/type%20i%20error>
- Brooks-Gunn, J., Phelps, E., & Elder, G. (1991). Studying live through time: Secondary data analyses in developmental psychology. *Developmental Psychology*, 27(6), 899-910.
- Brothwood, M., Wolke, D., Gamsu, H., Benson, J., & Cooper, D. (1986). Prognosis of the very low birthweight baby in relation to gender. *Archives of disease in childhood*, 61(6), 559-564. Retrieved November 29 2015 from <http://adc.bmj.com/>
- Brotman, D. J., Golden, S. H., & Wittstein, I.S. (2007). The cardiovascular toll of stress. *The Lancet*, 370, 1089a1100.
- Bruehl, H., Wolf, O., & Covit, A. (2009). A blunted cortisol awakening response and hippocampal atrophy in type 2 diabetes mellitus. *Psychoendocrinology*, 34(6), 815-821.
- Bruyere, H. (2009). *100 cases studies in pathophysiology*. Philadelphia, PA: Lippincott, Williams & Wilkins.
- Bryan, S. & Hindmarsh, P. (2006). Normal and abnormal fetal growth. *Hormone Research*, 65(suppl.3), 19-27. doi: 10.1159/000091502
- Buchner, A., Erdfelder, Faul & Lang, A-G.. (2014). *G*Power Manual*. Retrieved November 15, 2016 from www.gpower.hhu.de/fileadmin/redaktion/Fakultaeten/.../gpower/GPowerManual.pdf
- Burnett, A., Anderson, P., Cheong, J., Doyle, L., Davey, C., & Wood, J. (2011). Prevalence of psychiatric diagnoses in preterm and full-term children, adolescents and young adults: A meta-analysis. *Psychological Medicine*, 41, 2463-2474. doi: 10.107/S003329171100081X
- Businelle, M., Mills, B., Chartier, K., Reingle, J., & Shuval, R. (2013). Do stressful events account for the link between socioeconomic status and mental health? *Journal*

of Public Health, 36(2), 205-202. doi:10.1093/pubmed/fdt060

- Buske-Kirschbaum, A. S. Krieger, C. Wilkes, W. Rauh, S. Weiss, D. H. Hellhammer. (2007). Hypothalamic-pituitary-adrenal axis function and the cellular immune response in former preterm children. *Journal of Clinical Endocrinology Metablism*, 92(9), 3429-3435. doi: 10.1210/jc.2006-2223
- Butler, J. (1993). Definitions of Stress. *Occasional Paper College General. Practice* (61), 1-5. Retrieved March 3, 2015 from <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2560943/>
- Campbell-Sills, L., Cohan, S., & Stein, M. (2006). Relationship of resilience to personality, coping, and psychiatric symptoms in young adults. *Behavior Research and Therapy*, 44(4), 585-599.
- Cantor, N. (2000). Social intelligence. In R.J. Sternberg, (Ed.), *Handbook of Intelligence* (2nd ed.). Cambridge UK: Cambridge University Press.
- Carpenter, I., Gawuga, C., Tyrka, A., Lee, J., Anderson, G., & Price, L. (2010). Associations between plasma IL-6 response to acute stress and early-adversity in healthy adults. *Neuropsychopharmacology*, 35, 2617-2613. doi: 10.1038/npp.2010.159
- Carver, Charles, S. (2011). Coping. In R. J. Contrada, & A. Baum (eds.), *The handbook of stress science: biology, psychology and health* (pp. 221-229). New York, NY: Springer Publishing Co., LLC.
- Cassidy, A., Drotar, D., Ittenbach, R., Hottinger, S., Wray, J., Wernovsky, G., Newburger, J., Mahony, L., Mussatto, K., Cohen, M., & Marino, B. (2013, March 8). The impact of socio-economic status on health related quality of life for children and adolescents with heart disease. *Health and Quality of Life Outcomes*, 11(99). Retrieved February 22, 2015 from <http://www.hqlo.com/content/11/1/99>
- Castle, Jane. (2003). Maximizing research opportunities: Secondary data analysis. *Journal of Neuroscience Nursing*, 35(5), 287-290.

- Castro, M., Elias, P., Martinelli, C., Antonnini, S., Santiago, I., & Moreira, A. (2000). Salivary cortisol as a tool for physiologic studies and diagnostic strategies. *Brazilian Journal of Medical and Biological Research*, 33, 1171-1175.
- Center for Disease Control and Prevention (CDC). (2013). Health-Related quality of life (HRQL). Retrieved October 20, 2015 from <http://www.cdc.gov/hrqol/wellbeing.htm>
- Center for Disease Control and Prevention (CDC). (2014). *Preterm birth*. Retrieved October 20, 2015 from <http://www.cdc.gov/reproductivehealth/maternalinfanthealth/pretermbirth.htm>
- Centers for Disease Control and Prevention (CDC). (2015). Preterm birth-prematurity-maternal infant health. *Reproductive Health*. Retrieved February 24, 2015 from www.cdc.gov/reproductivehealth
- Cherniss, C., Extein, M., Goleman, D., & Weissberg, W. (2006). Emotional intelligence: What does the research really indicate? *Educational Psychologist*, 41(4), 239-245.
- Child Trend Data Bank, (2011). Low and very low birth weight infants. Retrieved August 14, 2011. <http://www.childtrendsdatabank.org/?q=node/67>.
- Chrousos, G., & Gold, P. (1992). The concepts of stress and stress system disorders: *Overview of physical and behavioral homeostasis*, *Journal of the American Medical Association*, 267(9), 1244-1252.
- Ciarrochi, J., Deane, F., & Anderson, S. (2002). Emotional intelligence moderates the relationship between stress and mental health. *Personality and Individual Differences*, 32, 197-209.
- Cirino, P., Chin, C., Sevcik, R., Wolf, M., Lovett, M., & Morris, R. (2002). Measuring Socioeconomic status. *Assessment*, 9(2), 145-155.
- Clarke, K., Cooper, P., & Creswell, C. (2013). The parental overprotection scale: Associations with child and parental anxiety. *Journal of Affective Disorders*, 15(2), 618-624. Retrieved from <http://dx.doi.org/10.1016/j.jad.2013.07.007>

- Clow, A., Hucklebridge, F., & Thorn, L. (2010). The cortisol awakening response in context. *International Review of Neurobiology*, 93, 153-175.
- Cohen, S., Janicki-Deverts, D., & Miller, G. E. (2007). Psychological stress and disease. *Journal of the American Medical Association*, 298 (14), 1685-1687.
<http://jama.jamanetwork.com>.
- Cohen, S., Kessler, R., & Gordon, L. (1995). Strategies for measuring stress in studies of psychiatric and physical disorders. *Measuring stress: A guide for health and social scientists*, 3-26.
- Compas, B. (2006). Psychobiological processes of stress and coping: implications for resilience in children and adolescents-comments on the papers of Romeo & McEwen and Fisher et al. *Annals New York Academy of Science*, 1094, 226-234.
- Conti, J. (2005). A review and critique of emotional intelligence measures. *Journal of Organizational Behavior*, 26, 433-440. doi: 10.1002/job.319
- Contrada, R., & Baum, A. (Eds.). (2011). *The handbook of stress science: biology, psychology, and health*. New York, NY: Springer Publishing Co.
- Cooke, R. (2004). Health, lifestyle, and quality of life for young adults born very preterm. *Archives of disease in childhood*, 89, 201-206.
doi: 10.1136/adc.2003.030197
- Dahl, R. (2001). Affect regulation, brain development, and behavioral/emotional health in adolescence. *Central Nervous System (CNS) spectrums* 6(01), 60-72.
- Davis, M. C., Burleson, M. H., & Kruszewski, D. M. (2011). Gender: its relationship to Stressor exposure, cognitive appraisal/coping processes, stress response, and health outcomes. In R. J. Contra & A. Baum (Eds). *The handbook of stress science: biology, psychology and health* (pp.248-261). New York, NY: Springer Publishing Co. Inc.

- Davis, S., & Humphery, K. (2012). The influence of emotional intelligence (EI) on coping and mental health in adolescence: divergent roles for trait and ability EI. *Journal of Adolescence*, 35(5), 1369-1379. doi: 10.1016/j.adolescence.2012.05.007
- Dawda, D., & Hart, S. (2000). Assessing emotional intelligence: reliability and validity of the Bar-On Emotional Quotient Inventory (EQ-i) in university students. *Personality and Individual Differences*, 28, 797-812.
- De Boo, H., & Harding, J. (2006). The developmental origins of adult disease (Barker) hypothesis. *Australian and New Zealand Journal of Obstetrics and Gynaecology*, 46, 4-14. Retrieved February 22, 2015 from <http://pedneph.info>
- De Weerd, M., & Rossi, G. (2012). The Bar-On emotional quotient inventory (EQ-i): Evaluation of psychometric aspects in the Dutch speaking part of Belgium, *Psychology - Selected Papers*, Dr. Gina Rossi (Ed.), Shanghai, China: InTech. Retrieved January 3, 2015 from: <http://www.intechopen.com/books/psychologyselected-papers/the-bar-on-emotional-quotient-inventory-eq-i-evaluation-of-psychometric-aspects-in-a-flemish-populat>
- De Weerth, C., Zijl, R.H., & Buitelaar, J. K. (2003). Development of cortisol circadian rhythm in infancy. *Early Human Development*, 73, 39-52. doi:10.1016/S0378-3782(03)00074-4.
- Dickerson, S., & Kemeny, M. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, 130(3), 355-391. doi: 10.1037/0033-2909.130.3.355
- Di Fabio, A., & Sakloske, D. (2014). Promoting individual resources: The challenge of trait emotional intelligence. *Personality and Individual Differences*, 65, 19-23. Retrieved from <http://dx.doi.org/10.1016/j.paid.2014.01.026>
- Dohrenwend, B. S., Krasnoff, L., Askenasy, A., & Dohrenwend, B. P. (1978). Exemplification of a method for scaling life events: The peri life events scale. *Journal of Health and Social Behavior*, 19 (June): 205-229.

- Donnellan, C., Hevey, D., Hickey, A., & O'Neill, D. (2006). Defining and quantifying coping after a stroke. *Journal of Neurology and Neurosurgical Psychiatry*. Retrieved March 22, 2015 from <http://jnnp.bmj.com>
- Donofrio, B., Class, Q., Lahey, B., & Larsson, H. (2014). Testing the developmental origins of health and disease hypothesis for psychopathology using family-based, quasi-experimental designs. *Child Development Perspectives*, 8(3), 151-157. doi: 10.1111/cdep.12078
- Dorn, L.D., Lucke, J.F., Loucks & Berga, S.L. (2007). Salivary cortisol reflects serum cortisol: analysis of circadian profiles. *Annals of Clinical Biochemistry*, 44, 281-284.
- Dougherty, D., & Conway, P. (2008). The “3t’s” road map to transform US health care: the “how” of high-quality care. *Journal of the American Medical Association*, 299(19), 2319-2321.
- Dowd, J., Simanek, A., & Aiello, A. (2009). Socio-economic status, cortisol and allostatic load: a review of literature. *International Journal of Epidemiology*, 39, 1297-1309. doi: 10.1093/ije/dyp277
- Drake, A., Tang, J., & Nyirenda, M. (2007). Mechanisms underlying the role of glucocorticoids in the early life programming of adult disease. *Clinical Science*, 113, 219-232. doi: 10.1042/CS20070107
- Durm, M., & Glaze, P. (2002). Sex, perception of immediate stress, and response to coping resource inventory, emotional domain. *Psychological Reports*, 90, 270-272
- Dusek, J. A., Otu, H. H., Wohlhueter, A. L., Bhasin, M., Zerbini, L. F., Joseph, M. G., ... & Libermann, T. A. (2008). Genomic counter-stress changes induced by the relaxation response. *PloS one*, 3(7), e2576.
- Edier, R. D., Veira, Y., & Granger, D. A. (2009). Prenatal cocaine exposure and infant cortisol reactivity. *Child Development*, 80(2), 528-543.

- Edmunds, M.W., & Mayhew, M.S. (2009). *Pharmacology for the primary care provider*. St. Louis, Missouri: Mosby Elsevier.
- Eiland, L., & McEwen, B. (2012). Early life stress followed by subsequent adult chronic stress potentiates anxiety and blunts hippocampal structural remodeling. *Hippocampus*, 22, 82-91. doi: 10.1002/hipo.20862
- Engert, V., Efanov, S.I., Dedovic, K., Duchesne, A., Dagher, A., & Pruessner, J.C. (2010). Perceived early-life maternal care and the cortisol response to repeated psychosocial stress. *Journal of Psychiatry and Neuroscience*, 35(6), 370-377
- Epel ES, Blackburn EH, Lin J, Dhabhar FS, Adler NE, Morrow JD, & Cawthon RM. (2004) Proceedings of the National Academy of Sciences of the United States of America. 2004 Dec 7; 101(49): 17312-5. Epub 2004 Dec 1. Accelerated telomere shortening in response to life stress. *101*(49), 17312-17315
- Epstein, H. (1986). Stages in human brain development. *Developmental Brain Research*, 30(1), 114-119. doi: 100-1016/0165-3806
- Ericksson, J. (2006). Patterns of growth: relevance to developmental origins of health and disease. In P. Gluckman & M. Hanson (Eds.), *Developmental Origins of Health and Disease* (pp.223-232). New York, NY: Cambridge University Press.
- Eriksson, J., Forsen, T., Tuomilehto, J., Osmond, C., & Barker, D. (2001). Early growth and coronary heart disease in later life: longitudinal study. *British Medical Journal*, 332(21), 949-953.
- Everly, G. & Benson, H. (1989). Disorders of arousal and the relaxation response: speculations on the nature and treatment of stress-related diseases. *International Journal of Psychosomatics*, 36(1-4), 15-21.
- Everly, J., & Lating, J. (2013). A clinical guide to the treatment of the human stress response. New York, NY: Springer. doi: 10.1007/978-1-4614-5538-7_2
- Farb, N., Anderson, A., Mayberg, H., Bean, J., McKeon, D & Segal, Z. (2010). Minding one's emotions: training alters the neural expression of sadness,

Emotions, 36(1-4), 15-21.doi: 10.1037/a0017151

- Farrington, D. (1991). Longitudinal research strategies: advantages, problems, and prospects. *Journal of the American Academy of Child and Adolescent Psychiatry*, 30(3), 369-374.
- Faul, F., Erdfelder, E., Buchner, A., & Lang, A.-G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods*, 41, 1149-1160.
- Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, 39, 175-191.
- Feder, A., Nestler, E. J., & Charney, D. S. (2009). Psychobiology and molecular genetics of resilience. *Nature Reviews Neuroscience*, 10(6), 446-457.
- Fekedulegn, D. B., Andrew, M. E., Burchfiel, C. M., Violanti, J. M., Hartley, T. A., Charles, L. E. and Miller, D. B. (2007). Area under the curve and other summary indicators of repeated waking cortisol measurements. *Psychosomatic Medicine*, 69, 651-659.
- Felder, Dorine. (2003). *Schematic overview of the hypothalamic-pituitary-adrenal (HPA) axis. Stress activates the HPA-axis and thereby enhances the secretion of glucocorticoids from the adrenals*. CC BY-SA 4.0. Retrieved December 15, 2016 from <http://creativecommons.org/licenses/by-sa/4.0> , via Wikimedia Commons.
- Feldman, R., Weller, A., & Sirota, L. (2002). *Skin-to-skin contact (kangaroo care) promotes self-regulation in premature infants: sleep-wake cyclicality, arousal modulation, and sustained exploration*. *Developmental Psychology*, 38(2), 194-207. doi: 10.1037//0012-1649.38.2.194.
- Fernandez-Berrocal, P., & Extremera, N. (2006). Emotional intelligence: A theoretical and empirical review of its first 15 years of history. *Psicothema*, 18(S), 7-12.
- Ficchione, G. (2015). Mind body resiliency: reducing allostatic loading and

enhancing health. *Stress and relaxation response: the fundamentals of mind body medicine*. Boston, MA: MGH Psychiatry Academy.

Folkman, S., & Moskowitz, T. (2004). Coping: pitfalls and promise. *Annual Reviews of Psychology*, 55, 745-774. doi: 10-1146/annualrev.psych.55.090902.141456

Freedman, J. (2010). White paper: The business case for emotional intelligence. Retrieved April 5, 2016 from www.6seconds.org

Freshwater, D., & Stickley, T. (2004). *Nursing Inquiry*, 11, 91-98.

Fries, F. E., Dettenborn, L. & Kirschbaum, C. (2009). The cortisol awakening response (CAR): facts and future directions. *International Journal of Psychophysiology*, 72(1), 67-73.

Frodi, T., & O'Keane, V. (2013). How does the brain deal with cumulative stress? A review with focus on developmental stress, HPA axis function and Hippocampal structure in humans. *Neurobiology of Disease*, 52, 24-37. doi: 10.1016/j.nbd.2012.03.012

Frye, A., & Liem, J. (2011). Diverse patterns in the development of depressive symptoms among emerging adults. *Journal of Adolescent Research*, 26(5), 570-590.

Furr, R., & Bacharach, V. (2014). *Psychometrics: an introduction (2nd ed.)*. Thousand Oaks, CA: Sage Pub., Inc.

Ganzel, B., Morris, P., & Wethington E. (2010). Allostasis and the human brain: integrating models of stress from the social and life sciences. *Psychology Review*, 117(1), 134-174. doi: 10.1037/a00177773

Garber J., Clarke G., Weersing V., Beardslee WR., Brent DA., Gladstone TR., DeBar L., Lynch F., D'Angelo E., Hollon S., Shamseddeen W., Iyengar S. (2009). Prevention of depression in at-risk adolescents: a randomized controlled trial. *Journal of the American Medical Association*, 21,2215-24. doi: 10.1001/jama.2009.788.

- Garner, A., Shonkoff, J., Siegel, B., Dobbins, M., Earls, M., Garner, A., McGuinn, L., Pascoe, J., & Wood, D. (2012). From the American Academy of Pediatrics policy statement early childhood adversity, toxic stress, and the role of the pediatrician: translating developmental science into lifelong health, *Pediatrics*, 129(1), 2252-2259.
- Gardner, K., Qualter, P., & Whitely, H. (2011). Developmental correlates of emotional intelligence; temperament, family environment and childhood trauma. *Australian Journal of Psychology*, 63(2), 75-82. doi: 10.1111/ajpy.2011.63.issue-2issueroc
- Garson, D. (2015). *North Carolina State University, missing values analysis & data imputation*. Asheboro, NC: Statistical Publishing Associates. Retrieved November 22, 2016 from http://www.statisticalassociates.com/missingvaluesanalysis_p.pdf
- Georgetown University Center for Child and Human Development (GUCCHD). (2016, January). *Introduction to social and emotional health: definition and research support*. Retrieved February, 3, 2016 from: http://ecmhc.org/tutorials/social-emotional/mod1_0.html
- Geritis, L., Derksen, J., Verbruggen, & Katzko, M. (2005). Emotional intelligence profiles of nurses caring for people with severe behaviour problems. *Personality and Individual Differences*, 38(1), 33-43. doi: 10.1016/j.paid.2004.03.019.
- Ghasemi, A., & Zahediasl, S. (2012). Normality Tests for Statistical Analysis: A Guide for Non-Statisticians. *International Journal of Endocrinology and Metabolism*, 10(2), 486–489. Retrieved November 28, 2016 from <http://doi.org/10.5812/ijem.3505>
- Gibson, A., Carney, S, & Wales, J. (2006). Growth and the premature baby. *Hormone Research*, 65(suppl 3), 75-81. doi: 10.1159/000091510.
- Glanz, K., & Schwartz, M. (2008). Stress, coping, and health behavior, In Karen Glanz, Barbara Rimer, & K., Viswanath (Eds.), *Health behavior and health education: theory, research, and practice (4th ed.)*, Chapter 10, p 211-235. San Francisco: CA: Jossey-Bass.

- Gluckman, P., & Hanson, M. (Eds.). (2007). *Developmental origins of health and disease*. New York, NY: Cambridge University Press.
- Godfrey, K. (2006). The 'developmental origins' hypothesis: epidemiology. In P. Gluckman & M. Hanson (Eds.), *Developmental Origins of Health and Disease* (pp. 6-32). New York, NY: Cambridge University Press
- Gohm, C. L., Corser, G. C., & Dalsky, D. J. (2005). Emotional intelligence under stress: Useful, unnecessary, or irrelevant? *Personality and Individual Differences*, 39(6), 1017-1028.
- Goldin, J., & Gross, J. (2010). Effects of mindfulness-based stress reduction (MBSR) on emotion regulation in social anxiety disorder. *Emotion*, 10(1), 83-91.
- Goldenberg, R. L., Culhane, J. F., Iams, J. D., & Romero, R. (2008). Epidemiology and causes of preterm birth. *The Lancet*, 371(9606), 75-84.
- Goleman, Daniel. (2014) Emotional intelligence, social intelligence, ecological Intelligence. Retrieved March 18, 2016 from <http://www.danielgoleman.info>
- Goleman, D., Boyatzis, R., & Hay Group. (2005). EI Assessments. Retrieved November 13, 2016 from <http://www.danielgoleman.info/ei-assessments/>
- Gooding, P. A., Hurst, A., Johnson, J., & Tarrier, N. (2012). Psychological resilience in young and older adults. *International journal of geriatric psychiatry*, 27(3), 262-270.
- Gordis, L. (2009). *Epidemiology*. Philadelphia, PA: Saunders.
- Gottfried, A. (1985). Measures of socioeconomic status in child development research: data and recommendations. *Merrill-Palmer Quarterly*, 31(1), 85-92. Retrieved October 15, 2015 from <http://www.jstor.org/stable/2308613610.2307/23086136>
- Granger D., & Kivlighan, K. (2003). Integrating biological, behavioral, and social levels of analysis in early child development: progress, problems, and prospects.

Child Development, 74(4), 1058-1063. 009-3920/2003/7404-0010

Grunau, R. (2002). Early pain in preterm infants: A model of long-term effects. *Clinics in Perinatology*, 29, 373-394.

Grunau, R. (2013). Neonatal pain in very preterm infants: long-term effects on brain, neurodevelopment and pain reactivity. *Rambam Maimonides Medical Journal*, 4(4), e0025. Retrieved September 28, 2017 from www.rmmj.org.il

Gujral, H. (2013). Emotional intelligence buffers stress: A study on emotional intelligence and coping styles. *Excel International Journal of Multidisciplinary Management Studies*, 3(11), 76-82. Retrieved December 10, 2016 from <http://www.researchgate.net/publication/281630073>

Gunner, M., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology*, 27, 199-220.

Gunner, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, 58, 145-173. doi: 10.1146/annurevpsych.58.110405.085605

Gunner, M., & Quevedo, K. (2008). Early care experiences and HPA axis regulation in children: a mechanism for later trauma vulnerability. *Progress in Brain Research*, 167, 137-149. doi: 10.1016/S0079-6123(07)67010-1

Gutman, D. A., & Nemeroff, C. B. (2011). Stress and depression. *The handbook of stress science: Biology, psychology, and health*, 345-357.

Hack, M. (2009). Adult outcomes of preterm children. *Journal of Developmental & Behavioral Pediatrics*, 30(5), 460-470.

Hack, M., Flannery, D., Schuchter, M., Cartar, L., Borawski, E., & Klien, N. (2002). Outcomes in young adulthood for very-low-birth-weight infants. *The New England Journal of Medicine*, 346(3), 149-157. Retrieved March 24, 2015 from www.nejm.org

- Hack, M., Taylor, H. G., Drotar, D., Schluchter, M., Cartar, L., Andreias, L., ... & Klein, N. (2005). Chronic conditions, functional limitations, and special health care needs of school-aged children born with extremely low-birth-weight in the 1990s. *Journal of the American Medical Association*, 294(3), 318-325.
- Hack, M., Friedman, M., & Fanaroff, A. (1996) Outcomes of extremely low birth weight infants. *Pediatrics*, 98, 931-937.
- Halter, M.J., & Varcarolis, E.M. (2010). Understanding responses to stress. In Varcarolis, E.M., & Halter, M.J. (Eds.), *Foundations of Psychiatric Mental Health Nursing*, (pp.195-209). St. Louis, Missouri: Sanders/Elsevier.
- Harlow, L. (2014). *The essence of multivariate thinking: basic themes and methods*. New York, NY: Routledge
- Harpe, S.E. (2009). Using secondary data sources for pharmacoepidemiology and outcomes research. *Pharmacotherapy*, 29(2), 138-152.
- Harvey, A., Nathans, A., Bandiera, G., & Leblance, V. (2010). Threat and challenge: cognitive appraisal and stress responses in simulated trauma resuscitations. *Medical Education*, 44, 587-594. doi: 10.1111/j.1365-2923.2010.03623.x
- Healy, E. (2010). Behavioral outcome of preterm birth in childhood and adolescence. In C. Nosarti, R. Murray & M. Hack (Eds.), *Neurodevelopmental Outcomes of Preterm Birth: From Childhood to Adult Life* (pp. 113-126). New York, NY: Cambridge University Press.
- Heejung, K., & Sasaki, J. (2014). Cultural neuroscience: biology of the mind in cultural contexts. *Annual Review of Psychology*, 65, 487-514. doi: 10.1146/annurev-psych-010213-115040
- Heindel, J., & Vandenberg, L. (2015). Developmental origins of health and disease: a paradigm for understanding disease cause and prevention. *Therapeutics and Toxicology*, 27(2), 248-253. doi: 10.1097/MOP.0000000000000191

- Helhammer, D., Wust, S., & Kudielka, B. (2009). Salivary cortisol as a biomarker in stress research. *Psychoneuroendocrinology*, 34, 163-171.
doi: 10.1016/j.psyneuen.2008.10.026
- Herman, J., Ostrander, M., Mueller, N., & Figueiredo, H. (2005). Limbic system mechanisms of stress regulation: Hypothalamic-pituitary-adrenocortical axis. *Progress In Neuro-Psychopharmacology & Biological Psychiatry*, 29, 1201-1213. doi: 10.1016/j.pnpbp.2005.08.006
- Hertel, J., Schutz, A., & Lammers, C-H. (2009). Emotional intelligence and mental disorder. *Journal of Clinical Psychology*, 65, 942-954. doi: 10.1002/jclp.20597
- Hickey, J. & Sullivan, M. (2012). [Anxiety, psychiatric disorders and diurnal salivary cortisol levels]. Major paper in partial fulfillment of MS degree. University of Rhode Island.
- Hobel, C., Hyvarinen, M., Okada, D., & Oh, w. (1973). Prenatal and intrapartum high-risk screening. *American Journal of Obstetrics and Gynecology*, 117(1), 1-9.
- Hofman, P., Regan, F., & Cutfield, W. (2006). Prematurity-another example of perinatal metabolic programming? *Hormone Research In Pediatrics*, 66(1), 33-39.
doi: 10.1159/000093230
- Hoffman, J., Benson, H., Arns, P., Stainbrook, G., Landsberg, L., Young, J., & Gill, A. (1982). Reduced sympathetic nervous system responsivity associated with the relaxation response, *Science*, 215, 190-200.
- Holahan, C., J., Moos, R. H., Holahan, C. K., Brennan, P. L., & Schutte, K. K. (2005). Stress generation, avoidance coping, and depressive symptoms: a 10-year model. *Journal of Consulting and Clinical Psychology*, 73(4), 658-666.
doi:10.1037/0022-006X.73.4.658
- Hollingshead, A. B. (1975). *Four factor index of social status*. New Haven, CT: Yale University Press.

- Hollingshead, A. (1977). *Four factor index of social status*. Unpublished Manuscript, Yale University, New Haven, Conn.
- Hollingshead, A. (2011, Fall). Four factor index of social status. *Yale Journal of Sociology*, 8(Fall). Retrieved October 20, 2015 from http://elsinore.cis.yale.edu/sociology/yjs/yjs_fall_2011.pdf
- Holm, J., & Holroyd, K. (1992). The daily hassles scale (revised): Does it measure stress or symptoms? *Behavioral Assessment*, 14, 465-482.
- Holmes, T., & Rahe, R. (1967). The social readjustment rating scale. *Journal of Psychosomatic Research*, 11(2), 213-218. doi: 10.1016/0022-3999(67)90010-4
- Howell, D., C. (2016). *Outline of StatPages - University of Vermont*, August 22, 2016. Retrieved November 6, 2016 from <https://www.uvm.edu/~dhowell/StatPages/>,
- Hulme, P.A., French, J.A., & Agrawal, S. (2011). Changes in diurnal salivary cortisol levels in response to an acute stressor in healthy young adults. *Journal of the American Psychiatric Nursing Association*, 17(5), 350-355. doi: 10.1177/1078390311419352.
- Hultman, C.M. & Nosarti, C. (2010). Preterm birth and fetal growth in relation to adult psychopathology. In C. Nosarti, R.M. Murray, & M. Hack (Eds.), *Neurodevelopmental outcomes of preterm birth from childhood to adult life* (pp.127-139). Cambridge,UK: Cambridge University Press.
- Hutchinson, A-M., Stuart, A., & Pretorius, H. (2007). Coping with stressors in late adolescence/young adulthood: a salutogenic perspective. *Health Sa Gesondheid*, 12(3), 37-45.
- Ingels, S., Glennie, E., Lauff, E., & Wirt, J. (2012). *Trends among young adults over three decades, 1974-2006*(NCES 2012-345). U.S. Department of Education, National Center for Education Statistics. Washington, DC. Retrieved from <http://nces.ed.gov/pubsearch>

- Ingemarsson, I. (2003), Gender aspects of preterm birth. *BJOG: An International Journal of Obstetrics & Gynaecology*, 110: 34–38. doi: 10.1046/j.1471-0528.2003.00022.x
- Innovacon, (2006). 8 panel multi-card for drugs of abuse test package insert. San Diego, CA.
- Innovcan, Inc. (2012). *Drugs of abuse product information*, San Diego, CA. Retrieved February 16, 2012 from info@innovaconinc.com
- International Business Machines (IBM). (2011). Rerunning the analysis for Little's MCAR Test. Retrieved November 12, 2016 from <http://www.ibm.com/support/knowledgecenter>
- Jackson, P., & Finney, M. (2002). Negative life events and psychological distress among young adults. *Social Psychology Quarterly*, 65(2), 186-201.
- James, W., Nelson, M., Ralph, A., & Leather, S. (1997). Socioeconomic determinants of health. The contribution of nutrition to inequalities in health. *British Medical Journal*, 314(7093), 1545-1549.
- Jekielek, S., & Brown, B. (2005). The transitions to adulthood: Characteristics of young adults age 18-24 in America. *The Annie E. Casey Foundation*, Population Reference Bureau, and Child Trends. Census 2000. Retrieved April 10, 2016 from www.prb.org
- Jensen, J., Beijers, R., Riksen-Walraven, M., & de Weerth, C. (2010). Cortisol reactivity in young infants. *Psychoneuroendocrinology*, 35(3), 329-338.
- Johansson, S., & Cnattingius. (2010). Introduction: epidemiology of preterm birth. In C. Nosarti, R.M. Murray & M. Hack (Eds.), *Neurodevelopmental outcomes of preterm birth: from childhood to adult life* (pp.1-16). Cambridge, UK: Cambridge University Press.
- Johnson, S., & Marlow, N. (2011). Preterm birth and childhood psychiatric disorders. *Pediatric Research*, 69(11R-18R). doi: 10.1203/PDR.ob013e318212faao

- Joseph, D., & Newman, D. (2010). Emotional intelligence: An integrative meta-analysis and cascading model. *Journal of Applied Biology*, 95(1), 54-78.
doi: 10.1037/a0017286
- Juster, R.P., Raymond, C., Desrochers, A., Bourdon, O., Durand, N., Wan, N., Pruessner, J., Lupien, S. (2016) *Psychoneuroendocrinology* 63, 282-290. Retrieved March 4, 2017 from <http://www.researchgate.net/publication/283511728>
- Kandal, E., Schwartz, J & Jessell, T. (1991). *Principles of neural science*, USA: Appleton & Lange/McGraw-Hill.
http://cwx.prenhall.com/bookbind/pubbooks/morris5/medialib/images/F02_09.jpg
- Kanner, A. D., Coyne, J. C., Schaefer, C., & Lazarus, R. R. (1981). Comparison of two modes of stress measurement: Daily hassles and uplifts versus major life events. *Journal of Behavioral Medicine*, 4, 1-39.
- Kaseva, N., Pyhälä, R., Wehkalampi, K., Feldt, K., Kati, A., Heinonen, A., Hovi, L., Järvenpää, K., Eriksson, J., Andersson, S., Räikkönen, K., & Kajantie, E. (2014). Adrenalin, noradrenalin and heart rate responses to psychosocial stress in young adults born preterm at very low birth weight. *Clinical Endocrinology*, 81(2). 231–237
- Katz, J., Lee, A., Kozuki, N., Lawn, J.E., Cousens, S., Blencowe, H., Ezzati, M., Bhutta, and 25 more. (2013). Mortality risk in preterm and small- for -gestational–age infants in low-income and middle-income countries: a pooled country analysis. *Lancet*, 382(9890), 417-425. doi: 10.1016/S0140-6736(13)60993-9
- Kelly, M., Tyrka, A., Anderson, G., Price, L., & Carpenter, L. (2007). Sex differences in emotional and physiological responses to the Trier Social Stress Test. *Journal of Behavior Therapy*, 39, 87-98.
- Kessler, R., Adler, L., Ames, M., Demler, O., Faraone, S., Hiripr, E., Howes, M., Jin, R., Secnik, K., Spencer, T., Ustun, B., & Walters, E. (2005). The World Health Organization adult ADHD self-report scale (ASRS): a short screening scale for use in the general population. *Psychological Medicine*, 35, 245-256.

doi: 10.1017/S00033291704002892

Kessler, R. C., Amminger, G. P., Aguilar-Gaxiola, S., Alonso, J., Lee, S., & Ustun, T. B. (2007). Age of onset of mental disorders: A review of recent literature. *Current Opinion in Psychiatry*, 20(4), 359–364.
<http://doi.org/10.1097/YCO.0b013e32816ebc8c>

Kessenich, M. (2003). Developmental outcomes of premature, low birth weight, and medically fragile infants. *Newborn & Infant Nursing Reviews*. Retrieved March 28, 2017 from www.medscape.com

Khoury, J., Gonzalez, A., Levitan, R., Pruessner, J., Chopra, K., Basile, V., Masellis, M., Goodwill, A., & Atkinson, L. (2015). Summary cortisol reactivity indicators: interrelations and meaning. *Neurobiology of Stress*, 2, 34-43.
 doi: 10.1016/j.ynstor.2015.04.002

Kidd, T., Carvalho, L., & Steptoe, A. (2014). The relationship between cortisol responses to laboratory stress and cortisol profiles in daily life. *Biological Psychology*, 99, 34-40. doi: 10.1016/j.biopsycho.2014.02.010. 25.

Kim, S. (2010). The nature of theoretical thinking in nursing (3rd ed.). New York, NY: Springer Publishing Co.

Kim, S. (2014). The role of theory in clinical nursing practice. Retrieved March 12, 2016 from klinisksygepleje.munksgaarddanmark.dk/.../Hesook_Suzie_Kim_The_R...

Kirschbaum, C. (2010). Trier social stress test. In I. Stolerman (Ed.), *Encyclopedia of Psychopharmacology* (pp.1346-), Berlin, Germany: Springer Berlin Heidelberg.
 doi: 10.1007/978-3-540-68706-1_53

Kirschbaum, C., & Hellhammer, D. H. (2000). Salivary cortisol. *Encyclopedia of stress*, 3(379-383). Retrieved January 24, 2015 from
<http://p113367.typo3server.info/uploads/media/lit0003.pdf>

- Kirschbaum, C., Pirke, K-M., & Hellhammer, D. (1993). The 'Trier Social Stress Test'- a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, 28, 76-81.
- Kirschbaum C, Wüst S, Hellhammer D. (1992). Consistent sex differences in cortisol responses to psychological stress. *Journal of Psychosomatic Medicine*, 54(6), 648-657.
- Kolb., B., & Whishaw. I. (Eds.). (2016). An introduction to brain and behavior (5th Ed.). New York: NY, Worth Publishers.
- Koop. M., & Rethelyi, J. (2004). Where psychology meets physiology: Chronic stress and premature mortality-the Central-Eastern European health paradox. *Brain Research Bulletin*, 62, 351-367. doi: 10.1016/j.brainresbull.2003.12.001
- Kowalczyk, D. (2015). Emotional regulation: definition, theory, & strategies. Retrieved December, 2, 2015 from: <http://study.com/academy/lesson/emotion-regulation-definition-theory-strategies.html>
- Kramer, M. R., & Hogue, C. R. (2009). What causes racial disparities in very preterm birth? A biosocial perspective. *Epidemiologic reviews*, 31(1), 84-98.
- Kristensen, H., Parker, J., Taylor, R., Keefer, K., Kloosterman, P., & Summerfeldt, L. (2014). The relationship between trait emotional intelligence and ADHD symptoms in adolescents and young adults. *Personality and Individual Differences*, 65, 36-41. doi: 10.1016/j.paid.2014.01.031
- Kudielka, B. (2008, August 20). The Trier Social Stress Test (TST). *SciTopics*. Retrieved January 25, 2011, from http://www.scitopics.com/The_Trier_Social_Stress_Test_TSST.html
- Kudielka, B., Buske-Kirschbaum, A., Hellhammer, D., & Kirschbaum, C. (2004). HPA axis responses to laboratory psychosocial stress in healthy elderly adults, young adults, and children: impact of age and gender. *Psychoneuroendocrinology*, 29, 83-98.

- Kudielka, B., Hellhammer, D., & Kirschbaum, C. (2010). Ten years of research with the Trier Social Stress Test (TSST)-revisited. In E. Harmon-Jones & P. Winkielman (Eds.), *Social Neuroscience*. New York, NY: Guilford Press.
- Kurina, L.M., Schneider, B., & Waite, L.J. (2004). Stress, symptoms of depression and anxiety, and cortisol patterns in working parents. *Stress and Health*, 20, 53-63. doi:10.1002/smi.998.
- Lakens, D. (2013). Calculating the reporting effect sizes to facilitate cumulative science: A practical primer for t-tests and ANOVAs. *Frontiers in Psychology*, 4, 863. Retrieved January 15, 2017 from <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3840331/>
- Landy, Frank. (2005). Some historical and scientific issues related to research on emotional intelligence. *Journal of Organizational Behavior*, 26, 411-424. doi: 10.1002/job.317
- Langkamp, D., Lehman, A., & Lemeshow, S. (2010). Techniques for handling missing data in secondary analyses of large surveys. *Academic Pediatrics*, 10(3), 205-210.
- Larkin, M. (2013). Can brain biology explain why men and women think and act differently? Retrieved February 3, 2016 from <https://www.elsevier.com/connect/can-brain-biology->
- Lazar, S. W., Kerr, C. E., Wasserman, R. H., Gray, J. R., Greve, D. N., Treadway, M. T., ... & Rauch, S. L. (2005). Meditation experience is associated with increased cortical thickness. *Neuroreport*, 16(17), 1893.
- Lazarus, R. (1999). *Stress and emotion: a new synthesis*. New York, NY: Springer Publishing Co.
- Leal, L. (2001). *Psychology II*. Piscataway, NJ: Research & Education Association.
- Lee, J., Fried, R., Thayer, Z., & Kuzawa, C. (2014). Preterm delivery as a predictor of diurnal cortisol profiles in adulthood: evidence from Cebu, Philippines.

American Journal of Human Biology, 26, 598-602. doi: 10.1002/ajhb.22569

Lee, J., & Niemeier, D. (1996). Advantages and disadvantages: Longitudinal vs. repeated cross-section surveys a discussion paper. Project battelle 94-16. Retrieved January 18, 2017 from l.bts.gov/data/letter_am/bat.pdf

Lenze, E.J., Mantella, R.C., & Shi, P. (2010). Escitalopram treatment in older adults with GAD and elevated cortisol. *The Brown University Geriatric Psychopharmacology Update*, 14(11), 1-5.

Leonhardt, D. (2014, Oct. 10). Heavier babies do better in school. Retrieved December 20, 2016 from <http://nyti.ms/1ti8rbh>

Lester, B. (1985). *Prediction of developmental outcomes from cry analysis* (Grant #R01-MD-19195). Bethesda, MD, National Institutes of Health, National Institute of Child Health and Development.

Lester, B., Hawes, K., Abar, B., Sullivan, M., Miller, R., Bigsby, R., Laptook, A., Salisbury, A., Taub, B., Lagasse, L., & Padbury, J. (2014). Single-family room care and neurobehavioral and medical outcomes in preterm infants. *Pediatrics*, 134(4), 754-760.

Li, S., Zhang, M., Tian, H., Liu, Z., Yin, X., & Xi, B. (2014). Preterm birth and risk of type 1 and type 2 diabetes: systematic review and meta-analysis. *Obesity Reviews*, 15(10), 804-811.

Lipinsk-Grobeiny, A. (2011). Effects of gender role on personal resources and coping with stress. *International Journal of Occupational Medicine and Environmental Health*, 24(1), 18-28. doi: 10.2478/s13382-011-0002-6

Lister, C. E., Merrill, R. M., Vance, D. L., West, J. H., Hall, P. C., & Crookston, B. T. (2015). Victimization among Peruvian adolescents: insights into mental/emotional health from the Young Lives study. *Journal of school health*, 85(7), 433-440.

Lovaglio, W. R. (2005). *Stress and health: biological and psychological interactions*. Thousand Oaks, CA: Sage Publications, Inc.

- Lynn, L., Cuskelly, M., O'Callaghan, M., & Gray, P. (2011). Self-regulation: A new perspective on learning problems experienced by children born extremely preterm *Australian Journal of Educational & Developmental Psychology*, 11, 1-10.
- Lushner, D., Swim, E., Hong, P., & Vitacco, M. (2011). Psychopathy and ability emotional intelligence: widespread or limited association among facets. *Personality and Individual Differences*, 50, 1029-1033. doi: 10.1016/j.paid.2011.01.018
- Lucke, J., Loucks, T., & Berga, S. (2007). Salivary cortisol reflects serum cortisol: analysis of circadian profiles. *Annals of Clinical Biochemistry*, 44, 281-284.
- Lupen, S., King, S., Meaney, M., & McEwen, B. (2001). Can poverty get under your skin? Basal cortisol levels and cognitive function in children from low and high socioeconomic status. *Development and Psychopathology*, 13, 653-676.
- Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: A critical evaluation and guidelines for future work. *Child development*, 71(3), 543.
- Lyons, B. (2012). Stress, coping, and health: a conceptual overview. In V. Rice (Ed). *Handbook of stress, coping and health*, (p 2-20). Thousand Oaks, CA: SAGE Pub., Inc.
- MacArthur Research Network, John D., & Catherine T. (2007). Salivary cortisol measurements. *Socioeconomic Status and Health*, (June 9, 2000). Retrieved September 5, 2015 from http://www.macses.ucsf.edu/Research/Allostatic/notebook/salivary_cort.html
- MacMillan, H., Georgiades, K., Duku, E., Shea, A., Steiner, M., Niec, A., Tanaka, M., Gensey, S., Spree, S., Vella, E., Walsh, C., De Bellis, M., Van der Meulen, J., Boyle, M., & Schmidt, L. (2009). Cortisol response to stress in female youths exposed to childhood maltreatment: Results of the youth mood project. *Biological Psychiatry*, 66(1). doi: 10.1016/j.biopsych.2008.12.014

- MacMillan, H. L., Tanaka, M., Duku, E., Vaillancourt, T., & Boyle, M. H. (2013). Child physical and sexual abuse in a community sample of young adults: Results from the Ontario Child Health Study. *Child Abuse & Neglect*, 37(1), 14-21.
- Magee, T., Lee, S., Giuliano, K., & Munroe, B. (2006). Generating new knowledge from existing data. *Nursing Research*, 55(2S), S50-S56.
- Main, E. (2010). California maternal quality care toolkit to transform maternity care. *March of Dimes*. Retrieved March, 4, 2011 from <http://www.medscape.org/viewarticle/736635>.
- Man, V. Y., Dougan, C. M., & Recotr, N. A. (2012). Examination of coping in the anxiety disorders with and without comorbid depression. *International Journal of Cognitive Therapy*, 5(2), 186-196.
- Manian, J., Antoniadis, C., & Morris, J. (2014). Early-life stress, HPA axis adaptation, and mechanisms contributing to later health outcomes. *Frontiers in Endocrinology*, 5(73). doi: 10.3389/fendo.2014.00073
- Mannisto, T., Vaarasmaki, M., Sipola-Leppanen, M., Tikanmaki, M., Matinolli, H-M., Pensonen, A-K., Raikkonen, K., Jarvelin, M-R., Hovi, P., & Kajantie, E. (2015). Independent living and romantic relations among young adults born preterm. *Pediatrics*, 135(2), 290-297. Retrieved March 27, 2017 from pediatrics.aappublications.org
- Manzanares, N., Monseny, R., Ortega, L., Montalvo, I., Franch, J., Gutierrez-Zotes, Reynolds, R., Walker, B., Vilella, E., & Labad, J. (2014). Unhealthy lifestyle in early psychoses: The role of the hypothalamic-pituitary-adrenal axis. *Psychoneuroendocrinology*, 39, 1-10. doi: 10.1016/j.psyneuen.2013.09.023
- March of Dimes, (2008). Low birth weight. Retrieved August, 14, 2011, from <http://www.marchofdimes.com/medical-resources-lowbirthweight.html>

- Martin, C., & Fisher, P. (2012). Racial and ethnic differences in diurnal cortisol rhythms in preadolescent: the role of parental psychosocial risk and monitoring. *Hormones and Behavior*, Retrieved from March 5, 2012. <http://www.sciencedirect.com>
- Martin, J., & Osterman, M. (2013). Preterm births-United States, 2006 and 2010. *Morbidity and Mortality Weekly Report (MMWR)*, Center for Disease Control and Prevention, 62(03), 136-138. Atlanta, GA: Centers for Disease Control and Prevention. Retrieved March 22, 2015 from <http://www.cdc.gov/mmwr/preview/mmwrhtml/su6203a22.htm>
- Martin, J.A., Osterman, M.J.K., & Sutton, P.D. (2010). Are preterm births on the decline in the United States: recent data from the national vital statistics system. *National Center for Health Statistics Data Brief*, 39, 1-7.
- Martins, A., Ramalho, N., & Morin, E. (2010). A comprehensive meta-analysis of the relationship between emotional intelligence and health. *Personality and Individual Differences*, 49, 554-564. doi: 10.1016/j.paid.2010.05.029
- Matud, M. (2004). Gender differences in stress and coping styles. *Personality and Individual Differences*, 37(7), 1401-1415. doi: 10.1016/j.paid.2004.01.010
- Matthews, S. (2002). Early programming of the hypothalamic-pituitary-adrenal axis. *trends in Endocrinology and Metabolism*, 13(9), 373-380. doi: 10.1043-2760/02
- Matthews, G., Zeidner, M., & Roberts, R. (2007). *The science of emotional intelligence: knowns and unknowns*. New York, NY: Oxford University Press, Inc.
- Matthews, G., Zeidner, M., & Roberts, R. (2004). *Emotional intelligence: science & myth*. Boston, MA: First MIT Press.
- Mayer, J., Salovey, P., & Caruso, D. (2000). Emotional intelligence as zeitgeist, as personality and as a mental ability. Raven Bar-On, James Parker, & Daniel Goleman (Eds.), *The handbook of emotional intelligence*, San Francisco, CA: John Wiley & Sons, Inc.

- Mayer, J., Salovey, R., Caruso, D., & Sitarenios, G. (2001). Emotional intelligence as a standard intelligence. *Emotion* (1)3, 232-242. Retrieved October 10, 2015 from http://ei.yale.edu/wpcontent/uploads/2014/02/pub62_MayerSalovey2001_Elasstandardintelligence.pdf
- Mayer, J., Salovey, P., & Caruso, D. (2002). *Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT)*. Consortium for Research on Emotional Intelligence in Organizations. Retrieved October 10, 2015 from www.eiconsortium.org
- Mayer, J., Salovey, P., & Caruso, D. (2004). Emotional intelligence: Theory, findings and implications. *Psychological Inquiry*, 15(3), 197-215.
- Mayer, J., Salovey, P., & Caruso, D. (2004). MSCEIT™. Retrieved September 20, 2014 from <http://www.mhs.com/product.aspx?gr=io&id=overview&prod=msceit>
- Mayr, S., Erdfelder, Bucher, A., & Faul, F. (2007). A short tutorial of GPower. *Tutorials in Quantitative Methods for Psychology*, 3(2), p.51-59.
- McBeth, J., Silman, A.J., Gupta, A., Chiu, Y.H., Ray, D., Morris, R., Dickens, C., King, Y., & MacFarlane, G.J. (2006). Moderation of psychosocial risk factors through dysfunction of the hypothalamic-pituitary-adrenal stress axis in the onset of chronic widespread musculoskeletal pain: findings of a population-based prospective cohort study. *Arthritis & Rheumatism*, 56(1), 360-371.
- McCall, R., & Appelbaum, M. (1991). Some issues of conducting secondary analyses. *Developmental Psychology*, 27(6), 911-917.
- McEwen, B. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine*, 338(3), 171-179.
- McEwen, B. (2000). Allostasis and allostatic load: Implications for neuropsychopharmacology, *Neuropsychopharmacology*, 22, 108-124.
- McEwen, B. (2003). Early life influences on life-long patterns of behavior and health. *Mental Retardation and Developmental Disabilities Research Reviews*, 9, 149-154.

- McEwen, B. (2006). Protective and damaging effects of stress mediators: Central role of brain. *State of the Art*, 368-381.
- McEwen, B. S. (2008). Central effects of stress hormones in health and disease: understanding the protective and damaging effects of stress and stress mediators. *European Journal of Pharmacology*, 538, 174-185. doi:10.1016/j.ejphar.2007.11.071
- McEwen, B. (2012). Brain on stress: How the social environment gets under the skin. *Proc. National Academy of Science USA*, 16(S2), 17180-17185. doi: 10.1073/pnas.1121254109
- McEwen (2012). The ever-changing brain: cellular and molecular mechanisms for the effects of stressful experiences. *Developmental Neurobiology*, 72(6), 878-890.
- McEwen B., & Gianaros PJ. (2010). Central role of the brain in stress and adaptation: links to socioeconomic status, health, and disease. *Annals of the New York Academy of Sciences*; 1186, 90-222. doi: 10.1111/j.1749-6632.2009.05331.x
- McEwen, B., Gray, J., & Nasca, C. (2015). Recognizing resilience: learning from the effects of stress on the brain. *Neurobiology of Stress*, 1, 1-11. doi: 10.1016/j.ynstr.2014.09.001
- McEwen, B. S., & Seeman, T. (2006). Protective and damaging effects of mediators of stress: elaborating and testing the concepts of allostasis and allostatic load. *Annals of New York Academy of Science*. www.ncbi.nlm.nih.gov/pubmed/10681886.
- McEwen, B., & Wingfield, J. (2002) The concept of allostasis in biology and biomedicine. *Hormones and Behavior*, 43, 2-15. doi: 10.1016/S0018-506X(02)00024-7
- McEwen, B., & Wingfield, J. (2010). What's in a name? Integrating homeostasis, allostasis and stress. *Hormone Behavior*, 57(2), 105. doi: 10.1016/j.yhbeh.2009.09.011

- McGrath, M. (1989). *Developmental outcomes of 4 year old children born at risk* (Grant # R01 NR 02263). Bethesda, MD, National Institutes of Health, National Institute of Nursing Research.
- McGrath, M. (1994). *Competency of school age term and preterm children* (Grant # R01 NR 003695-01). Bethesda, MD, National Institutes of Health, National Institute of Nursing Research.
- McGrath, M. (1998). *Adolescent competency: Risk and protective processes* (Grant #R01 NR03695-05). Bethesda, MD, National Institutes of Health, National Institute of Nursing Research.
- McGrath, M. (2003). *Longitudinal trajectories of infants born preterm* (Grant # R01 NR R01 003695-09). Bethesda, MD, National Institutes of Health, National Institute of Nursing Research.
- McGregor, J., Leff, M., Orleans, M., & Baron, A. (1992). Fetal gender differences in preterm birth: findings in North American Cohort. *American Journal of Perinatology*, 9(1), 43-48.
- McQueen, A. (2004). Emotional intelligence in nursing work. *Journal of Advanced Nursing*, 47(1), 101-108.
- McVicar, A., Ravalier, J., & Greenwood, C. (2014). Biology of stress revisited: intercellular mechanisms and the conceptualization of stress. *Stress and Health*, 30(4), 272-279. doi: 10.1002/smi.v30.4/issuetoc
- Menard, S. (Ed.). (2007). *Handbook of longitudinal research: design, measurement, and analysis*. Burlington, MA: Elsevier.
- Merriam-Webster Medical Dictionary. (2015). Alexithymia. Retrieved December 19, 2016 from <http://beta.merriam-webster.com/medical/alexithymia>

- Milad, M. R., Pitman, R. K., Ellis, C. B., Gold, A. L., Shin, L. M., Lasko, N. B., ... & Rauch, S. L. (2009). Neurobiological basis of failure to recall extinction memory in posttraumatic stress disorder. *Biological Psychiatry*, 66(12), 1075-1082.
- Miller, R., & Brewer, D. (2003). *The A-Z of social research: A dictionary of key social science research concepts*. Thousand Oaks, CA: SAGE Publishers, Ltd.
- Miller, G., Chen, E., & Zhou, E. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, 133(1), 25-45. doi: 10.1037/0033-2909.133.1.25
- Miller, L., & Smith, A. (2015). Stress: The different kinds of stress. *American Psychological Association (APA)*. Retrieved March 17, 2017 from <http://www.apa.org/helpcenter/stress-kinds.aspx>
- Miller, R.J., Sullivan, M.C., Hawses, K., & Marks, A.K. (2009). The effects of perinatal morbidity and environmental factors on health status of preterm children at age 12. *Journal of Pediatric Nursing*, 24(2), 101-114.
- Mishra, P. (2013). Concepts, constructs and variables. Retrieved December 12, 2015 from www.scribd.com/doc
- Mishra, S. I., Scherer, R. W., Geigle, P. M., Berlanstein, D. R., Topaloglu, O., Gotay, C. C., & Snyder, C. (2015). Exercise interventions on health-related quality of life for cancer survivors (Review). Retrieved November 05, 2016 from <http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD007566.pub2/abstract>
- Moos, R. (1993), *Coping Response Inventory-Adult Form: Professional Manual*. Lutz, FL: Psychological Assessment Resources, Inc.
- Moos, R.H. (1993). *Coping responses inventory: CRI-Adult form professional manual*. Lutz, Florida: Psychological Assessment Resources Inc.
- Moos, R. H. (1995). Development and applications of new measures of life stressors, social resources, and coping responses. *European Journal of Psychological*

Assessment, 11(1), 1-13

Moos, R. (2004). *Coping Response Inventory-An Update on Research Applications and Validity Manual Supplement*. Klutz: FL: Psychological Assessment Resources, Inc.

Moos, R. H., & Holahan, C. J. (2003). Dispositional and contextual perspectives on coping: toward an integrative framework. *Journal of Clinical Psychology*, 59(12), 1387-1403. doi: 10.1002/jclp.10229

Moradi, A., Pishva, N., Ehsan, H. B., & Hadadi, P. (2011). The relationship between coping strategies and emotional intelligence. *Procedia-Social and Behavioral Sciences*, 30, 748-751.

Morelius E., Nelson, N., & Theodorsson, E. (2004). Salivary cortisol and administration of concentrated oral glucose in newborn infants: improved detection limit and smaller sample volumes without glucose interference, 64(2), 113-118. doi: 10.1080/0036551410004452.

Morris, M., Rao, U., Wang, L., & Garber, J. (2014). Cortisol reactivity to experimentally-manipulated psychosocial stress in young adults at varied risk for depression. *Depression and Anxiety*, 31(1) doi: 10.1002/da.22125

Msall, M. E., Sullivan, M. C., & Park, J. (2010). Pathways of risk and resiliency after prematurity: role of socioeconomic status. In C. Nosarti, R.M. Murray & M. Hack (Eds.), *Neurodevelopmental outcomes of preterm birth: from childhood to adult life* (pp.224-236). Cambridge, UK: Cambridge University Press.

Msall, M. E., & Park, J. J. (2008, February). The spectrum of behavioral outcomes after extreme prematurity: regulatory, attention, social, and adaptive dimensions. In *Seminars in Perinatology* (Vol. 32, No. 1, pp. 42-50). WB Saunders.

Mukaka, M. (2012). Statistics corner: a guide to appropriate use of correlation coefficients in medical research. *Malawi Medical Journal*, 24(3), 69-71.

- Munsey, C. (2006). Emerging adults: the in-between age. *American Psychological Association Bulletin*, 37(6), 68. <http://www.apa.org/monitor/jun06/emerging.aspx>
- Mustillo, S., Krieger, N., Gunderson, E. P., Sidney, S., McCreath, H., & Kiefe, C. I. (2004). Self-reported experiences of racial discrimination and Black-White differences in preterm and low-birth weight deliveries: the CARDIA Study. *American Journal of Public Health*, 94(12), 2125-2131. <http://www.ncbi.nlm.gov/pmc/articles/PMC1448602/>
- National Center for Health Statistics, (2009). Born a bit too early: recent trends in late preterm births. *NCHS Data Brief*, 24.
- National Institute of Health Research (NIHR), (2015). Research priorities Q&A. Retrieved March 15, 2017 from www.cossa.org/CPR/CPR_NIH_Priorities_QA.html
- National Institute of Mental Health. (2007a). Attention Deficit Hyperactivity Disorder (ADHD). Retrieved March 12, 2017 from <http://www.nimh.nih.gov/publicat/adhd.cfm>
- National Institute of Mental Health. (2007b). Antidepressant Medications for Children and Adolescents: Information for Parents and Caregivers. Retrieved February 3, 2017 from http://www.nimh.nih.gov/healthinformation/antidepressant_child.cfm
- National Institute of Mental Health. (2009). *Anxiety Disorders*. Bethesda, MD: Science Writing, Press & Dissemination Branch. Retrieved February 18, 2017 from <http://www.nimh.nih.gov>.
- Nelson, L. J. (2003). Rites of passage in emerging adulthood: Perspectives of young Mormons. *New directions for child and adolescent development*, 100, 33-50.
- Nelson, R. (2011). *An introduction to behavioral endocrinology* (4th ed.). Sunderland, MA: Sinauer Associates, Inc. Publishers.

- Nestler, E., Barrot, M., DiLeone, R., Eisch, A., Gold, J., & Monteggia, L. (2002). Neurobiology of Depression. *Neuron Review*, 34, 13-26.
- Ng, P.C. (2008). Is there a “normal” range of serum cortisol concentration for preterm infants? *Pediatrics*, 122(4), 873. Retrieved February 26, 2011 from www.pediatrics.org.
- Niswander, K. R., & Gordon, M. (1972). *The collaborative perinatal study: The women and their pregnancies*. Philadelphia: W.B. Saunders.
- Noble, K., Norman, M., & Farah, M. (2004). Neurocognitive correlates of socioeconomic status in kindergarten children *Developmental Science*, 8(1), 74-87
- Noorbakhsh, S., Besharat, M., & Zarei, J. (2010). Emotional intelligence and coping. *Procedia Social and Behavioral Sciences*, 5, 818-822. Retrieved September 15, 2015 from www.sciencedirect.com
- Normura, Y., & Chemtob, C. M. (2007). Conjoined effects of low birth weight and childhood abuse on adaptation and well – being in adolescence and adulthood. *Archives of Pediatric & Adolescent Medicine*, 161(2), 186-192.
- North Carolina Institute of Medicine. (2010). Socioeconomic determinants of health Chapter 11. *Prevention for the Health of North Carolina: Prevention Action Plan*, 243-272. Retrieved March 22, 2015 from <http://www.nciom.org/wp-content/uploads/NCIOM/projects/prevention/finalreport/Prevention-Chpt11.pdf>
- Nosarti, C., Murray, R., & Hack, M. (Eds.). (2010). *Neurodevelopmental outcomes of premature birth from childhood to adult life*. New York, NY: Cambridge University Press.
- Orleans, T. (2008). Forward, In Karen Glanz, Barbara Rimer, & K., Viswanath (Eds.), *Health Behavior and Health Education: Theory, Research, and Practice (4th ed.)*, p xiii-xv. San Francisco: CA: Jossey-Bass.
- Oswald, L., Zandi, P., Nestadt, G., Potash, J., Kalaydjian, A., & Wand, G. (2006). Relationship between cortisol responses to stress and personality.

Neuropsychopharmacology, 31, 1583-1591. doi: 10.1038/sj.npp.1301012

Ozer, D. J., & Benet-Martinez, V. (2006). Personality and the prediction of consequential outcomes. *Annual Review Psychology* 57, 401-421.

Pallant, J. (2010). SPSS Survival Manual (4th Ed.). Berkshire, England: McGraw-Hill.

Parent, J., Garai, E., Forehand, R., Roland, E., Potts, J., Haker, K., ... Compas, B. E. (2011). Parent mindfulness and child outcome: the roles of parent depressive symptoms and parenting. *Mindfulness*, 1(4), 254–264.
<http://doi.org/10.1007/s12671-010-0034-1>

Pariante, C., & Miller, A. (2001). Glucocorticoid receptors in major depression: relevance to pathophysiology and treatment. *Biological Psychiatry*, 49(5), 391-404.

Park, C., L., & Folkman, S. (1997). Meaning in the context of stress and coping. *Review of General Psychology*, 1(2), 115-144.

Parker, J., Saklofke, D., Wood, L., Eastabrook, J., & Taylor, R. (2005). Stability and change in emotional intelligence: exploring the transition to young adulthood. *Journal of Individual Differences*, 26(2), 100-106. doi: 10.1027/1614-0001.26.2.100

Pau, A. K., & Croucher, R. (2003). Emotional intelligence and perceived stress in dental undergraduates. *Journal of Dental Education*, 67(9), 1023-1028.

Perou, R., Bitsko, R. H., Blumberg, S. J., Pastor, P., Ghandour, R. M., Gfroerer, J. C., ... & Parks, S. E. (2013). Mental health surveillance among children—United States, 2005–2011. *Morbidity and Mortality Weekly Report Surveillance Summary*, 62(Suppl 2), 1-35.

Pesonen, A., Raikkonen, K., Heinonen, K., Anderson, S., Hovi, P., Jarvenpaa, A., Eriksson, J., & Kajantie, E. (2008). Personality of young adults born prematurely: the Helsinki study of very low birth weight adults. *Journal of Child Psychology and Psychiatry*, 49(6), 609-617

- Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: reliability, validity, and initial norms. *Journal of Youth and Adolescence*, 17(2), 117-133.
- Phillips, D. (2001). Fetal origins of adult growth: Fetal growth and programming of the hypothalamic-pituitary-adrenal axis. *Clinical and Experimental Pharmacology and Physiology*, 28, 967-970.
- Pinquart, M. (2014). Achievement of developmental milestones in emerging and young adults with and without pediatric chronic illness-a meta-analysis. *Journal of Pediatric Psychology*, 39(6), 577-585. doi: 10.1093/jpepsy/jsu017
- Pluobidis, G., Benova, L., Grundy, E., & Layton, D. (2014). Lifelong socio-economic position and biomarkers of later life health: testing the contribution of competing hypothesis. *Social Science & Medicine*, 119, 258-265. doi: 10.1016/j.socscimed.2014.02.018
- Polik, D., & Beck, C. (2012). *Nursing research: Generating and assessing evidence for nursing practice* (9th ed.). Phil., USA: Wolters Kluwer/Lippincott Williams & Wilkins.
- Por, J., Barriball, L., Fitzpatrick, J. & Roberts, J. (2011). Emotional intelligence: its relationship to stress, coping, well-being and professional performance in nursing students. *Nurse Education Today*, 31, 855-860. doi: 10.1016/j.nedt.2010.12.023.
- Prechtel, H., & Beitema, D. (1967). *The neurological examination of the full-term newborn infant*. London, England: Heineman.
- Pruessner, J. C., Kirschbaum, C., Meinlschmid, G., & Hellhammer, D. H. (2003). Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time – dependent change. *Psychoneuroendocrinology*, 916-913. doi: 10.1016/S0306-4530(2)00108-7.
- Pujol, J., Vendrell, P., Junque, C., Marti-Vilalta, J., & Capdevila, A. (1993). When does human brain development end? Evidence of corpus callosum growth up to adulthood. *Annals of Neurology*, 34(1), 71-75. doi: 10.1002/ana.v34:1/issuetoc.

- Quantitative Specialists (QS), (2014). Interpreting Leven's Test in SPSS. Retrieved December 22, 2016 from https://www.youtube.com/watch?v=F_MnYOkInEB
- Quesda, A., Tristao, R., Pratesi, R., & Wolf, O. (2014). Hyper-responsiveness to acute stress, emotional problems and poorer memory in former preterm children. *Stress*, 17(5), 389-399. doi: 10.3109/10253890.2014.949667
- Rabkin, J., & Stuenkel, E. (1976). Live events, stress, and illness. *Science*, 194(4269), 1023-1020. doi: 10.1126/science.790570
- Raff, H., Homan, P., & Skoner, D. (2003). New enzyme immunoassay for salivary cortisol. *Clinical Chemistry*, 49(1), 203-204.
- Rapaport, M., Schettler, P., & Bresee, C. A preliminary study of the effects of a single session of Swedish massage on hypothalamic-pituitary-adrenal and immune function in normal individuals. *The Journal of Alternative and Complementary Medicine*, 16(10), 1079-1088. doi: 10.1089/acm.2009.0634.
- Reef, J., Diamantopoulou, S., van Meurs, I., Verhulst, F., & van der Ende, J. (2010). Predicting adult emotional and behavioral problems from externalizing problem trajectories in a 24-year longitudinal study. *European child & adolescent psychiatry*, 19(7), 577-585.
- Regan, P., & Salsberry, P. (2005). Race and ethnic differences in determinants of preterm Birth in the USA: broadening the social context. *Social Science Medicine*, 60(10), 2217-2228.
- Reschke-Hernandez, A., Okerstrom, K., Edards, A., & Tranel, D. (2017). Sex and stress: men and women show different cortisol responses to psychological stress induced by the Trier social stress test and the Iowa singing social stress test. *Journal of Neuroscience Research*, 95, 106-114.
- Rey, L., Extremera, N., & Trillo, L. (2013). Exploring the relationship between emotional intelligence and health-related quality of life in patients with cancer. *Journal of Psychosocial Oncology*, 31, 51-64. doi: 10.1080/07347332.2012.703770

- Reynolds, P. (2007). *A primer in theory construction*. Boston, MA: Pearson, Allyn and Bacon Classics
- Reynolds, R. (2013). Glucocorticoid excess and the developmental origins of disease: two decades of testing the hypothesis – 2012 Curt Richer award winner. *Psychoneuroendocrinology*, 38, 1-11. doi: 10.1016/j.psyneuen.2012.08.012
- Rew, L., Koniak-Griffin, D., Lewis, M., Miles, M., & Sullivan, A. (2000). Secondary data analysis: New perspective for adolescent research, *Nursing Outlook*, 48(5), 223-229. doi: 10.1067/mno.2000.104901
- Rice, V (Ed.). (2012). *Handbook of stress, coping, and health: Implications for nursing research, theory, and practice* (2nd ed.). Los Angeles, CA: Sage.
- Rochman, B. (2011, August 01). How long do the effects of being born premature last? Until adulthood. Retrieved March 15, 2015 from TIME.com @ <http://healthland.time.com/2011/08/01>
- Roesch, S., Weiner, B., & Vaughn, V. (2002). Cognitive approached to stress and coping. *Current Opinions in Psychiatry*, 15(6). Retrieved March 23, 2017 from http://www.medscape.com/viewarticle/444154_print
- Rumsey, D., J. (2010). How to interpret a correlation coefficient *R*. *Statistics Essentials for Dummies* (2nd Ed.). Hoboken, NJ: Wiley Publishing, Inc. Retrieved November 11, 2016 from <http://www.dummies.com/education/math/statistics/how-to-interpret-a-correlation-coefficient-r/>
- Salimetrics, Inc. (2008, February 1). *Expanded range high sensitivity salivary cortisol EIA kit insert*. Retrieved January 3, 2016 from <http://salimetrics.com.pdf>
- Saigal, S. (2014). functional outcomes of very premature infants into adulthood. *Seminars in Fetal & Neonatal Medicine*, 19, 125-130.

- Saigal, S., Burrows, E., Stoskopf, B. L., Rosenbaum, P. L., & Streiner, D. (2000). Impact of extreme prematurity on families of adolescent children. *The Journal of pediatrics*, 137(5), 701-706.
- Saigal, S., & Doyle, L. W. (2008). An overview of mortality and sequelae of preterm birth from infancy to adulthood. *The Lancet*, 371(9608), 261-269.
- Salonen, M., Kajantie, E., Osmond, C., Forsen, T., Yliharsila, H., Paile-Hyvarinen, M., Barker, D., & Eriksson, J. (2011, July 22). Developmental origins of physical fitness: the Helsinki birth cohort. *PLOS One: Public Library of Science*, 6(7), e22302. doi: 10.1371/journal.pone.0022302
- Salovey, P., & Mayer, J. D. (1990). Emotional intelligence. *Imagination, cognition and personality*, 9(3), 185-211. Retrieved February 18, 2017 from https://scholar.google.com/scholar?hl=en&as_sdt=0,40&q=Salovey+%26+Mayer+1990
- Salovey, P., Stroud, L., Woolery, A., & Epel, E. (2002). Perceived emotional intelligence, stress reactivity, and symptom reports: Further explorations using the Trait Meta-Mood scale. *Psychology and Health*, 17(5). Retrieved February 1, 2017 from <http://www.tandfonline.com/doi/abs/10.1080/08870440290025812>
- Sapolsky, R. (2001). Atrophy of the hippocampus in posttraumatic stress disorder: How and when? *Hippocampus*, 11(2), 90-91.
- Sapolsky, R. (2004). *Why zebras don't get ulcers*. New York, NY: St. Martin's Press.
- Scheibe, S., & Carstensen, L. (2009). Emotional aging: recent findings and future trends. *Journal of Gerontological Behavioral Psychological Science and Social Science*, 65B(2): 135-144. doi: 10.1093/geronb/gbp132
- Schoendorf, K. C., Hogue, C. J., Kleinman, J. C., & Rowley, D. (1992). Mortality among infants of black as compared with white college-educated parents. *New England Journal of Medicine*, 326(23), 1522-1526.

- Schommer, N., Hellhammer, D., & Kirschbaum, C. (2003). Dissociation between reactivity of the hypothalamus-pituitary-adrenal axis and the sympathetic-adrenal-medullary system to repeated psychosocial stress. *Psychosomatic Medicine*, 65, 450-460.
- Schutte, N., & Malouff, J. (2012). Emotional intelligence mediates the relationship between mindfulness and subjective well-being. *Personality and Individual Difference*, 50, 1116-1119. doi: 10.1016/j.paid.2011.01.037
- Schutte, N., Malouff, J., Thorsteinsson, E., Bhullar, N., & Rooke, S. (2007). A meta-analytic investigation of the relationship between emotional intelligence and health. *Personality and Individual Differences*, 42, 921-933. doi: 10.1016/j.paid.2006.09.003
- Schwarzer, R. (2012). Stress and coping resources: theory and review. userpage.fu-berlin.de/~gesund/publicat/ehps_cd/health/stress.htm
- Schwarzer, R., & Schwarzer, C. (1996). A critical survey of coping instruments. In M. Zeider & N.S. Endler (Eds.), *Handbook of Coping*. New York, NY: Wiley.
- Scully, J., Tosi, H., & Banning, K. (2000). Life event checklists: Revisiting the social readjustment rating scale after 30 years. *Educational and Psychological Measurement*, 60(6), 864-876. doi: 10.1177/00131640021970952
- Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and human immune system: a meta-analytic study of 30 years of inquiry. *Psychological Bulletin*, 130(4), 601-630. doi: 10.1037//0033-2909
- Sekisui Diagnostics. (2012). Retrieved April 2, 2012 from <http://www.sekisuidiagnostic.com>
- Senterfitt, J., Long, A., Shih, M., & Teutsch, S. (2013, January). How social and economic factors affect health. *Social Determinants of Health, Issue no 1*. Los Angeles: Los Angeles County Department of Public Health. Retrieved March 22, 2015 from www.publichealth.lacounty.gov/epi

- Shonkoff J., Boyce W., McEwen B. (2009) Neuroscience, molecular biology, and the childhood roots of health disparities: building a new framework for health promotion and disease prevention. *Journal of the American Medical Association*, 301(21), 2252-2259. doi: 10.1001/jama.2009.754.
- Siela, D., & Wieseke, A. (2012). Stress, self-efficacy and health. In Virginia Rice Editor, *Stress, Coping, and Health: Implications for Nursing Research, Theory and Practice* (pp 484-509). Los Angeles, CA: Sage.
- Singer, J., & Willett, J. (2003). *Applied longitudinal data analysis: Modeling change and event occurrence*. New York, NY: Oxford University Press.
- Singh, Y., & Sharma, R. (2012). Relationship between general intelligence, emotional intelligence, stress levels and stress reactivity. *Annals of Neurosciences*, 19(3) 107-111. Retrieved February 3, 2017 from www.annalsofneurosciences.org
- Simpson, R. (2009). *MIT: young adult development project*, 1-13. Retrieved from <http://hrweb.mit.edu/worklife/youngadults/imdex.html>
- Skinner, E., & Zimmer-Gembeck, M. (2007). The development of coping. *Annual Review Psychology*, 58, 119-144. doi: 10.1146/annurev.psych.58.110405.085705
- Skogen, J., & Overland, S. (2012). The fetal origins of adult disease: a narrative review of the epidemiological literature. *Journal of the Royal Society of Medicine Short Reports*, 3(59). doi: 10.1258/shorts.2012.012048
- Sloboda, D., Newnham, J., Moss, T., & Challis, J. (2006). The fetal hypothalamic-pituitary- adrenal axis: relevance to developmental origins of health and disease. In P. Gluckman & M. Hanson (Eds.), *Developmental origins of health and disease* (pp. 191-205). Cambridge, UK: Cambridge University Press.
- Smith, K., Profetto-McGrath, J., & Cummings, G. (2009). Emotional intelligence and nursing: An integrative literature review. *International Journal of Nursing Studies*, 46, 1624-1636. doi: 10.1016/j.ijnurstu.2009.05.024

- Smith, S., & Vale, W. (2006). The role of the hypothalamic-pituitary-adrenal axis in neuroendocrine responses to stress. *Dialogues in Clinical Neuroscience*, 8(4), 383-406. Retrieved from www.dialogues-cns.org
- Society for Maternal-Fetal Medicine (SMFM). (2015). Impact of fetal gender on risk of preterm birth. *Science Daily*. Retrieved February 2, 2015 from www.sciencedaily.com/releases/2015/02/150202123708.htm
- Somerfield, M. R., & McCrae, R. R. (2000). Stress and coping research. *American Psychologist*, 55(6), 620-625. doi: 10.1037//0003-066X.55.6.620
- Sorensen, H., Sabroe, S., & Olsen, J. (1996). A framework for evaluation of secondary data sources for epidemiological research. *International Journal of Epidemiology*, 25(2), 435-442.
- Spence, J.T., Cotton, J.W, Underwood, B. J. & Duncan, C.P. (1976). *Elementary statistics*. Englewood Cliffs, N.J.: Prentice-Hall, Inc.
- Standridge, J., Adams, S., & Zotos, A. (2010). Urine drug screening: a valuable office procedure. *American Family Physician*, 81(5), 635-640.
- Sterling, P., & Schulkin, J. (2004). Allostasis, homeostasis, and the costs of physiological adaptation. *Principles of Allostasis: Optimal design, predictive regulation, pathophysiology, and rational therapeutics*. Cambridge University Press, Cambridge.
- Stevenson, D., Verter, J., Fanaroff, A., Oh, W., Ehrenkranz, R., Shankaran, S., ... Papile, L. (2000). Sex differences in outcomes of very low birthweight infants: the newborn male disadvantage. *Archives of Disease in Childhood. Fetal and Neonatal Edition*, 83(3), F182–F185. Retrieved from <http://doi.org/10.1136/fn.83.3.F182>
- Stough, C., Saklofske, D. H., & Parker, J. D. (2009). A brief analysis of 20 years of emotional intelligence: An introduction to assessing emotional intelligence: Theory, research, and applications. In *assessing emotional intelligence* (pp. 3-8). US: Springer

- Strang-Karlsson, S. (2011). *Very low birth weight infants as young adults: Focus on aspects of cognition, behavior and sleep* (Unpublished doctoral dissertation). University of Helsinki, Finland.
- Strobino, D. (2006). Measures of Neonatal Morbidity. Course Slides: John Hopkins University and Donna Strobino. Retrieved March 20, 2017 from <http://ocw.jhsph.edu/courses/PreventingInfantMortality/PDFs/Lecture9.pdf>
- Strobino, D., & Baruffi, G. (1984). Evaluation of a Measure of Neonatal Morbidity. *Medical Care*, 22(9), 818-826. Retrieved January 7, 2017 from <http://www.jstor.org/stable/3764607>
- Stuss, D., (2011). Functions of the frontal lobes: relation to executive functions. *Journal of International Neuropsychological Society*, 17, 759-765. doi: 10.1017/S1355617711000695
- Substance Abuse and Mental Health Services (SAMHSA) of the National Institute of Mental Health (NIMH). (2014). 2014 SAMHSA NSDUH mental health findings: any mental illness (AMI) among U.S. adults. *The National Institute of Health*. Retrieved March 28, 2016 from www.nimh.nih.gov
- Sullivan, M. C. (2008 - 2013). *Risk and protection in trajectories of preterm infants: Birth to adulthood*. (Grant # NIH R01 NR003695-14). Bethesda, MD, National Institutes of Health, National Institute of Nursing Research.
- Sullivan, M., Hawes, K., Winchester, S., & Miller, R. (2008). Developmental origins theory from prematurity to adult disease. *Journal of Obstetric, Gynecologic, and Neonatal Nursing*, 37(2), 158-164. doi: 10.1111/j.1552-6909.2008.00216.x
- Sullivan, M., Msall, M., & Miller, R. (2012). 17-year outcomes of preterm infants with diverse neonatal morbidities: Part, 1, impact on physical neurological and psychological health status. *Journal for Specialists in Pediatric Nursing*, 17(3), 226-241.
- Sullivan-Bolyai, S., & Bova, C. (2010). Experimental and quasi-experimental designs. In Geri LoBiondo-Wood & Judith Haber (Eds.), *Nursing Research Methods and*

Critical Appraisal for Evidence-Based Practice (pp.117-194).
New York, NY: Mosby.

Tamres, L., & Helgeson, D. (2002). Sex differences in coping behaviors: a meta-analytic Review and an examination of relative coping. *Personality and Social Psychology Review*, 6(1), 2-130. doi: 10.1207/S15327957PSPR0601_1

Tang, Y., Ma, Y., Wang, J., Fan, Y., Feng, S., Lu, Q., Sui, D., Rothbart, M., Fan, M., & Posner, M. (2007). Short-term meditation training improves attention and self-regulation. *Proceedings of the National Academy of Sciences of the United States of America (PNAS)*, 104(43), 171532-17156. Retrieved April 19, 2015 from www.pnas.org/cgi/doi/10.1073/pnas.0707678104

Tarasuik, J., Ciorciari, J., & Stough, C. (Eds.)(2009). Understanding the neurobiology of emotional intelligence: a review. *Assessing Emotional Intelligence*. Springer Science+Business Media: Berlin, Germany. doi: 10.1007/978-0-387-88370-0_16

Taylor, G. (2000). Recent developments in alexithymia theory and research. *Canadian Journal of Psychiatry*, 45(2), 134-142.

Taylor, G., Bagby, R., & Parker, J. (1991). The alexithymia construct: A potential paradigm for psychosomatic medicine. *Psychosomatics*, 32(2), 153-164.

Thomas L. & Krebs C., (1997). A review of statistical power analysis software. *Bulletin of the Ecological Society of America*, 78, 126-139.

Thompson, R. (1994). Emotion regulation: A theme in search of a definition. *Monographs of the Society for Research in child development*, 59(2/3), 25-52. Retrieved June 16, 2016 from <http://s3.amazonaws.com/academia.edu.documents/30501353/thompson1994.pdf>

Thompson, M. (2015). How does the “toxic stress” of poverty hurt the developing brain? Retrieved from PBS NEWSHOUR aired June 27, 2015 retrieved March 24, 2017 from www.pbs.org/newshour/bb/toxic-stress-poverty-hurt-developing-brain

- Tollenarr, M.S., Janse, J., Beijers, R., Riksen - Walraven, M., & de Weerth, C. (2010). Cortisol in the first year of life: normative values and intra-individual variability. *Early Human Development*, 86,13-16. doi: 101016/j.earlhumdev.2009.12.003.
- Trickey, M., Farhall, J., Wertheim, E., Hinch, C., & Ong, B. (2001). An examination of the relationships among emotion management, interpersonal hassles, and depressive symptomatology. *Australian Journal of Psychology*, 63, 100-106. doi: 10.1111/j.1742-9536.2011.00016.x
- Trouillet, R., Gana, K., Lourel, M., & Fort, I. (2009). Predictive value of age for coping: the role of self-efficacy, social support satisfaction and perceived stress. *Aging and Mental Health*, 13(3), 357-366.
- Tsaousis, I., & Nikolaou. (2005). Exploring the relationship of emotional intelligence with physical and psychological health functioning. *Stress and Health*, 21, 77-86. doi: 10.1002/smi.1042
- Turner-Cobb, J. (2005). Psychological and stress hormone correlates in early life: a key to HPA-axis dysregulation and normalization. *Stress*, 8(1), 47-57. doi: 10.1080/10253890500095200
- University of California, Los Angeles (UCLA). (2016). G*Power data analysis examples one-way ANOVA power analysis. Retrieved December 10, 2016 from www.ats.ucla.edu/stst/power/anova.html
- University of Florida Maternal Child Health Education and Research and Data Center (UF). (January 3, 2006). Black baby girls more likely to live when born very prematurely. Retrieved March 12, 2016 from <http://news.ufl.edu/archive/2006/01/black-baby-girls-more-likely-to-live-when-born-very-premature.html>
- University of Rhode Island (URI). (2011). Effects of premature birth can reach into adulthood. *Science Daily*. Retrieved June 16, 2011 from www.sciencedaily.com/releases/2011/06/110615171408.htm
- U.S. National Institute of Mental Illness. (2014). *Any mental illness (AMI) among U.S.*

- adults. 2014 SAMHSA NSDUH Mental Health Findings. Retrieved February 3, 2017 from: <https://www.nimh.nih.gov/health/statistics/prevalence/any-mental-illness-ami-among-us-adults.shtml>
- Uston, B., & Kennedy, C. (2009). What is 'functional impairment'? Disentangling disability from clinical significance. *World Psychiatry*, 8(2), 82-85.
- Van Rooy, D., & Viswesvaran, C. (2004). Emotional intelligence: A meta-analysis investigation of predictive validity and nomological net. *Journal of Vocational Behavior*, 65, 71-95. doi: 10.1016/S0001-8791(03)00076-9
- Vyas A, Mitra R, Rao B., & Chattarji S. (2002). Chronic stress induces contrasting patterns of dendritic remodeling in hippocampal and amygdaloid neurons. *Journal of Neuroscience*, 22(15): 6810-6828.
- Velasco, C., Fernandez, I., Paez, D., & Campos, M. (2006). Perceived emotional intelligence, alexithymia, coping and emotional regulation. *Psicothema*, 18(Supl), 89-95. Retrieved June 18, 2016 from www.psicothema.com
- Vreeberg, S., Hoogendijk, W., van Pelt, J., DeRijk, R., Verhagen, J., van Dyck, R., Smit, J., Zitman, F., & Penninx, B. (2009). Major depressive disorder and hypothalamic-pituitary-adrenal axis activity: results from a large cohort study. *Archives of General Psychiatry*, 66(6), 617-626.
- Vohr, B.R., Wright, L.L., Dusik, A.M., Mele, L., Verter, J., Steichen, J.J., Simon, N.P., Wilson, D.C., et al. (2000). Neurodevelopmental and functional outcomes of extremely low birth weight infants in the national institute of child health and human development neonatal research network, 1993-1994. *Pediatrics*, 105(6), 1216-1226.
- Wadhwa, P., Culhane, J., Rauh, V., & Barve, S. (2001). Stress and premature birth: Neuroendocrine, immune/inflammatory, and vascular mechanisms. *Maternal and Child Health*, 5(2), 119-125. doi: 10.1023/A:1011353216619
- Wandhwa, P., Buss, C., Entringer, S., & Swanson, M. (2009). Developmental origins of health and disease: brief history of the approach and current focus on epigenetic mechanisms. *Seminars Reproductive Medicine*, 27(5), 358-368.

doi: 10.1055/s-0029-1237424

- Wang, J., Korzykowski, M., Rao, H., Fan, Y., Pluta, J., Gur, R., McEwen, B., & Dttre, J. (2007). Gender differences in neural response to psychological stress. *Social Cognitive Affective Neuroscience*, 2(3), 227-239.
- Ware, J. H. (1985). Linear models for the analysis of longitudinal studies. *The American Statistician*, 39(2), 95-101.
- Waterland, R., & Michels, K. (2007). Epigenetic epidemiology of the developmental origins hypothesis. *Annual Review of Nutrition*, 27, 363-388.
doi: 10.1146/annurev.nutr.27.061406.093705
- Weintrib, A.Z., Sephton, S.E., Degesst, K., Penedo, F., Bender, D., Zimmerman, B., Kirschbaum, C., Sood, A.K., Lubaroff, D.M., & Lutgendorf, S.K. (2010). Diurnal cortisol dysregulation, functional disability, and depression in women with ovarian cancer. *Cancer*, 116(18), 4410-4419.
- Welberg, L., & Seckl, J. (2001). Prenatal stress, glucocorticoids and programming of the brain. *Journal of Neuroendocrinology*, 13, 113-128.
- Winchester, S., Sullivan, M., Marks, A., Doyle, T., & McGrath, M. (2009). Academic, social, and behavioral outcomes at age 12 of infants born prematurely. *Western Journal of Nursing Research*, 31(7), 853-871.
doi: 10.1177/0193945909339321
- Winchester, S., Sullivan, M., & Msall, M. (2014). Executive function in adult survivors of prematurity. In Katherine P. Bennett, Editor, *Executive Functioning*. Hauppauge, New York: Nova Science Publishers, Inc.
- Winchester, S., Sullivan, M., Roberts, M., & Granger, D. (2016). Prematurity, birth weight, and socioeconomic status are linked to atypical diurnal hypothalamic-pituitary-adrenal axis activity in young adults, *Research in Nursing & Health*, 15-29. doi: 10.1002/nur.21708

- Wilkinson, R., & Marmot, M. (2003). *The Solid Facts*, second edition. Richard Wilkinson & Michael Marmot (Eds.). Retrieved March 23, 2015 from http://www.euro.who.int/__data/assets/pdf_file/0005/98438/e81384.pdf
- Windle, Pamela. (2010). Secondary data analysis: Is it useful and valid? *Journal of PeriAnesthesia Nursing*, 25(5), 322-324. doi: 10.1016/j.jopan.2010.07.005
- Wolf, S. (2005). *Emotional Competence Inventory (ECI): Technical Manual*. Hay Group, McClelland Center for Research and Innovation. Retrieved January 3, 2016 from http://www.eiconsortium.org/pdf/ECI_2_0_Technical_Manual_v2.pdf
- Wolman, C., Resnick, M. D., Harris, L. J., & Blum, R. W. (1994). Emotional well-being among adolescents with and without chronic conditions. *Journal of Adolescent Health*, 15(3), 199-204.
- Women & Infants Hospital. (Unpublished, 2012). Urine pregnancy test: OSM hCG combo test. *Point of Care Testing Program*.
- Woodward, S. H., Kaloupek, D. G., Streeter, C. C., Martinez, C., Schaer, M., & Eliez, S. (2006). Decreased anterior cingulate volume in combat-related PTSD. *Biological Psychiatry*, 59(7), 582-587.
- World Health Organization. (2003). *The solid facts*, second edition. Richard Wilkinson & Michael Marmot (Eds.). Retrieved March 23, 2015 from http://www.euro.who.int/__data/assets/pdf_file/0005/98438/e81384.pdf
- World Health Organization. (2015). The determinants of health. *Health Impact Assessment (HIA)*. Retrieved November 29, 2015 from <http://www.who.int/hia/evidence/doh/en/>
- World Health Organization. (2015, November). *Preterm birth*, Fact sheet N363. Retrieved November 29, 2015 from <http://www.who.int/mediacentre/factsheets/fs363/en/>

- World Health Organization (WHO). (2015). Gender and women's mental health. Retrieved November 29, 2015 from http://www.who.int/mental_health/prevention/genderwomen/en/
- Wright, C., & Sullivan, M. (2011). [Psychiatric symptoms and pre-term infants: is perception of vulnerability a factor?]. Major paper in partial fulfillment of MS degree. University of Rhode Island.
- Washington State University (WSU). (2013). A primer of scientific terms. Retrieved January 23, 2017 from www.wsu.edu/~busselle/Terms.html
- Wu, A., and Bruno D. Zumbo . (2008). Understanding and using mediators and moderators. *Social Indicators Research*, 87(3), 367-392.
- Wust, S., Entringer, S., Federenko, I., Schlotz, W., & Hellhammer, D. (2005). Birth weight is associated with salivary cortisol responses to psychosocial stress in adult life. *Psychoneuroendocrinology* 30(6): 591-598.
doi: 10.1016/j.psyneuen.2005.01.008
- Wust, S., Wolf, J., Hellhammer, D. H., Federenko, I., Schommer, N., & Kirschbaum, C. (2000). The cortisol awakening response – normal values and confounds. *Noise & Health: A Bimonthly Inter-disciplinary International Journal*, [serial online] 2000 [cited 2012 Jan 22] 2,79-88. Retrieved from January 3, 2017 from <http://www.noiseandhealth.org/text.asp?2000/2/7/79/31739>
- Yea, J., & Niemeier, D. (1996). Advantages and disadvantages: Longitudinal vs. repeated cross-section surveys a discussion paper. Project Battelle, 94-16. Retrieved February 2, 2017 from 1.bts.gov/data/letter_am/bat.pdf
- Young, E., Abelson, J., & Cameron, O. (2004). Effect of comorbid anxiety disorders the hypothalamic-pituitary-adrenal axis response to a social stressor in major depression. *Biological Psychiatry*, 56, 113-120.
- Yu, C. (2010). *Reliability of self-report data*. Retrieved August 13, 2011 from <http://www.creative-wisdom.com>.

Zeidner, M., Matthew, G., & Roberts, R. (2009). *What we know about emotional intelligence: How it affects learning, work, relationships, and our mental health*. Cambridge, MA: MIT Press.