Attachment and Emotion Regulation in Adolescents with FAS-Dysmorphism

A minor dissertation submitted in partial fulfilment of the requirements for the award of the degree of Master of Arts (Psychological Research)

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DECLARATION: I hereby declare that this submission is my own work, both in concept and execution, and that to the best of my knowledge and belief it contains no material written by another person or material that has been accepted for the award of any other degree or diploma of the university or other institute of higher learning, except where due acknowledgment has been made in the text.

Signature: Signed by candidate Date: 15 March 2021

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Abstract

Children with fetal alcohol spectrum disorders (FASD) are frequently reported by caregivers and teachers to have difficulties in socio-emotional functioning. It has been suggested that deficits in emotion regulation underlie these deficits in socio-emotional functioning. Children with prenatal alcohol exposure (PAE) have also been found to demonstrate higher rates of insecure attachment, as well as disorganised attachment. Previous research proposes that the attachment relationship serves as a foundation for the development of emotion regulation. However, while this association has been examined in typically developing samples, the relation between attachment and emotion regulation has yet to be examined in individuals with FASD. Previous research has also suggested that a portion of individuals who are considered heavily exposed but are non-syndromal (HE) who do not present with the full phenotype characteristic of FAS or PFAS may nevertheless present with subtle facial dysmorphism only detectable using dense surface modelling and signature analyses of 3D facial images. Furthermore, it has been shown that HE children with this subtle facial dysmorphism perform more poorly on cognitive assessments than HE children who do not present with this subtle facial dysmorphism. These findings therefore suggest that presence of even subtle dysmorphism (i.e. only detectable on signature analyses of 3D facial images) consistent with FAS can be a good indicator of cognitive performance. Given this, the aims of the current study were to examine the association between FAS-dysmorphism and insecure as well as disorganised attachment. Furthermore, the study aimed to examine the extent to which FAS-dysmorphism was associated with emotion regulation difficulties in adolescence after controlling for the effects of attachment security in infancy. Participants included 77 adolescents (*M* age = 17.66, *SD* = .7): 13 with fetal alcohol syndrome (FAS), 12 with partial FAS (PFAS), 7 HE-dysmorphic (HE+), 18 HE-nondysmorphic (HE-) and 27 non- or minimally exposed controls. At the 13-month infant assessment, the mother-infant

attachment relationship was assessed using the Ainsworth Strange Situation Paradigm (Hay, Jacobson, Molteno, Viljoen, & Jacobson, 2004; Jacobson & Jacobson, 2013). At the adolescent follow-up assessment, emotion regulation was assessed using two caregiver-report measures – the Emotion Regulation Checklist (ERC; Shields & Cicchetti, 1997) and the Affect Regulation Checklist (ARC; Moretti, 2003). Results showed that infants with FAS-dysmorphism+ did not demonstrate significantly higher levels of insecure and disorganised attachment than those in the FAS-dysmorphism- group. In terms of emotion regulation, FAS-dysmorphism+ did not significantly predict any of the adaptive or maladaptive emotion regulation subscales. However, insecure attachment predicted maladaptive emotion regulation, but not adaptive emotion regulation. These results suggest that while individuals with FASD are often reported to exhibit difficulties with emotion regulation, other variables, including IQ and attachment security in infancy, may explain some of the variation in the emotion regulation difficulties observed for individuals with FASD.

Introduction

This thesis is submitted for the degree of Master of Psychological Research at the University of Cape Town. The hypotheses tested in this thesis were formed by reviewing the body of literature and determining where gaps in that literature existed. I then collected the emotion regulation data, conducted the data analysis, wrote up the results, and interpreted the findings. This process is described in this thesis. This thesis was initiated and conducted under the supervision of Professor Sandra Jacobson, who supervised the data analysis and initial interpretation of the findings. This thesis was also supervised by Associate Professor Susan Malcolm-Smith in the Department of Psychology and co-supervised by Dr Nadine Lindinger in the Department of Human Biology, both at the University of Cape Town. Drs. Sandra Jacobson and Joseph Jacobson are Distinguished Professors in the Department of Psychiatry and Behavioral Neuroscience at Wayne State University, Michigan, and Honorary Professors in the Departments of Human Biology and of Psychiatry and Mental Health at the University of Cape Town. The Cape Town Longitudinal Cohort study was funded by grants from the National Institutes of Health (R01-AA09524, U01-AA14790, R01-AA016781, R01-AA023503) to Professor Sandra Jacobson and the Lycaki-Young Fund from the State of Michigan to Professors Sandra and Joseph Jacobson.

This thesis serves as an exploration of infant attachment and emotion regulation in South African adolescents with FAS-dysmorphism. While a small body of research suggests that infants with prenatal alcohol exposure (PAE) exhibit higher rates of insecure and disorganised attachment, the association between attachment and FAS-dysmorphism, specifically, has yet to be examined. The current study therefore aimed to examine the association between FAS-dysmorphism and infant attachment patterns. Research has also suggested that individuals with FASD present with impairments in their ability to regulate their emotions. Given that the attachment relationship to the caregiver is thought to be the

basis on which emotion regulation develops, numerous previous studies have examined the relationship between attachment and emotion regulation in typically developing samples. However, to date, this relationship has yet to be examined in individuals with FASD. Considering that individuals with PAE have been found to exhibit both higher levels of insecure attachment, as well as difficulties with emotion regulation, the current study therefore also aimed to examine the extent to which FAS-dysmorphism was associated with these emotion regulation difficulties after controlling for the effects of attachment security.

This thesis is written up across four sections. The first section is the literature review, in which I discuss relevant previous research on FASD, emotion regulation, and attachment. To begin, I provide an overview of FASD with reference to the history of the disorder, the various subtypes and their respective diagnostic criteria, as well as the cognitive and behavioural impairments associated with FASD. I also provide context regarding FASD in South Africa. I then discuss the socio-emotional impairments frequently reported in FASD. Given that emotion regulation is a mechanism that has been proposed to underlie these impairments in socio-emotional functioning, I go on to review the literature on emotion regulation, including that which has been conducted with individuals with FASD. The co-regulation that takes place in an infant's early attachment relationship with their caregiver is proposed to be the basis from which emotion regulation capacity is developed. As such, I then move on to review the literature on attachment with reference to attachment in FASD, as well as the association between attachment in infancy and later emotion regulation. The second section of the thesis comprises of the methods and context of the current study. In the third section, I present the results of the study. Finally, in section 4, I provide a discussion of the findings relating to each hypothesis with reference to previous literature. The strengths and limitations of the current study, as well as recommendations for future research are then presented before concluding the thesis.

To my knowledge, this study is the first to investigate the association between FAS-dysmorphism and attachment pattern. Furthermore, it is the first study to consider the influence of attachment when examining the association between FAS-dysmorphism and emotion regulation in adolescence.

Literature Review

Overview of FASD

The link between maternal alcohol consumption during pregnancy and the problems observed in the children born to these mothers was first described in the 1960s (Lemoine et al., 1968). However, it was only several years later that the specific pattern of impairment associated with the children who were most severely affected by prenatal alcohol exposure (PAE) was described and coined 'fetal alcohol syndrome' (FAS; Jones & Smith, 1973; Jones et al., 1973). As clinicians became more familiar with the profile of impairment of children with PAE, it became evident that varying levels of PAE could result in varying degrees of physical, cognitive and behavioural impairments. Today we know that the impact of PAE on the developing brain vary depending on the amount, frequency and timing of maternal alcohol consumption (Mattson, Riley, Gramling, Delis, & Jones, 1998; Olson, 2015; Paley & O'Connor, 2007). The resulting abnormalities and deficits occur on a continuum, ranging from mild to severe and are subsumed under the term fetal alcohol spectrum disorders (FASD; Stratton, Howe, & Battaglia, 1996).

The Continuum

The consumption of alcohol during pregnancy interferes with the typical growth and development of the fetus (Carmichael-Olson, Jirikowic, Kartin, & Astley, 2007; Carter et al., 2017). The variability of the effects of PAE on the foetus results in a continuum of mild to severe impairments. The pattern of drinking, including the timing and dosage (i.e., amount

and frequency) of alcohol exposure, collectively influences the severity of the observed effect (Jacobson et al., 2008). Binge drinking, in particular, has been found to cause the most harm to the developing fetus (Olson et al., 2007). However, other maternal and child risk factors (e.g., maternal malnutrition, maternal *ADH1B* polymorphic differences which affect the rate at which alcohol is metabolised, other substance abuse), and genetic and epigenetic differences have also been found to influence the severity of the presenting impairments (Carter et al., 2017, 2018; Dodge, Jacobson, & Jacobson, 2014; Jacobson & Jacobson, 1999; Jacobson et al., 2008; May & Gossage, 2011). Age at which the child is examined for dysmorphic features is also an influential factor (Jacobson et al., 2021). In addition to these factors, severity of impairment may also be impacted by maternal age, stature, and socioeconomic status (SES). That is, older mothers of a smaller stature who consume alcohol during pregnancy have been found to be more likely to give birth to children who exhibit greater PAE-related impairment (Carter, Jacobson, Sokol, Avison, & Jacobson, 2013; Jacobson, Jacobson, Sokol, Chiodo, & Corobana, 2004). Similarly, children born to mothers of a lower SES have also been found to be more severely affected (May et al., 2008). Thus, while higher levels of PAE generally correspond with more severe and pervasive impairments (Stratton et al., 1996), the factors listed above further contribute to the severity of the effects observed. The phenotypes resulting from the interplay of the effects of PAE and the various maternal risk factors are grouped into 4 distinct subtypes: FAS, partial FAS (PFAS), alcohol-related birth defects (ARBD), and alcohol-related neurodevelopmental disorder (ARND).

Diagnosis along the FASD continuum

Strict criteria governing the diagnosis of FASD were first outlined by the United States Institute of Medicine (IOM) of the National Academy of Science in 1996. These criteria have since been revised in 2005 and 2016 (Hoyme et al., 2005, 2016). In order to

assign a diagnosis, evidence within the following four categories is needed: (a) maternal alcohol consumption during pregnancy; (b) the presence of a specific pattern of facial anomalies; (c) prenatal and/or postnatal growth retardation; and (d) abnormal central nervous system (CNS) development. The subtype is then diagnosed based upon the combination of attributes with which the individual presents.

FAS, the most severe of the fetal alcohol disorders along the FASD continuum, is characterised by a pattern of craniofacial abnormalities (e.g. microcephaly, short palpebral fissures, flat philtrum, thin upper lip, flat midface, and low nasal bridge), prenatal and/or postnatal growth retardation, and abnormal central nervous system (CNS) development and functioning (Hoyme et al., 2005: 2016). In order for an individual to be diagnosed with FAS, they must present with at least 2 of the craniofacial abnormalities. It must be noted, however, that similar physical anomalies can also occur in other syndromes such as Williams Syndrome or Fetal Hydantoin Syndrome. Great attention should therefore be given to distinguishing phenotypical PAE-related anomalies from those associated with these other syndromes (Hoyme et al., 2016). If an individual presents with all of the abovementioned characteristics of FAS, maternal alcohol consumption during pregnancy can be assumed and a diagnosis of FAS may be made, provided other syndromes that resemble PAE-related abnormalities have been eliminated.

In order for an individual to be diagnosed with PFAS, they must present with at least 2 of the characteristic craniofacial abnormalities, together with either prenatal and/or postnatal growth retardation, microcephaly, or deficits in CNS development and functioning (Hoyme et al., 2005, 2016). As of 2016, the revised IOM criteria no longer require maternal alcohol consumption during pregnancy to be confirmed in order to assign a diagnosis of PFAS. However, if alcohol consumption during pregnancy can be confirmed, only the craniofacial abnormalities and the deficits in CNS development and functioning need to be

present for a diagnosis to be assigned. Once again, it is important that PAE-related abnormalities are distinguished from those that could be attributed to other syndromes.

Children who present with alcohol-related birth defects (ARBD) exhibit PAE-related congenital structural abnormalities in the absence of other deficits characterising FAS (e.g. craniofacial abnormalities, deficits in CNS development and functioning; Hoyme et al., 2005, 2016). Alcohol-related neurodevelopmental disorder (ARND) refers to prenatally alcohol exposed individuals with CNS deficits and/or cognitive and behavioural deficits who do not meet criteria for a diagnosis of FAS or PFAS (i.e. they do not exhibit craniofacial abnormalities or growth retardation). For a diagnosis of either ARBD or ARND, maternal alcohol consumption during pregnancy must be confirmed.

Facial Dysmorphism

Typically, facial anomalies characteristic of FAS, which are also present to some extent in the less severe subtypes along the spectrum, are identified by manually measuring the distance between facial landmarks (Douglas & Mutsvangwa, 2010). However, the techniques and methodology used for identifying individuals along the spectrum of FASD has advanced substantially in the last two decades (May et al., 2020). For instance, several relatively recent studies have explored the use of three dimensional (3D) technologies to develop image analysis techniques to aid in identifying individuals with facial features characteristic of FAS (Douglas & Mutsvangwa, 2010; Fang et al., 2008; Suttie et al., 2013). For example, Fang and colleagues (2008) developed a method that allowed the automatic computation of facial features from 3D images. Upon applying pattern recognition to these images, they were able to successfully distinguish between individuals with FAS and ethnically similar controls.

Not only are these techniques helpful in identifying those with FAS, but they are also able to identify facial dysmorphism along the less severe end of the spectrum. In 2009,

Jacobson et al. (2011) organised a diagnostic clinic at which the children enrolled in the Cape Town Longitudinal Cohort, from which the sample for the current study was recruited, were independently examined for growth and dysmorphic features by two expert dysmorphologists, H.E. Hoyme, MD, and L. Robinson, MD, who were blind regarding prenatal alcohol history of the children. At this clinic 3D photos were also taken of the children. Based on subsequent case conferences conducted by Hoyme, S. Jacobson, J. Jacobson, and R.C. Carter, in which alcohol exposure was also considered, the children were assigned to one of four diagnostic categories: FAS, PFAS, HE (individuals who were heavily exposed but non-syndromal), and controls (Jacobson et al., 2021).

Using dense surface modelling and signature analyses of the 3D facial images, Suttie and colleagues (2013) subsequently sought to determine level of agreement of face classification with clinical categorization across the spectrum of fetal alcohol disorders. Perfect agreement between face classification and clinical categorization was achieved for FAS, and agreement remained strong when PFAS was included. Importantly, Suttie and colleagues (2013) were able to detect subtle facial dysmorphism consistent with FAS and PFAS in a subset of children from the HE category. Interestingly, this subset of HE children also demonstrated greater cognitive impairment on the WISC-IV Verbal Comprehension Index (Wechsler, 2004) and the children's version of the California Verbal Learning Test (CVLT-C; Delis, Kramer, Kaplan, & Ober, 1994), based on data previously collected as part of the Cape Town Child Development Longitudinal Cohort study (Jacobson et al., 2008). That is, while the HE children who were identified as exhibiting subtle facial dysmorphism similar to children with FAS (HE-dysmorphic) performed like those with FAS and PFAS on tests of IQ and learning, the remaining HE children whose facial features did not resemble those of the syndromal children (HE-nondysmorphic) performed similarly to controls. These differences in cognitive performance between HE children with FAS facial dysmorphism and

those without were reported in the Suttie et al. (2013) paper. It was noted that levels of PAE did not differ significantly between the subset of HE children who exhibited dysmorphism and those who did not. Furthermore, no differences were found for maternal age or prenatal smoking between the two HE subsets. There are, however, other factors that this particular study did not interrogate that could have also accounted for the varying effects of PAE in this sample of children. For example, Suttie and colleagues (2013) did not consider the pattern of drinking, maternal malnutrition, post-natal infections, or genetic and epigenetic differences. Nevertheless, these results provide evidence of the variability of outcomes within the HE category, even when the level of PAE is similar. Furthermore, these results suggest that the presence of even subtle facial dysmorphism may be a good indicator of cognitive performance, irrespective of the level of PAE.

Epidemiology

Today, the adverse effects of PAE on the developing foetus are recognized as a major public health concern. In fact, PAE is considered to be the leading cause of preventable developmental impairment worldwide (May & Gossage, 2011). Prevalence studies have been conducted in the United States to determine the rates of FAS, PFAS as well as the full spectrum of disorders (May et al., 2009, 2014, 2018). May and colleagues (2014) reported the prevalence of FAS to be between 6 and 9 per 1000 children, while the prevalence of PFAS was reported to be between 11 and 17 per 1000 children. When the full spectrum of effects described by FASD was considered, prevalence was reported to be between 24 and 48 per 1000 children (May et al., 2014). More recently, May and colleagues (2018) conducted a study to determine the prevalence of FASD across 4 US communities. The prevalence of FAS in these communities was estimated to be between 0 and 7.8 per 1000 children, whereas PFAS prevalence was estimated to be between 8.4 and 59.1 per 1000 children. When the full

spectrum of disorders was considered, prevalence was estimated to be between 31.1 and 98.5 per 1000 children (May et al., 2018).

The highest prevalence rates of FASD in the world have been documented in the Western Cape Province of South Africa. A number of epidemiological studies conducted in the Western Cape have reported exceptionally high rates of FASD (May et al., 2000, 2007, 2009, 2013, 2016, 2020; Viljoen et al., 2005). The most recent prevalence study, conducted by May and colleagues (2020) reported rates of FAS between 51.3 and 80.2 per 1000 children and rates of PFAS between 77.1 to 111.2 per 1000 children. Prevalence estimates considering the full spectrum of effects described by FASD reported rates of between 279.0 and 332.7 per 1000 children (May et al., 2020).

Historically, wine farms in this region distributed wine to farm workers as partial payment for their labour at the end of the work week (May et al., 2007). This practice, which was referred to as the "Dop" system, critically influenced the drinking patterns of the impoverished labour force, bringing about a form of institutionalized alcohol abuse and social control (London, 1999). It is argued that regular binge drinking, particularly over weekends, became a ritualized practice for farm workers during which they were able to experience a brief reprieve from their impoverished situation. While the Dop system has since been outlawed, the culture of frequent, heavy, and concentrated alcohol consumption remains, even in urban communities where the members do not work in the viticulture industry.

FASD in the South African Context

In South Africa, the burden of FASD weighs on a number state systems, including health, social services, educational, and criminal justice (Adnams, 2010). For school-aged children, this burden falls predominantly on the education system. However, current policies intended to cater for children with developmental, intellectual, or learning disabilities are often not properly implemented. Furthermore, while South Africa supports the concept of

inclusive education, in practice, the country's mainstream education system does not have the necessary resources to adequately cope with the special learning needs of children with FASD (Adnams, 2010).

It is also worthwhile to note that children and adults in low-resource countries, such as South Africa, often present with greater intellectual impairment when compared to those who live in better resourced countries (e.g. Lewis et al., 2015). This difference in performance is attributed to longstanding adverse influences such as poor nutrition, deprived home environment, and indeed, an inadequately resourced education environment (Adnams, 2017).

FASD and the Brain

The development of the CNS can be categorized into distinct stages during which PAE will likely result in abnormalities specific to that point in development (Guerri, 1998). During the first trimester of pregnancy, when the neural tube and crest are being formed, PAE can result in conditions such as microcephaly and hydrocephaly (for review see Roebuck, Mattson, & Riley, 1998). Alcohol exposure during the first trimester also gives rise to the facial dysmorphism that is characteristic of FAS (Sulik et al., 1981). During the second trimester, neuronal generation and migration are occurring (Miller, 1993). PAE at this time point can prompt the abnormal migration of cortical neurons. During the third trimester, alcohol exposure interferes with the process of synaptogenesis, resulting in neuroapoptosis (Farber et al., 2010; Ikonomidou et al., 2000). Any observed cognitive, behavioural, or psychomotor impairments are therefore the result of combinations of these alcohol-related structural and/or functional abnormalities produced at any one or at multiple time points during development (Guerri, 1998).

Neuroimaging studies of individuals with FASD have sought to determine which brain structures are most vulnerable to the effects of PAE. From the earliest studies in this

field, white matter has been demonstrated to be particularly vulnerable to the effects of PAE. Specifically, FASD is associated with a smaller corpus callosum area as well as alterations in the micro- and macro-structure of white matter throughout the brain (Archibald et al., 2001; Riley et al., 1995; Roebuck et al., 1998). Studies have also noted reduced overall cerebellar volume, as well as neuroanatomical differences in the frontal lobes, the basal ganglia, the hippocampus, and the amygdala in individuals with FASD (for a review see Spadoni, McGee, Fryer, & Riley, 2007). In addition to these structural differences, functional magnetic resonance imaging (fMRI) studies of FASD demonstrate that these individuals tend to activate alternate, or more extensive neural networks in order to perform tasks when compared to their unexposed peers (Meintjies et al., 2010). It is proposed that, when performing a given task, the recruitment of an alternate, or larger network serves to compensate for functional deficits in the network usually responsible for performing that task. Together, the structural and functional abnormalities of the brain resulting from PAE give rise to impairments in a wide range of cognitive and behavioural domains.

Cognitive Impairments in FASD

Individuals with FASD show impairments in multiple cognitive and behavioural domains (for a review, see Mattson, Bernes, & Doyle, 2019). Impairments in general intellectual functioning (IQ) are consistently reported as a consequence of PAE (Carmichael-Olson, Feldman, Streissguth, Sampson, & Bookstein, 1998; Carter et al., 2016; Jacobson et al., 2004; Mattson, Riley, Gramling, Delis, & Jones, 1997; Streissguth et al., 1991; Streissguth, Barr, Sampson, Darby, & Martin, 1989). In addition, individuals with FASD have been found to demonstrate impairments in attention (e.g. Burden, Jacobson, Sokol, & Jacobson, 2005; Coles, 2001; Mattson, Calarco, & Lang, 2006), verbal and non-verbal learning and memory (e.g. Carter et al., 2016; Lewis et al., 2015; Mattson & Riley, 1999), processing speed (e.g. Burden et al., 2005; Jacobson et al., 2004), visuospatial perception

(Doney et al., 2016), motor functioning (e.g. Hen-Herbst, Jirikowic, Hsu, & McCoy, 2020; Mattson et al., 1998), sensory processing (e.g. Fjeldsted & Xue, 2019; Hen-Herbst et al., 2020; Jirikowic, Olson, & Kartin, 2008), language (e.g. Hendricks, Malcolm-Smith, Adnams, Stein, & Donald, 2019; McGee, Bjorkquist, Riley, & Mattson, 2009; Wyper & Rasmussen, 2011), quantitative reasoning (e.g. Howell, Lynch, Platzman, Smith, & Coles, 2006; Rasmussen & Bisanz, 2009; Streissguth et al., 1994), and executive functioning, such as working memory, planning, inhibition, and goal setting (e.g. Burden et al., 2005; Carmichael-Olson et al., 1998; Fuglestad et al., 2015; Green et al., 2009; Khoury, Milligan, & Girard, 2015; Kodituwakku, Handmaker, Cutler, Weathersby, & Handmaker, 1995; Rasmussen, 2005). These impairments are a common result of PAE, irrespective of whether the individual presents with the full FAS phenotype (Mattson et al., 1998).

Secondary Difficulties

Much of the FASD-related research conducted to date has concentrated specifically on identifying the outcomes of PAE on various domains of cognitive functioning (Carmichael-Olson et al., 1998; Kodituwakku, 2009; Mattson et al., 2010, 2013; Mattson et al., 2011; Paley & O'Connor, 2007). However, individuals with FASD are frequently reported to also be at a greater risk for developing what the literature has termed 'secondary difficulties', including poor mental health, substance abuse problems, disruptive behaviour, problems with the law, and even inappropriate sexual behaviour (Kully-Martens, Denys, Treit, Tamana, & Rasmussen, 2012; Mattson et al., 2011; O'Connor & Paley, 2009; Paley, O'Connor, Kogan, & Findlay, 2005; Rasmussen, Mcauley, & Andrew, 2007; Streissguth et al., 1991). It is suggested that deficits in social-emotional functioning may be the primary source of many of these secondary difficulties (Greenbaum, Stevens, Nash, Koren, & Rovet, 2009; Kerns, Siklos, Baker, & Müller, 2016; Kodituwakku & Kodituwakku, 2014; McGee, Bjorkquist, Price, Mattson, & Riley, 2009; Schonfeld, Paley, Frankel, & O'Connor, 2006).

Despite the studies that have suggested this association, and the many anecdotal reports of social-emotional difficulties in the literature on FASD (Mattson & Riley, 2000; Nash et al., 2006), impairments relating to socio-emotional functioning in individuals with FASD have received relatively less attention in the research literature.

Social-Emotional Deficits in FASD

Children with FASD demonstrate significantly poorer social-emotional development compared to their non-exposed peers, and as a result, exhibit poorer social functioning and adaptive behaviour in comparison (Kully-Martens et al., 2012; Nash et al., 2006; Rasmussen et al., 2007; Thomas et al., 1998; Tsang et al., 2016). Both caregiver- and teacher-reports confirm that children with FASD demonstrate inappropriate social behaviours and difficulty in their interactions with peers (Kully-Martens et al., 2012; Paley et al., 2005; Rasmussen et al., 2007; Whaley et al., 2001). Research also indicates that these impairments in social competency in children with FASD are likely to become more challenging and prevalent with age as social demands increase (Whaley et al., 2001). Furthermore, the degree of impairment in the social-emotional domain appears to surpass what would be predicted given the degree of general intellectual impairment (McGee, Bjorkquist, Price, et al., 2009). Poor self-regulation, and specifically emotion regulation, has been proposed as the potential underlying mechanism resulting in these social-emotional deficits (Kochanska et al., 2001; Kopp, 1982; Schonfeld et al., 2006, 2009).

Self-Regulation

Broadly, self-regulation refers to the capacity to modulate emotion, attention, and behaviour in response to the demands of a particular situation or stimulus (Calkins, 2007; Kochanska, Coy, & Murray, 2001; McClelland & Cameron, 2012). Processes involving the management of emotions, focusing and shifting of attention, and activating or inhibiting behaviours are all subsumed under the term self-regulation. Successful self-regulation

depends on an individual's ability to co-ordinate these processes to respond adaptively to their environment (McClelland & Cameron, 2012). The capacity to voluntarily separate one's behavioural response from its associated immediate emotional impulse can thus be attributed to self-regulation (Eisenberg et al., 2004; Kopp, 1982).

Calkins and Fox (2002) propose a multilevel approach to the conceptualization of self-regulation, as well as to explaining the development of self-regulatory skills in early childhood. According to this model, self-regulation comprises multiple domains of regulatory skills; and the development of skills belonging to each domain follow a particular trajectory. That is, skills relating to physiological regulation are the first to develop, which in turn provide the foundation for the development of attentional regulation. The capacity to regulate attention paves the way for the development of skills relating to emotion regulation, which then allow for the development of behavioural regulation. The current research project focuses specifically on the domain of emotion regulation.

Emotion Regulation

Emotion regulation, a sub-domain of self-regulation, refers to an individual's ability to use various tactics to control and alter the intensity and duration of emotional responses in order to achieve a prioritized objective (Eisenberg et al., 2001; Gross, 1998). Emotion regulation also serves to enable an individual to adapt appropriately to their social environment (Campos et al., 2011; Thompson, 2011). Zeman and colleagues (2006) add that the process of emotion regulation relies on the organization and management of a number of different components, including internal systems such as cognition and neurophysiology, behavioural components such as facial expressions and actions, and social components such as social context, cultural values, and personal incentives. The ability of an individual to organize and manage these various components determines whether they are able to achieve their objectives in a socially appropriate manner.

One method of studying emotion regulation is to examine the strategies that an individual employs to regulate their emotions. The literature has generally delineated between strategies that are adaptive, and those that are maladaptive (Bridges et al., 2004). An emotion regulation strategy is considered adaptive if it enables an individual to successfully function in their environment. On the other hand, maladaptive emotion regulation strategies are those that are not sufficiently flexible and thus prevent an individual from functioning adaptively in their environment. It is important to note here that emotion regulation strategies are not inherently adaptive or maladaptive (Thompson, 2011). Rather, the adaptivity of a given emotion regulation strategy is determined relative to an individual's goals and the social context in which they find themselves. That being said, some strategies have been found to be consistently associated with adaptive (e.g. reappraisal) or maladaptive (e.g. affective suppression) outcomes in a variety on contexts (for a review, see Aldao, Nolen-Hoeksema, & Schweizer, 2010).

Sequelae of Emotion Regulation. The importance of adaptive emotion regulation as a predictor of social functioning and psychological adjustment has been highlighted in several studies of typically developing children (Blair, 2002; Woodward et al., 2017). For example, children who are better at regulating their emotions tend to perform better academically, demonstrate higher self-esteem, and are better able to cope with stress (Carlson & Wang, 2007; Kochanska & Aksan, 2006; Montroy et al., 2014). Emotion regulation skills are also crucial for managing the often-competing demands during interpersonal situations and accomplishing objectives in a socially appropriate manner (Blair, 2002). For this reason, those who are better at regulating their emotions generally are able to foster better relationships with their peers.

On the other hand, poor regulators have been found to present with more internalizing and externalizing symptoms, such as impulsivity, irritability, and negative affectivity

(Eisenberg et al., 2001, 2017; Eisenberg, Spinrad, & Eggum, 2010; Olson, Choe, & Sameroff, 2017; Sanghag, Nordling, Yoon, Boldt, Kochanska, 2013). As a result, children who are poor regulators are more likely to socially isolate themselves or demonstrate socially inappropriate behaviour (Calkins, 1994; McClelland & Cameron, 2012), as well as break the law and abuse substances at a later age (Woodward et al., 2017).

Correlates of Emotion Regulation. Adolescence is a particularly crucial period in the development of emotion regulation abilities. Adolescents tend to encounter more of the difficulties of daily life, experience fewer positive emotions and more negative emotions than in childhood (Larson & Ham, 1993). Adolescents also tend to experience greater mood variability (Maciejewski et al., 2015). However, although adolescence has been highlighted as a transformative period in the development of emotion regulation, there are still considerable between- and within-individual differences in the timing of this development (Zimmermann & Iwanski, 2018). For instance, Zimmerman and Iwanski (2014) investigated the development of 7 distinct emotion regulation strategies in relation to 3 different emotions – sadness, anger, and fear. Their findings demonstrated that only 2 strategies, *adaptive emotion regulation* and *social support-seeking*, showed consistent age-related development across the 3 emotions. Specifically, they found that while adolescents tend to utilize these strategies more between the ages of 12-14, the use of these strategies declines toward mid-adolescence, reaching a low point between the ages of 15-17, and then begins to increase again towards late adolescence and early adulthood, from the age of 18 and above (Zimmermann & Iwanski, 2014). Thus, the development of both *adaptive emotion regulation* and *social support seeking* follows a more U-shaped, rather than a linear trend line. For the remaining 5 emotion regulation strategies (*passivity, avoidance, expressive suppression, dysfunctional rumination,* and *dysregulation*), however, the age-related developmental

trajectory is more complex as the increases and decreases in their usage over the adolescent period is emotion-specific (Zimmermann & Iwanski, 2014).

It has been postulated that there are also sex differences in the development of emotion regulation. For instance, females have been found to be significantly more likely to use the maladaptive strategy of rumination, which has been linked to increased mood lability (Sanchis-Sanchis et al., 2020; Zimmermann & Iwanski, 2014). Females are also more likely to use the strategy of reappraisal as well as externally oriented emotion regulation strategies such as emotional expression and social support seeking (Sanchis-Sanchis et al., 2020; Zimmermann & Iwanski, 2014; Zlomke & Hahn, 2010). Males, on the other hand, have been found to be more likely to use strategies such as affect suppression, passivity, and avoidance (Zimmermann & Iwanski, 2014).

The development of emotion regulation is particularly influenced by the family environment. As such, another variable that is proposed to impact an individual's ability to regulate their emotions is their family's socio-economic status (SES). Low SES families have a low household income, and a low parental education level. As a result of their low SES, these families are also often exposed to a number of external obstacles, such as poor healthcare and educational services, turbulent home environments and dangerous neighbourhoods (Holmes et al., 2019). Indeed, studies have consistently shown an association between low SES and an impaired development of, and ability to regulate emotions (Herd et al., 2020; Holmes et al., 2019). These studies suggest that the circumstances and stressors that are described by the term low SES may translate into a stressful home environment, which in turn negatively impacts the development of emotion regulation abilities.

Prenatal cigarette exposure is another variable shown to be associated with impaired emotion regulation. For instance, infants with prenatal cigarette exposure are reported to present neurobehavioural deficits such as decreased self-regulation. Such impairments often

manifest as irritability, and negative affect in infancy (Froggatt et al., 2020). To my knowledge, no literature currently exists on the association between prenatal cigarette exposure and emotion regulation later in life. However, the literature on cognitive control suggests that the regulatory difficulties observed in infancy continue into early childhood (Huijbregts et al., 2008; Mezzacappa et al., 2011).

Finally, very few studies have examined the association between IQ and emotion regulation; the studies that have, have generally done so among clinical populations and have yielded mixed results. For instance, in a study conducted by Zantinge, van Rijn, Stockmann, and Swaab (2017), young children with autism spectrum disorder (M_{age}=5 years) were observed and their behaviours coded as they performed a frustrating task. The study found that a higher IQ was associated with more adaptive emotion regulation strategies. Other studies, however, have found emotion regulation to be relatively independent of IQ (Berkovits et al., 2017; Graziano et al., 2007; Sobanski et al., 2010). However, the samples in the latter studies did not exhibit the wide range of IQ scores often observed among individuals with FASD. As such, it is possible that IQ may nevertheless be an important factor influencing the development of emotion regulation.

Emotion Regulation in FASD

In the recently proposed diagnostic criteria for neurodevelopmental disorder associated with PAE (ND-PAE) outlined in the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition,* (DSM-5), emotion regulation, specifically, is recognised as a major area of impairment (APA, 2013). Specifically, the DSM-5 refers to the manifestation of mood lability, impulsivity, negative affect and irritability in children with PAE. Indeed, clinical observations of individuals with FASD suggest that they demonstrate significant impairment in emotion regulation (for a review see Temple, Cook, Unsworth, Rajani, & Mela, 2019). These emotion regulation impairments are reportedly found in individuals

across the full FASD spectrum, including in individuals who do not present with the facial dysmorphology associated with the more severe phenotypes (i.e., FAS and PFAS).

Research has demonstrated that PAE-related problems in emotion regulation begin to manifest already in infancy, with infants with PAE exhibiting poor sleep regulation, greater stress reactivity, and a more difficult temperament than their typically developing counterparts (Alvik et al., 2011; Haley et al., 2006; Jirikowic et al., 2016). These difficulties in emotion regulation are therefore one of the first observable signs of PAE.

Studies conducted with children and adolescents with FASD also suggest significant impairments in emotion regulation. For instance, children with FASD have been reported to demonstrate a number of behavioural problems. Both caregiver and teacher reports indicate that children with PAE frequently demonstrate negative affectivity, impulsivity, irritability, temper tantrums and general mood lability (Carmichael-Olson et al., 1992, 2007; McDougall et al., 2020; Paley et al., 2005). They also reportedly experience greater distress and anger and tend to engage negatively with their peers. Individuals with FASD are also reported to have higher rates of externalizing disorders, including attention deficit/hyperactivity disorder (ADHD), conduct disorder (CD), and oppositional defiant disorder (ODD), as well as higher rates of internalizing disorders, such as depression and anxiety (Fryer, McGee, Matt, Riley, & Mattson, 2007; Khoury, Jamieson, & Milligan, 2018; O'Connor & Kasari, 2000; O'Connor et al., 2002). These reports of behavioural problems and psychopathology in individuals with FASD provide a good, albeit indirect, indication of problems with emotion regulation.

However, the topic of emotion regulation has seldom been interrogated directly (i.e., with a questionnaire or task specifically designed to examine emotion regulation strategies) in children or adolescents with PAE. Rather, the presence of emotion regulation impairments has been inferred given the mood symptoms and the high rate of psychopathology observed in individuals with PAE. However Shields and Cicchetti (1997) argue for the importance of

examining the *processes* of emotion regulation directly. To do so accurately, they suggest that it is necessary to consider the intensity and valence of an individual's emotional expression, as well as the flexibility and situational appropriateness of the strategies employed to regulate their emotional expression.

Very few studies have sought to examine these emotion regulation processes directly in the context of FASD. Of those that have, some have made use of the behaviour rating inventory of executive function (BRIEF; Gioia, Isquith, Guy, & Kenworthy, 2000). The behavioural regulation index (BRI), a subscale of the BRIEF, specifically examines skills related to the regulation of emotions and behaviours. In these previous studies, individuals with FASD have been found to score in the clinical range for the BRI, indicating a significant deficit in the ability to regulate (Rasmussen et al., 2007; Schonfeld et al., 2006). It is interesting to note though, that neither Rasmussen et al. (2007) nor Schonfeld et al. (2006) found a significant difference in BRI scores between FAS, PFAS, and ARND groups. This provides further evidence for the fact that, as previously mentioned, individuals along the FASD spectrum demonstrate a comparable amount of impairment in their ability to regulate their emotions. In addition, one intervention study has made use of a questionnaire interrogating emotion regulation processes directly in a sample of 30 children (aged 4-8) with PAE (Petrenko et al., 2017). This study made use of the caregiver-report version of the Emotion Regulation Checklist (ERC; Shields & Cicchetti, 1997). The ERC was administered before commencing an intervention, and then again post-intervention. Pre-intervention scores for both the *Lability/Negativity* and the *Adaptive Emotion Regulation* subscales were suggestive of poor emotion regulation. While the aforementioned studies (Petrenko et al., 2017; Rasmussen et al., 2007; Schonfeld et al., 2006) suggest that individuals with FASD present with significant impairment in relation to the processes they use to regulate their emotions, an important limitation of these studies is that they did not include an unexposed,

control group in their studies. As a result, they were unable to make a comparison between individuals with FASD and individuals who were unexposed. These studies could therefore not rule out the potential influence of other variables, such as IQ or SES on their participants' ability to regulate emotions. Further research interrogating the processes relating to emotion regulation in FASD is therefore necessary.

While the use of a measure assessing emotion regulation processes directly is advised, doing so via self-report may be difficult in children and adolescents with FASD as they may not be equipped with the necessary insight to be able to be sufficiently introspective on issues related to the regulation of emotions. In addition, given that PAE has been known to impact various language domains (Hendricks et al., 2019), the individual may lack the language skills required to express their shortcomings (Temple et al., 2019). As such, caregiver reports of the child's ability to regulate emotions may be more informative.

Development of Emotion Regulation

The nascent ability to regulate one's emotions is considered a major developmental milestone during the first 3 years of childhood (Calkins, 2007; Carlson & Wang, 2007; Kochanska et al., 2000). During these 3 years, children undergo several important changes in their ability to understand emotions (Calkins, 1994; Kopp, 1982).

Their ability to analyse the more complex systems within emotion-generating situations improves. As a result, they are better able to understand the causes and consequences of emotions. They also begin to explore the different ways to express a given emotion (Stegge & Meerum Terwogt, 2007). This greater emotional understanding and awareness is coupled with greater independent emotion regulation.

Research suggests that early emotion regulatory efforts are controlled largely by instinctive physiological mechanisms (Kopp, 1982; Posner & Rothbart, 2000). Before 3 months of age, these efforts manifest as approach and withdrawal behaviours when

confronted with pleasant and aversive stimuli, respectively. By 3 months of age, the infant has progressed towards more complex mechanisms of reacting to stimuli, such as reflexive signalling (e.g. crying), basic motor movements (e.g. moving away) and self-soothing (e.g. sucking). A major shift in the development of the infant can be observed between 3 and 6 months. During this period, the infant's ability to modify their own arousal levels by performing simple actions begins to emerge (Kochanska et al., 2001; Rothbart et al., 1992). To a large extent, this ability is reliant on the development of simple motor skills and attentional mechanisms, as well as the ability to coordinate them in an effort to engage and disengage attention when necessary. Essentially, this means that the infant is now able to self-distract in order to move their attention away from the source of the negative arousal.

By the age of 1, the infant's attempts to control their arousal is more active and more purposeful (Kopp, 1982). For instance, their attempts to signal and redirect attention become explicitly social as they begin to understand that their caregivers may be able to assist them in regulating their affective state. By the age of 2, the shift from passive to more active, socially directed forms of emotion regulation is complete (Rothbart et al., 1992).

However, while the various emotional, behavioural and cognitive processes involved in emotion regulation emerge in infancy, the ability to draw on and co-ordinate these processes shows rapid development between the ages of 3 and 7 years (McClelland & Cameron, 2012; Montroy et al., 2016). Although the development of emotion regulatory skills follows a somewhat predictable trajectory, there exists variability in the manner by which infants learn to regulate their state (Calkins, 1994). While some caregivers may communicate directly to their children about emotions and strategies they can use to successfully modulate their emotional responses, some children may simply learn through observing how their caregivers express and regulate their own emotions (Denham et al.,

1991). Variability also exists in the strategies a child may apply to a certain situation given the success of previous outcomes.

This learning process begins in infancy, through repeated experiences of regulation guided by the primary caregiver. Over time, the type of emotional expression and regulation practiced by a child's caregiver gradually becomes internalized by the child, and the abilities acquired are incorporated into their own repertoire of emotion regulation skills (Calkins & Fox, 2002). Research suggests that the development of adaptive emotion regulation is supported by caregiving that is consistently sensitive and predictably responsive, such as that provided in a secure attachment relationship (Sameroff, 2010).

Attachment

The concept of attachment was introduced by British psychoanalyst John Bowlby (1969). Through his investigation into children's early interactions with their caregivers, Bowlby became interested in how these interactions went on to shape the child's development. He hypothesized that a child's initial interactions with a caregiver were considerably important in determining not only the course of their personality development, but also their future interpersonal relationships (Ainsworth & Bowlby, 1991). The child's experiences within these interactions would then, in turn, lead the child to develop generalized beliefs and expectations about the self, relationships, and the world. Bowlby (1980) called these generalized beliefs and expectations 'working models'. While these working models are thought to be persistent across the lifespan, experiences throughout the lifespan may prompt them to be revised.

Attachment to a caregiver is evidenced by certain behaviours that are enacted by an infant when he/she experiences feelings of discomfort (Bowlby, 1969). For example, the infant may enact a range of proximity- and contact-seeking attachment behaviours to gain and maintain proximity to the object of attachment during times of stress. These include

following, approaching, and clinging, as well as signalling behaviours such as calling, smiling and crying (Ainsworth & Bell, 1970). The intensity of these behaviours may be increased or reduced depending on the situation. Although attachment behaviours are initially directed indiscriminately, at the age of approximately 6 months, infants direct these behaviours exclusively to the attachment figure with the purpose of eliciting a response (Ainsworth, 1989).

The concept of security is a vital aspect of attachment theory (Waters & Cummings, 2000). Security within the attachment relationship is promoted when the caregiver is consistently responsive and appropriately sensitive to the infant's needs (Ainsworth, 1979; Ainsworth & Bell, 1970). An infant who experiences such caregiving is confident that their caregiver is trustworthy and can be used as a secure base from which the infant can engage in exploration of their environment. By engaging in exploration of the environment, the infant begins to gain social competence and independence (Malekpour, 2007).

The assessment of infant attachment, as pioneered by Ainsworth and colleagues (1979), involves the classification of infants according to the perceived security of their attachment to the primary caregiver, in most cases the mother, by observing their behaviour during a *Strange Situation* paradigm (SSP). The SSP comprises of a series of eight 3-minute episodes which include brief separations from the primary caregiver, and subsequent reunions with her/him (Ainsworth & Bell, 1970). The procedure is designed to activate the attachment system and stimulate the performance of attachment behaviours toward the caregiver (Ainsworth, 1985). During this procedure, coders are trained to observe infant behaviour toward the stranger and caregiver, with particular focus on the infant's reactions to the separations and reunions, and their exploration of the environment throughout all episodes (Ainsworth & Bell, 1970). The infant's attachment relationship with the caregiver is then classified according to the specific combination of behaviours demonstrated by the

infant. Ainsworth's initial studies (Ainsworth, 1964, 1979) identified one distinct pattern of organised secure attachment behaviour - secure (B), as well as two patterns of organised insecure attachment behaviour - avoidant (A), resistant (C).

However, after reviewing approximately 200 videotapes of infants from multiple different samples, it was determined that some infants could not be suitably classified into one of the 3 existing organised attachment categories (Main & Solomon, 1986). Specifically, studies on infants who had been abused or neglected seemed to suggest that these infants demonstrated no single organised strategy to cope with the SSP. Previously, these infants had been 'forced' into the secure category. In order to address this apparent limitation of the original Ainsworth system of attachment classification (1978), Main and Solomon (1986) developed a third classification of insecure attachment – disorganised attachment.

Infants considered to be securely attached (B) are those who successfully use their primary caregiver as a secure base from which to explore their environment. Upon separation from the primary caregiver, they demonstrate distress and any prior exploration diminishes. However, once reunited, they seek proximity and comfort from the caregiver. This comfort is successful in soothing the infant, thus allowing him/her to return to exploration. (Ainsworth, 1979). There are two types of organised insecure attachment: avoidant attachment and resistant attachment. Avoidant (A) infants rarely demonstrate overt distress upon separation from the caregiver. However, studies have shown that avoidant infants nevertheless demonstrate physiological signs of distress, such as high levels of salivary α-amylase (an indication of sympathetic arousal), during this separation period (Hill-Soderlund et al., 2008). Upon reunion, these infants are likely to avoid the caregiver, sometimes disregarding their presence altogether. Infants classified as resistant (C) often cling to their caregiver and are reluctant to explore the environment. Upon separation, they demonstrate intense distress. When the caregiver returns, resistant infants behave ambivalently toward the caregiver, both

seeking proximity and resisting interaction with her simultaneously. When proximity to the caregiver is obtained, the resistant infant is not easily soothed as comfort provided by the caregiver is not necessarily effective. Infants classified as disorganised (D) are those who are insecurely attached but do not demonstrate an organised pattern of attachment behaviour toward their caregiver within the SSP. Disorganised infants tend to demonstrate approach, as well as fearful and avoidant behaviours that are followed, or interrupted, by contradictory behaviours (Main & Solomon, 1986). For example, although the disorganised infant may exhibit excessive distress during the separation episode, he/she may demonstrate indifference towards the caregiver upon her return in the reunion episode (Ijzendoorn et al., 1999). The disorganised infant may also show signs of misdirected, stereotypical and even freezing behaviour. For instance, the infant's behaviour may be misdirected toward the stranger rather than the caregiver upon reunion. The infant may also display stereotypical behaviour such as hair pulling in the context of separation stress. This is often accompanied by a dazed expression and is performed even when the caregiver is available. On the other hand, freezing occurs when the infant cannot decide between seeking proximity or avoiding the caregiver and thus ceases to move at all, as if he/she has dissociated from the present moment (Main & Morgan, 1996).

Sequelae of Attachment

A sense of security with one's attachment figure can be thought of as a resilience resource and a promoter of social adjustment and overall mental health (Mikulincer & Shaver, 2019). A large body of research has consistently demonstrated an association between attachment security and developmental outcomes. Specifically, individuals presenting with secure attachment demonstrate more positive emotions as well as fewer negative emotions, fewer behavioural problems, and a lower incidence of affective disorders (Groh et al., 2017; Kochanska, 2001; Kochanska & Kim, 2013; Sroufe, 2005). Secure

attachment has also been found to be associated with better social competence (Groh et al., 2014).

On the other hand, disturbances in this sense of security may encourage negative emotions to be more frequently activated or suppressed. This, in turn, can be a risk factor for social and emotional problems as well as psychopathology. Insecure attachment has been found to be associated with increased internalizing and externalizing behavioural problems, social and emotional withdrawal, as well as poorer peer relationships (Fearon, Bakermans-kranenburg, van IJzendoorn, Lapsley, & Roisman, 2010; Groh, Roisman, van Ijzendoorn, Bakermans-Kranenburg, & Fearon, 2012; Waters, Merrick, Treboux, Crowell, & Albersheim, 2000). Some studies have also determined that children with insecure attachment have difficulty recognizing emotions, which likely contributes to their diminished social competence (Laible & Thompson, 1998; Steele, Steele, & Croft, 2008). An association has also been reported between insecure attachment patterns and psychopathology (Sroufe et al., 1999; Ward et al., 2006; Weinfield et al., 2008). This prediction to psychopathology is particularly seen for individuals who exhibit a disorganised attachment pattern, especially in the presence of other risk factors (e.g., low IQ; Fonagy & Target, 2002; Ijzendoorn et al., 1999).

Attachment in FASD

Despite the vast amount of research detailing the neurodevelopmental impairments associated with PAE, to my knowledge, only four studies have examined attachment relationships in the context of PAE (Bergin & McCollough, 2009; Hay, Jacobson, Molteno, Viljoen, & Jacobson, 2004; O'Connor, Sigman, & Brill, 1987; O'Connor, Kogan, & Findlay, 2002). O'Connor and colleagues (1987) were the first to examine the relation between PAE and infant attachment. In this study, 46 mother-infant dyads were administered the SSP when the infants were 12 months old. Importantly, their sample was recruited from a low-risk

population. That is, the mothers, who were from the United States, were predominantly white (92%), middle class (100%), married (98%) and highly educated (72% of the mothers were college graduates). Alcohol consumption pre-, during, and post-pregnancy was estimated using self-report questionnaires. Mother-infant dyads were then divided into 3 groups based on the amount of absolute alcohol (AA) they consumed per day: 1) abstinent-light (< 0.1 oz. AA/day), 2) light-moderate (between 0.11 and 0.99 oz. AA/day), and 3) moderate-heavy (> 1.0 oz. AA/day). When all 3 groups were included in the analyses, no significant between-group differences were observed for attachment security. However, when the infants of abstinent-light drinkers were compared to all mothers who consumed more (> 0.1 oz. AA/day) an effect for attachment security emerged. While only 35% of infants born to abstinent-light drinkers were classified as insecure, 70% of those born to mothers who were classified as moderate-heavy drinkers were considered to be insecure. Interestingly, O'Connor et al. (1987) also found the rate of disorganised attachment to be especially sensitive to the impact of PAE, with the rate of disorganised attachment being significantly different between abstinent-light drinkers and moderate to heavy drinkers. It is important to note here that post-pregnancy drinking was not found to be associated with the mother-infant attachment relationship. This suggests that the group differences in infant attachment is due to PAE rather than to the post-natal environment.

A later study conducted by O'Connor and colleagues (2002) thus sought to include participants from a more high-risk population. The majority of the mother-child dyads that participated in this study came from poor communities and the mothers were often single parents. This study sought to examine the relationship between PAE and attachment, in 4- to 5-year-old children. In this study, mother-child dyads were divided into 2 groups based on the amount of alcohol consumed per occasion: 1) abstinent-light drinkers (1 or fewer drinks/occasion), and 2) moderate-heavy drinkers (2 or more drinks per occasion). Security

of attachment was measured using the Attachment Q-set (Waters, 1995). A criterion score was used to analyse the Q-set, with a score of ≥ 0.37 indicating secure attachment, and < 0.37 indicating insecure attachment. The results demonstrated a significant association between PAE and attachment security. While 36% of children born to mothers who were abstinent or light drinkers were classified as insecure, 80% of the children born to mothers who were moderate-heavy drinkers were considered insecure. Once again, the relationship between post-pregnancy drinking and attachment was not found to be significant. It is important to note, however, that this study made use of retrospective reports of alcohol use during pregnancy, which may not be as accurate as reports obtained at the time of the pregnancy.

To my knowledge, since these first two seminal studies were conducted, only two additional studies have been done. A study conducted with the Cape Town Longitudinal Cohort (from which the sample and attachment data for the current study comes) used the SSP to investigate the relation between PAE and attachment in 115 high-risk mother-infant dyads, some of which were diagnosed with FAS (Hay et al., 2004). In this study, reports of maternal alcohol consumption were obtained during pregnancy. Hay and colleagues (2004) determined that PAE was associated with a significantly lower rate of secure attachment and that this association was mediated by the social environment. Indicators of the social environment in this study included SES, breastfeeding, maternal education and IQ, and maternal depression (Hay et al., 2004). The study also found that PAE was associated with increased rates of disorganised attachment. The association with disorganised attachment remained significant, even after controlling for social environment. Once again, post-pregnancy alcohol consumption was not found to be related to attachment. The study also reported that the rate of insecure and rate of disorganised attachment was related to severity of FASD diagnosis (Jacobson & Jacobson, 2013).

Another study conducted by Bergin and McCollough (2009) compared 35 low income mothers and their substance exposed (alcohol as well as tobacco, marijuana, and cocaine) infants to a non-exposed group, case-matched according to certain risk factors, such as maternal age, gestational age, and birth weight. The Attachment Q-set was used to assess attachment at 12 months of age. This study also considered the role of the quality of caregiving, which was assessed during a 2-hour videotaped interaction between mother and infant. When accounting for the influence of maternal sensitivity and involvement, it was found that substance exposure did not predict insecure attachment. However, in this study, all alcohol-exposed infants were grouped together, irrespective of level of exposure, and compared to unexposed infants. It is thus possible that the authors may not have been able to detect an association between heavier exposure and insecure attachment.

It is also worthwhile to note that in some of the studies detailed above, information is only provided regarding the mother's alcohol consumption during pregnancy (Bergin & McCollough, 2009; O'Connor et al., 1987; O'Connor et al., 2002). That is, attachment was studied in relation to continuous measures of PAE rather than to the FASD diagnostic categories. The one exception to this is Hay et al. (2004)'s findings showing that infants with FAS showed a higher rate of insecure and disorganised attachment compared with the PFAS, HE, and control groups (Jacobson & Jacobson, 2013). That is, infants with FAS showed a higher rate of disorganised attachment compared with the PFAS, HE, and control groups (Jacobson & Jacobson, 2013). Given Suttie et al. (2013)'s findings based on this same cohort that the presence of even subtle facial dysmorphism may be a good indicator of cognitive performance in individuals with FASD, the current study sought to explore whether differences in infant attachment security and organisation could also be found by comparing individuals with subtle facial dysmorphism and those without.

The Association Between Attachment and Emotion Regulation

Whereas no research currently exists examining the association between attachment and emotion regulation in the context of PAE or FASD, research exploring this relation in typically developing populations proposes that secure attachment is instrumental for the formation of adaptive emotion regulation (Cassidy, 1994; Kopp, 1982; Mikulincer et al., 2003; Schore, 2005; Sroufe, 2005). In fact, it is proposed that attachment is the foundation on which the capacity for emotion regulation develops (Schore, 2005; Sroufe, 2000). So, regulatory processes that are practiced initially within the context of attachment through caregiver-guided co-regulation are then gradually internalized over the period of early childhood and generalized to situations beyond the dyadic relationship (Fonagy & Target, 2002).

The notion of this relation between attachment patterns and emotion regulation competency has been supported by numerous empirical studies, both cross-sectionally and longitudinally. Significant associations between attachment patterns and emotion regulation performance have been found in the toddler/preschool years (Bosquet & Egeland, 2006; Brumariu & Kerns, 2013; Gilliom et al., 2002; NICHD Early Child Care Research, 2004; Vondra et al., 2001), the school age years (Borelli et al., 2010; Brumariu, Kerns, & Seibert, 2012; Heylen et al., 2017; Kerns, Abraham, Schlegelmilch, & Morgan, 2007), and from adolescence into adulthood (Girme et al., 2020; Hershenberg et al., 2011; Kobak et al., 1993; Scharf et al., 2004; Zimmermann, 1999).

This proposed relations between attachment patterns and emotion regulation is further supported by research showing that the development of the neural mechanisms involved in the process of emotion regulation is dependent on early experiences (Lemche et al., 2006; Moutsiana et al., 2014; Schore, 1996). For example, in a cross-sectional study conducted by Lemche and colleagues (2006), individuals who reported attachment avoidance or anxiety

demonstrated increased amygdala activation in reaction to stress. Furthermore, a prospective longitudinal study conducted by Moutsiana and colleagues (2014) determined that infants who were classified as insecurely attached in the SSP at 18 months showed greater neural activation in prefrontal regions in response to emotional stimuli at 22 years of age when compared to those who had been classified as securely attached. The attachment relationship is therefore essential in facilitating the development of the brain's regulatory pathways.

Consistent with this developmental trajectory, the style of emotion regulation employed by individuals with specific attachment patterns is shaped by their expectations of how their caregiver will respond to their emotions (Schore, 2005). That is, securely attached children expect their caregiver to demonstrate sensitivity and be supportive in helping them deal effectively with their emotions. As a result, they learn to express emotions openly and are not hesitant to seek comfort when distressed. In this way, securely attached children are provided with the 'tools' to develop adaptive and flexible emotion regulation strategies (Moutsiana et al., 2014). In contrast, insecurely attached children expect their caregiver to be inconsistently supportive or dismissive of their emotional expression. For instance, avoidantly attached children, who expect their caregivers to be dismissive and withhold support and comfort, are likely to minimize their expression of emotions (Cassidy, 1994). Resistantly attached children, who expect their caregiver to respond inconsistently to their distress, are likely to exaggerate their expression of emotion. Thus, insecurely attached children learn maladaptive emotion regulation strategies consistent with how they expect their caregivers, and later, society to respond (Mikulincer & Shaver, 2019).

Rationale, Aims, and Hypotheses

Infants with PAE/FASD have been reported to exhibit higher rates of insecure attachment patterns (Bergin & McCollough, 2009; Hay et al., 2004; O'Connor et al., 2002), as well as higher rates of disorganised attachment (O'Connor et al., 1987; Jacobson &

Jacobson, 2013). However, none of these studies have examined the association between attachment pattern and FAS-dysmorphism. The current study, therefore, aimed to examine the association between FAS-dysmorphism and insecure and disorganised attachment.

Behavioural, social, and emotional difficulties often noted in individuals with FASD have been suggested to stem from an impaired ability to successfully regulate their emotions (for a review, see Temple et al., 2019; Tsang et al., 2016). Given that the attachment relationship to the caregiver is thought to be the basis on which emotion regulation develops, a number of studies have examined the relation between attachment patterns and emotion regulation abilities in typically developing (TD) individuals (for a review, see Zimmer-Gembeck et al., 2017). Previous research conducted in TD populations provides empirical evidence for an association between a secure attachment pattern and the use of more adaptive emotion regulation strategies, while insecure attachment patterns have been associated with the use of more maladaptive regulation strategies (e.g. Borelli et al., 2010; Girme et al., 2020; Hershenberg et al., 2011; Lemche et al., 2006; Moutsiana et al., 2014; NICHD Early Child Care Research, 2004). However, to date, the relation between attachment and emotion regulation has yet to be examined in individuals with FASD. This is noteworthy given that, as noted above, individuals with PAE/FASD have been found to exhibit both higher levels of insecure attachment and disorganisation, as well as impaired emotion regulation. The current study, therefore, also sought to examine the unique predictive effect of FAS-dysmorphism on emotion regulation after controlling for the effects of attachment security.

Previous research has suggested that, even for individuals who do not present with the full phenotype characteristics of FAS or PFAS, the presence of even subtle facial dysmorphism can be a good indicator of cognitive performance. As previously described, Suttie et al. (2013) reported that individuals in the HE (highly exposed non-syndromal) group from our cohort who did not present with facial features similar to controls (HE-

nondysmorphic) performed significantly better on tests of cognitive performance than those in the HE group who did present with facial dysmorphism consistent with FAS (HE-dysmorphic). That is, individuals in the HE-dysmorphic group performed similarly to FAS and PFAS groups, while individuals in the HE-nondysmorphic group performed similarly to controls. The current study, therefore, sought to cluster those considered HE-dysmorphic with those with FAS and PFAS, while clustering those considered HE-nondysmorphic with controls. Thus, I sought to differentiate between and compare individuals with FAS-dysmorphism+ and those with FAS- dysmorphism- on attachment and emotion regulation.

The objectives of the current study were three-fold. First, it aimed to determine whether infants with FAS-dysmorphism+ exhibit higher rates of insecure attachment patterns. Second, it aimed to determine whether infants with FAS-dysmorphism+ would exhibit higher rates of disorganised attachment. Third, the study aimed to determine the extent to which FAS-dysmorphism was associated with emotion regulation in adolescence when controlling for attachment security in infancy as well as other potential predictors. The study, therefore, tested the following hypotheses:

1. Children with FAS-related facial dysmorphism will have had a higher rate of insecure attachment patterns during infancy in comparison to children without the FAS-related facial dysmorphism.
2. Children with FAS-related facial dysmorphism will have a higher rate of the disorganised attachment pattern during infancy in comparison to children without the FAS-related facial dysmorphism.
3. Children with FAS-related facial dysmorphism will demonstrate poorer caregiver-reported emotion regulation in adolescence in comparison to those without FAS-related facial dysmorphism, even after controlling for attachment security in infancy, as well as other potential confounders.

Method

Design and Setting

This study forms part of the research being conducted on the prospective Cape Town Longitudinal Cohort, ongoing since 1999 (Jacobson et al., 2008). This specific study employed aspects of both quasi-experimental and relational design in order to investigate group (FAS-dysmorphism+ vs. FAS-dysmorphism-) differences with regards to attachment classification, as well as emotion regulation. Data were collected in infancy at 13 months (Hay et al., 2004) and again during adolescence (Mean age = 17.66 years; SD = .70). All data were collected at the Child Development Research Laboratory (CDRL) at the University of Cape Town Health Sciences Campus. All examiners were blind to the participant's PAE history and FASD diagnosis.

Participants

Recruitment of the Cape Town Longitudinal Cohort

Between July 1999 and January 2002, 227 pregnant women were recruited from two obstetrical clinics at their first visit to the antenatal clinic (Jacobson et al., 2008). The two obstetrical clinics were identified because they serve an economically disadvantaged community with a high incidence of heavy maternal alcohol consumption during pregnancy (Croxford & Viljoen, 1999). Alcohol consumption during pregnancy was initially assessed using a screening interview administered at the antenatal clinics by the study's research nurse (Jacobson et al., 2008). Thereafter, mothers were invited to partake in the study based on certain inclusion criteria outlined below.

Maternal Alcohol Consumption. A 2-week timeline follow-back interview was used to determine maternal alcohol consumption prospectively during pregnancy (Jacobson & Jacobson, 2002). Upon recruitment into the study, each mother was interviewed concerning frequency and amount of drinking during pregnancy (Jacobson et al., 2008). Mothers were

asked about whether their drinking behaviours had changed since conception, when that change had happened, and how much they drank on a day-to-day basis during the previous 2 weeks. The interview also obtained detailed information about the type of beverage consumed and container size (for which pictures of different containers, bottles, cans, and glass size were used; Jacobson et al., 2008). Questions about sharing (i.e., size of container shared by how many women) were also included in the interview. This information was used in the calculation of standard drinks and then converted to oz absolute alcohol (AA; 2 standard drinks ≈ 1.0 oz AA). The timeline follow-back interview was repeated at 4 and 12 weeks after recruitment (Jacobson et al., 2008). Data from these three pre-natal maternal alcohol consumption interviews were then averaged to determine continuous measures of alcohol consumption around time of conception and across pregnancy (average oz AA consumed/day, oz AA/drinking day (dose/occasion), and frequency (days/week).

Inclusion Criteria. Women were considered to be heavy drinkers if they (1) reported consuming 14 or more standard drinks per week (approximately 1.0 oz of AA/day) or (2) reported engaging in binge drinking (5 or more drinks/occasion) when administered the 2-week timeline follow-back at the recruitment stage. These women were then invited to participate in the study. A random sample of women receiving care at the same antenatal clinic were also invited to join the study as control participants, if they were within 2 weeks of gestation of an enrolled heavy drinking woman and reported abstaining from alcohol or drinking only minimally (<7 standard drinks/week and no episodes of binge drinking).

Exclusion Criteria. Women who were younger than 18 years old, as well as those who reported having chronic health problems, such cardiac problems, epilepsy, or diabetes, were excluded from the study. Religiously observant Muslim women were excluded as their religion prohibits the consumption of alcohol, and their inclusion would have been

overrepresented in the control group. Exclusionary criteria relating to infants included multiple births, seizures, chromosomal anomalies, and neural tube defects.

Dysmorphology and FASD Diagnosis

We organised a diagnostic clinic in September 2005, at which two expert dysmorphologists (HE Hoyme, MD, and LK Robinson, MD) independently examined all of the children for dysmorphic features and growth restrictions using a standard diagnostic procedure (Hoyme et al., 2005). Case conferences were then held by the two dysmorphologists (HEH & LKR) and the primary investigators of the Cape Town longitudinal study (SWJ & JLJ) to reach consensus regarding which participants met criteria for either FAS or PFAS diagnoses. Diagnoses were determined based on this clinical examination, along with the information obtained during the timeline follow-back interviews regarding prenatal alcohol consumption and a case conference meeting held with the dysmorphologists, SWJ, JLJ, and CDM to decide on the final FASD diagnosis (Jacobson et al., 2008). Subsequent FASD diagnostic clinics, which included HEH, and other FASD dysmorphologists were conducted in 2009, 2013, and 2016, to confirm these diagnoses (Jacobson et al., 2021). Those not meeting criteria for FAS or PFAS were designated as heavily exposed but non-syndromal (HE) or controls, depending on whether the mothers drank heavily during pregnancy.

Facial Analysis

At the FASD diagnostic clinic held in 2009 (described above), when the participants were approximately 9 years old, 3D photos of the children were taken. A commercial photogrammetric camera (3dMD Inc, Atlanta) was used to capture 3-dimensional images of participants' faces (Suttie et al., 2013). Dense surface modelling and landmarking were conducted. Signature graph cluster analysis was then used to analyse the HE participants' faces. Facial areas of contraction, coincidence, and expansion were compared to matched

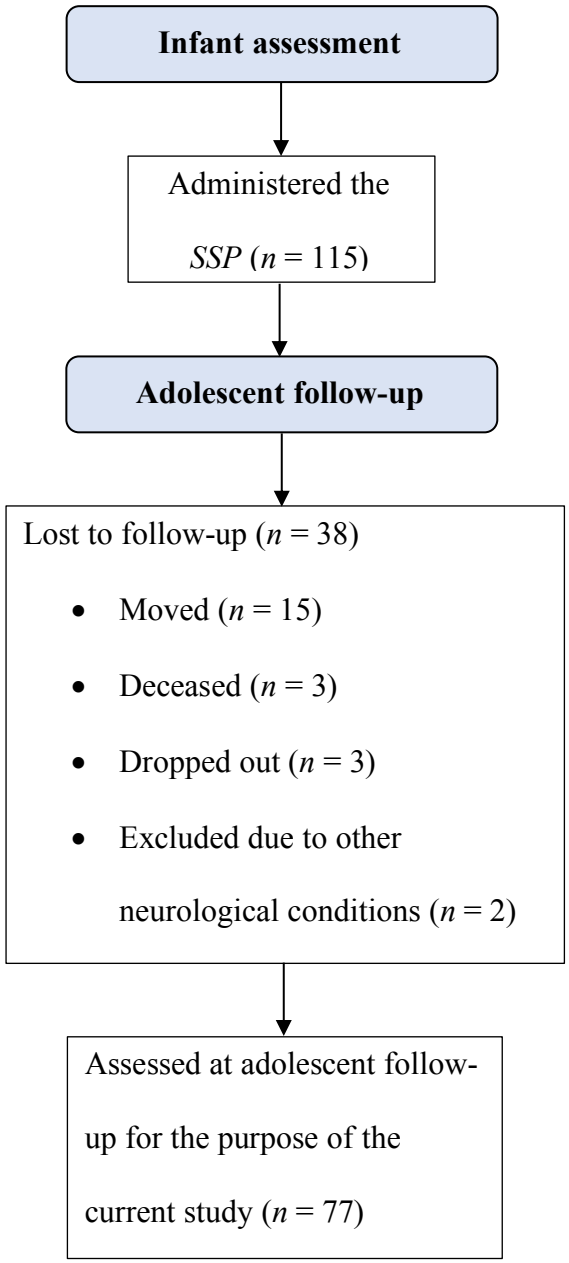
controls and visualized as heat maps. In a subset of the HE participants, the signature graphs detected subtle facial dysmorphism similar to the FAS and PFAS group, while the remaining HE participants had facial signatures resembling the controls. A more detailed procedure is described in Suttie et al. (2013). Hence, for the current study, the HE group was further divided into those who showed subtle facial dysmorphism that was more similar to the children with FAS (HE-dysmorphic) versus those who showed facial features that were more similar to the controls (HE-nondysmorphic) using dense surface modelling and signature graph analysis.

Current Sample

Due to participant dropout and availability of participants at the time of the current study, only 77 of the 115 infants assessed in the Hay et al. (2004) attachment study at 13 months old were evaluated at the adolescent follow-up (Mean age = 17.66 years; SD = .70; see Figure 1 below). The adolescent sample was comprised of five groups: FAS (13), PFAS (12), HE-dysmorphic (7), HE-nondysmorphic (18), and non- or minimally exposed controls (27). The sample consisted of 38 males (49.4%) and 39 females (50.6%).

Figure 1.

Flow of Participants for Study Procedures, Follow-up, and Analysis



Ethical Considerations

This research study adhered strictly to the ethical guidelines as described in the UCT Code concerning research with human subjects and in the Declaration of Helsinki (2013). Ethical approval was previously acquired for the larger study from UCT's Faculty of Health Sciences' Research Ethics Committee (HREC REF: 471/2015, Appendix A) and from Wayne State University's Institutional Review Board (IRB#: 026914B3F, Appendix A). In addition, the current study was approved through the Department of Psychology at UCT.

Written informed consent was obtained from mothers upon recruitment and again before commencing any follow-up assessments, including the adolescent assessments (Appendix B). Informed assent was also obtained from the adolescents prior to commencing the adolescent assessments (Appendix B). Both the mothers/primary caregivers and the adolescents were informed that their participation is voluntary and that they may choose to discontinue the assessments or withdraw from the longitudinal study at any time without any ramifications. All participants were also informed that, should they have any questions regarding their participation in the study, they may direct these to the researcher or to the local Principal Investigator and study paediatrician, C Molteno, M.D. Contact details for the Principal Investigators as well as the relevant Ethics Committees were provided to the mothers/primary caregivers, should they prefer to direct any queries to them.

Infants and adolescents presenting with any serious medical problems were referred to Red Cross Children's Hospital/Groote Schuur Hospital (or a local clinic) by the senior longitudinal cohort paediatrician (C Molteno). Referrals were also made for educational remediation or suspected psychological problems.

All data collected during adolescent assessments as well as during the primary caregiver interviews are kept strictly confidential. Each participant was assigned a unique participant number, and all data collected from the participant were labelled with this number

rather than his/her name to ensure anonymity. In addition, the master file linking the names with the IDs and all data are stored in a locked cabinet at the CDRL at UCT, which is access controlled.

Each participant, along with their mother/primary caregiver was transported to and from their homes in our research van, and provided breakfast, a snack, and lunch at the CDRL on all assessment days. Informed consent was obtained from the mother/primary caregiver and informed assent was obtained from the adolescent before commencing the assessments. The larger battery of adolescent assessments was conducted over 2 days to avoid potential fatigue effects. On both days, the adolescent was encouraged to take regular breaks as needed throughout the day, with a break for lunch in between. In line with the UCT ethical guidelines, both the mother/primary caregiver and the adolescent were compensated for their participation in this study - the mother with a monetary compensation of ZAR150, and the adolescent with a voucher valued at ZAR100 redeemable at a local department store.

Measures

Although no South African norms are available for the tests of attachment, emotion regulation, and general intellectual functioning, these measures have been used in clinical and/or FASD research in South Africa (Ferrett, 2011; Pileggi, 2017; Van Wyhe, 2012). Consistent with our previous reports on our Cape Town cohorts (e.g. Biffen et al., 2018; Jacobson et al., 2011; Woods et al., 2018), this study compared test scores for participants with FASD to control participants from the same community whose mothers abstained or drank only minimally during pregnancy rather than to Westernised normative data, which do not take into account major socioeconomic, educational and cultural differences (Foxcroft et al., 2004).

Furthermore, many of the participants (41.6%) who took part in this study were Afrikaans-speaking. The measures administered in this study (outlined below) were,

therefore, translated from English into Afrikaans by Master's level researchers, as well as back-translated, by native Afrikaans speakers. During this process, care was taken to preserve the item or instruction's original meaning and to retain the same level of complexity. In some instances, items were altered to render them more culturally appropriate. For example, in the vocabulary subtest, the word "crocodile" was used instead of "alligator", and "torch" was used instead of "flashlight".

Infant Attachment

The Strange Situation Paradigm (SSP; Ainsworth, 1979) was used to assess mother-infant attachment when the participants were 13 months old (Hay et al., 2004). The SSP is designed to activate attachment responses through several episodes of separation from the caregiver, followed by a reunion. In the first episode, the experimenter first describes the procedure, before introducing the infant and their caregiver to the room in which the procedure will take place. In the second episode, the infant and caregiver are alone in the room. In the third episode, a stranger enters the room, initially sitting silently before speaking to the caregiver and engaging the infant in play. The first separation is observed during the fourth episode when the caregiver exits the room, leaving the infant with the stranger. The first reunion then occurs in the fifth episode, during which the caregiver enters the room again and the stranger exits. The second separation occurs during the sixth episode when the caregiver exits the room again, leaving the infant completely alone. The seventh episode sees the stranger return to the room. Finally, the second reunion is observed during the eighth episode when the caregiver returns to the room and the stranger leaves.

During this procedure, the coder is trained to observe the infant's behaviour toward the stranger and caregiver, with particular focus on the infant's reactions to the reunions, and their exploration of the environment throughout the episodes (Ainsworth & Bell, 1970). The procedure was videotaped through a one-way mirror. The videotapes were rated by the late

A. Hay, Ph.D., who was trained and certified to score ABCD by the Institute of Child Development at the University of Minnesota. Infants were classified as either A) avoidant, B) secure, C) resistant, or D) disorganised. Other attachment research conducted in South Africa has also successfully used the SSP among low income populations (Tomlinson, Cooper, & Murray, 2005, 2016).

Emotion Regulation

The use of multiple methods has been advocated when measuring emotion regulation given the complexity of the construct (Adrian et al., 2011). In order to measure the construct as comprehensively as possible, I made use of two caregiver-report questionnaires, described below. Furthermore, given that neither of the two questionnaires had previously been used specifically with adolescents with FASD in South Africa, the use of these measures in this context was exploratory, and thus both measures were used in the data analysis. Before analysing the data produced by these measures, correlations between each of the subscales were examined to determine if composites could be made (for more information see the data analysis plan below).

Another consideration given to the measurement of emotion regulation in this study was that adolescents with PAE may not necessarily have the insight or the language ability to reflect upon and express the various manners in which they regulate their emotions. I therefore decided that the caregiver-reports would be most informative.

Affect Regulation Checklist. Emotion regulation was assessed using the *Affect Regulation Checklist* (ARC; Moretti, 2003). The ARC is a 12-item questionnaire comprised of items adapted from emotion regulation scales developed by Gross and John (1998). The ARC was designed to reflect the multidimensional structure of the construct by assessing both one adaptive (i.e., *Reflection*) and two maladaptive factors (i.e., *Lack of Control* and *Suppression of Affect*) associated with the regulation of emotion. Test items were also

conceptualized to assess regulatory processes without referring to specific emotional states. That is, items avoid referring to specific emotions (e.g., "I have a hard time controlling my feelings"). The original ARC is rated on a 3-point Likert scale. Previous research has confirmed the 3-factor structure of the ARC (Penney & Moretti, 2010) and satisfactory internal reliabilities have been found for all 3 factors (between .65 and .86; Moretti & Craig, 2013; Penney & Moretti, 2010). However, in an effort to eliminate a 'neutral' point on the scale, I decided to adapt the ARC into a 4-point Likert scale. Responses to the items were therefore rated either 0 = never like my child, 1 = sometimes like my child, 2 = often like my child, or 3 = always like my child. Only one study has made use of the measure in the South African context. In particular, Pileggi (2017) made use of this measure with two samples of South African adolescents. This study yielded good internal consistency reliability estimates of .70 and .72, especially considering the length of the scale (12 items) and the fact that Cronbach's alpha is sensitive to the number of items in a scale (Field, 2009). For the purpose of the current study, the ARC caregiver-report was administered verbally to the caregivers. Given that the 3 different subscales of the ARC measure distinct aspects of emotion-regulation, scores on each subscale were examined independently.

Emotion Regulation Checklist. Maternal/caregiver report of adolescent emotion regulation was also assessed using the *Emotion Regulation Checklist* (ERC; Shields & Cicchetti, 1997). The ERC comprises of 24 items intended to measure a child or adolescent's ability to regulate his/her emotions. The measure consists of two subscales – one assessing adaptive factors associated with the regulation of emotion (i.e. *Adaptive Emotion Regulation*) and one assessing maladaptive factors associated with the regulation of emotion (i.e. *Lability/Negativity*). The *Adaptive Emotion Regulation* subscale assesses emotional self-awareness, empathy, and situational appropriateness of displays of affect. On this subscale, higher scores indicate more adaptive emotion regulation. The *Lability/Negativity* subscale

assesses affective lability, lack of flexibility, and negative affectivity. Higher scores on the this subscale indicate greater lability and negativity. Parents' responses to each item were rated on a 4-point Likert scale (1 = never like my child, to 4 = almost always like my child). The ERC was administered verbally to the mothers/caregivers. An investigation into the internal consistency of the measure yielded values between .83 and .96 (Shields & Cicchetti, 1997). The ERC has successfully been used in several studies conducted in developing countries. Von Suchodoletz, Uka, and Larsen (2015) found good internal consistency (.77) for a sample of Albanian children, while a study conducted by Reis et al. (2016) also provided evidence of the construct validity of the measure for use with children in Brazil. In addition, the ERC has previously been used with children with PAE, yielding internal consistencies between .72 and .91(Petrenko et al., 2017). Once again, given that the 2 subscales of the ERC measure 2 contrasting aspects of emotion regulation, the scores on these subscales were evaluated separately.

General Intellectual Functioning

IQ, a measure of general intellectual functioning, was assessed in adolescence using the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999) as part of the Cape Town Longitudinal Cohort battery. The WASI is suitable for use with individuals aged 6 to 89 years. The measure consists of four subtests, including Block Design, Vocabulary, Similarities, and Matrix Reasoning, which together yield a full-scale IQ score (FSIQ). Verbal IQ (VIQ) is determined using the Vocabulary and Similarities subtests, while Performance IQ (PIQ) is determined using the Block design and Matrix reasoning subtests. The four WASI subtests were scored according to the scoring procedures outlined in the original WASI manual. Raw scores were converted to age-corrected *T*-scores. The WASI has good validity and reliability, with test-retest reliability coefficients of between .92 and .95 (Wechsler, 1999). Although the WASI has been normed for Western populations, it has been found to be

appropriate for, and has been used extensively with, South African samples (Ferrett, 2011; Van Wyhe, 2012). Given that previous research has provided some, albeit inconsistent, evidence for the association between IQ and emotion regulation, it was of interest to examine adolescent FSIQ as a potential predictor of emotion regulation abilities.

Procedure

This research study formed part of a broader protocol administered to the Cape Town Longitudinal Cohort. At the 13-month infant assessment, the primary caregiver and their infants were transported to the CDRL at the University of Cape Town (UCT) by a staff driver in the project research van. Upon arriving at the CDRL, the primary caregiver and infants were provided with breakfast, and at the end of the assessment, the primary caregiver received monetary compensation and a photograph of himself/herself with the infant. The infant also received a small gift. Assessment using the Ainsworth SSP took place in a large room unfamiliar to the mothers and the infants. Age appropriate toys were available for the infant to play with.

At the adolescent follow-up assessment, a larger battery of neuropsychological assessments, which included the measures described above, was conducted over 2 consecutive days at the CDRL at UCT. A research nurse was responsible for scheduling appointments for testing. The adolescents and their primary caregivers were transported by the study's driver to the CDRL at UCT in the project research van. On both assessment days spent at the CDRL, the adolescents and their caregivers were given breakfast, a snack and lunch.

Before administering any neuropsychological measures, informed consent and assent were acquired (Appendix B). Each adolescent was assessed individually, either in English or in Afrikaans, based on the primary language in which the adolescent was taught at school. All assessments were conducted in a controlled environment and administered by trained

psychology graduate research assistants. Assessments were scored according to the respective standardized procedures. To eliminate the possibility of experimenter bias, all test administrators were blind to the participant's history of PAE and FASD diagnosis. However, examiners were not able to remain blind in the few cases involving the most severely affected adolescents who presented with the characteristic facial dysmorphic features of FAS.

While the adolescent was being assessed, the primary caregiver was interviewed by the senior longitudinal cohort paediatrician (C Molteno) regarding their child's behaviour and emotion regulation, and health history.

Statistical Analysis Plan

The Statistical Package for the Social Sciences (SPSS; IBM Corporation, 2019) version 26.0 was used to analyse the data. Eta-squared effect size estimates are reported for all statistical analyses. Recommendations regarding hypothesis testing stress the importance of avoiding Type I errors (i.e. reporting an association between two variables where one does not exist; Field, 2013). In contrast, the use of strict methods to avoid Type I errors often results in a greater risk of making a Type II error (i.e. reporting no association between two variables when one does in fact exist). In public health research settings, specifically, there is more concern around dismissing genuine effects, and consequently failing to recognize a genuine health risk (Jacobson & Jacobson, 2005). As Jacobson and Jacobson (2005) point out, cognitive impairments resulting from PAE are often subtle and associated with small effect sizes. Nevertheless, these subtle impairments may be clinically significant. In line with this, a distinction has been made between confirmatory- and exploratory-type data analysis (Jaeger & Halliday, 1998). While the goal of confirmatory data analysis may be to minimize the risk of making a Type I error, the goal of exploratory data analysis is to minimize the risk of making a Type II error. As such, it is suggested that alpha be adjusted to be more conservative at for confirmatory-type analyses. The approach to data analysis in the current

study was informed by the context of public health research, as well as the guidelines distinguishing between confirmatory and exploratory data analysis. Alpha was, therefore, set at $\alpha = .01$ for confirmatory-type analyses, and at $\alpha = .05$ for exploratory-type analyses.

Variables Used in the Analyses

Exposure Variable. Several factors can impact the effect of PAE on the developing foetus. These include dosage and duration of alcohol consumption, maternal health and age at delivery, genetic variability, as well as how efficiently the mother metabolizes alcohol (Jacobson, Jacobson, Sokol, Chiodo, & Corobana, 2004). Examining continuous measures of alcohol (oz. AA/day, oz. AA/occasion, and number of drinking days/week) is one way to examine the extent to which the individual is affected by PAE. On the other hand, the FASD categories provide diagnostic information indicating the level of impairment attributable to PAE. The current study presents the categorical measure of FASD including the HE split (FAS, PFAS, HE-dysmorphic, HE-nondysmorphic and controls) as the independent variable. For the purpose of the inferential analyses, these categories were collapsed into two groups – those who presented with FAS or PFAS or even subtle dysmorphism similar to FAS (FAS-dysmorphism+ = includes FAS, PFAS, and HE-dysmorphic), and those who presented with features more similar to controls (FAS-dysmorphic- = includes HE-nondysmorphic and controls).

Outcome Variable. The outcome variables included the subscales of the emotion regulation questionnaires, the ARC and the ERC: *ARC Reflection, ARC Lack of Control*, *ARC Suppression of Affect, ERC Adaptive Emotion Regulation, and ERC Lability/Negativity*. As previously mentioned, the use of multiple methods has been advocated when measuring emotion regulation given the complexity of the construct (Adrian et al., 2011). Furthermore, given that neither of the two questionnaires had previously been used specifically with adolescents with FASD in a South African context, the use of these measures in this context

was exploratory, and thus both measures were examined in the data analysis. It must be noted that the possibility of creating composites with the ARC and ERC subscales was also considered. As Table 1 shows, there were only moderate correlations between the subscales, with the highest correlations observed between the *ARC Suppression of Affect* and the *ERC Lability/Negativity* and *ARC Lack of Control* subscales. However, given that each of these subscales dealt with a nuanced aspect of emotion regulation, which, according to previous research, may be differentially impacted by the predictor variables, I decided against creating composite variables.

Table 1.

Intercorrelations Between ARC and ERC Subscales

	1.	2.	3.	4.	5.
1. ARC Reflection	1				
2. ARC Lack of Control	-.36**	1			
3. ARC Suppression of Affect	-.10	.55**	1		
4. ERC Adaptive Emotion Regulation	.21	-.30*	-.23	1	
5. ERC Lability/Negativity	-.31*	.62**	.33*	-.18	1

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

Descriptive Statistics

The first phase of data analysis involved investigating the descriptive statistics. The purpose of this phase was to detect any possible outliers, examine the distribution of the outcome variables and predictors, and determine whether the assumptions underlying parametric statistical tests were met.

Sample characteristics for both the mothers and the adolescents were generated using a one-way analysis of variance (ANOVA), to describe the between-group differences for the

continuous demographic, alcohol, and intelligence variables in the sample. Sample characteristics were presented both for the five diagnostic groups, as well as for the dichotomous variable (FAS-dysmorphism+ vs. FAS-dysmorphism-). Where statistically significant results were found, *post-hoc* analyses were performed using Tukey's HSD tests. Given that these were essentially confirmatory-type analyses (i.e., group differences are expected), alpha was set at $\alpha = .01$ in order to reduce the probability of Type I errors. Descriptive statistics for attachment classification and performance on the measures of emotion regulation were also presented.

Inferential Statistics

Inferential data analysis was conducted in accordance with the specific aims of the study. Given that these were essentially exploratory analyses, I was more concerned about making Type II errors than Type I errors. Hence, the alpha level was left at $\alpha = .05$ for these analyses. First, chi-squared analyses were conducted to evaluate between-group differences (FAS-dysmorphism) on attachment security and attachment organisation. Five hierarchical multiple regression analyses were then conducted to determine the association between FAS-dysmorphism, attachment security, and various covariates with the caregiver-reported emotion regulation outcome variables. Given previous research, the following variables were evaluated as potential covariates: age at the time of adolescent assessment, sex, SES, prenatal smoking, and adolescent IQ. Several sources have argued that it is important for theory to guide the inclusion of appropriate constructs when building a regression model (e.g. Braun & Oswald, 2011). As such, the identified covariates were included in the hierarchical multiple regression model irrespective of whether they correlated with the given outcome variable.

Given that exposure to methamphetamine ($n = 2$), and cocaine ($n = 1$) were too infrequent to control for in this sample, analyses detecting significance were rerun without the drug users.

Post-hoc Power Analyses

G*Power was used to determine the power the study had to detect effects given the sample size (Faul et al., 2007). Research on other domains of cognitive impairment in FASD has yielded a range of small to large effect sizes (d = .2 - .8; Green et al., 2009; Kingdon, Cardoso, & McGrath, 2016; Mattson et al., 2010; Rasmussen et al., 2013; Wozniak et al., 2017). In line with Cohen's (1988) guidelines, statistical power estimates of .20, .50, and .80 were used, while alpha was set at .05. These parameters were used in order to estimate the power the study had to detect small, moderate, and large effects, given the sample size.

In terms of the ANOVAs run to generate descriptive statistics, ω^2 = .01 was considered a small effect size, ω^2 = .06 was considered a moderate effect size, and ω^2 = .14 was considered a large effect size (Field, 2009). For the purpose of the Chi-squared tests, G*Power estimated a required total sample size of 385 participants to detect a small effect of φ = .10, while only 43 were required to detect a moderate effect of φ = .30. The current sample size was, therefore, sufficient to detect moderate or large effects but underpowered to detect small effects. For the regression analyses, the sample size required to detect a small effect size of f^2 = .02 and a moderate effect size of f^2 = .15 (Cohen, 1988) was calculated to be 485 and 68 participants, respectively. The current sample size was, therefore, sufficiently powered to detect moderate to large effects but underpowered to detect small effects.

While it would have been preferable to have a larger sample size in order to be able to detect small effects, the sample size for the current study was limited by the longitudinal nature of the study. That is, only 115 infants from the larger cohort (n=227) were administered the SSP (Hay et al., 2004). Furthermore, by adolescence, 15 had moved away, 3 were deceased, 3 had dropped out, 2 were excluded due to a neurological condition, and 15 had completed the adolescent assessment battery prior to the addition of the emotion

regulation measures. As such, I was only able to assess 77 of the individuals from the larger cohort on the emotion regulation measures.

Results

Sample Characteristics

Maternal Sample Characteristics

Five group comparison. Table 2 below presents the socio-demographic and substance use characteristics of the participant mothers across the five diagnostic groups. Alpha was set at α = .01 for these analyses. No significant between-group difference was observed for maternal age at delivery. There was a significant between-group difference for maternal level of education, with a large effect size (ω^2 = .161). Tukey HSD *post-hoc* analyses revealed that mothers of adolescents with PFAS had significantly fewer years of education than mothers of control participants (p = .001). The between-group difference between mothers of PFAS adolescents and those who were HE-nondysmorphic (heavily exposed without facial dysmorphism) was not significant (p = .050). Between-group differences for mother's scores on the PPVT-R and on the Raven were both found to be insignificant. The between-group difference for maternal socioeconomic status (SES) as measured by the Hollingshead Four Factor Index of Social Status (2011) was significant, with a large effect size (p = .002, ω^2 = 162). Mothers of adolescents with FAS and PFAS were significantly (p = .010 and p = .002, respectively) more socio-economically disadvantaged (mean = level 5 - unskilled labourers – lowest level) than those in the HE-dysmorphic group and control groups (mean = level 4 - semi-skilled labourers).

As expected, there were significant between-group differences for average AA consumed per day during pregnancy (AA/day), average AA consumed per occasion across pregnancy (AA/drinking day), and for the frequency of drinking days per week across pregnancy (proportion of drinking days; all p's < .001). Large effect sizes were observed for all three of the continuous prenatal alcohol exposure variables (ω^2 = .199, ω^2 = .371, and ω^2 = .372, respectively). For AA/day, *post-hoc* tests revealed that mothers of adolescents with FAS

consumed more compared to those of controls (p = .001), while the comparison between mothers of adolescents with PFAS and controls fell just short of significance (p = .016). Both the HE-dysmorphic and the HE-nondysmorphic groups did not differ significantly from controls (both p's > .072). Furthermore, there were no significant differences between the four alcohol exposed groups (all p's > .108). That is, mothers of adolescents with FAS and PFAS did not consume significantly more alcohol per day than mothers of HE-nondysmorphic and HE-dysmorphic adolescents. Mothers of adolescents with FAS also did not consume significantly more than those of adolescents with PFAS. In terms of AA/occasion, again no significant between group differences were found between the four alcohol exposed groups (all p's > .963). However, mothers from all alcohol exposed groups consumed significantly more alcohol per occasion than mothers of controls (all p's < .004). Regarding frequency of alcohol consumption, mothers of adolescents with FAS consumed on average, 8-9 drinks, 1-2 days per week. *Post-hoc* tests revealed that mothers of adolescents from this group (FAS), as well as from the PFAS and HE-nondysmorphic groups consumed alcohol significantly more frequently than mothers of controls. However, no significant differences were found between the four alcohol exposed groups (all p's > .090). The above data suggests that a dose-dependent relation did not exist between PAE and FASD diagnosis for this sample. It is also important to note that there were no significant differences between HE-nondysmorphic and HE-dysmorphic adolescents for any of the continuous alcohol measures (all p's > .695).

Between-group differences for prenatal smoking during pregnancy fell short of significance. Similarly, no significant between-group differences were found for marijuana use. Only 1 mother reported consuming cocaine during pregnancy (2-3 days/month); and 2 mothers reported using methaqualone during pregnancy (2-4 days/month).

Table 2.

Maternal Sample Characteristics by Diagnostic Category Including HE Split (*N* = 77)

	FAS (*n* =13)	PFAS (*n* =12)	HE+ (*n* =7)	HE- (*n* =18)	Control (*n* =27)	*F* or χ^2	*p*	Omega Squared
Age at delivery (years)	29.38 (6.51)	27.60 (7.43)	27.21 (7.43)	24.97 (5.00)	26.05 (5.68)	1.23	.307	.012
Education (years)	8.15 (2.12)	6.67 (2.46)	9.43 (1.51)	8.78 (2.39)	9.63 (1.74)	4.69	.002	.161
PPVT-R	62.54 (17.66)	49.08 (13.45)	67.29 (18.90)	62.72 (16.47)	65.48 (20.05)	1.99	.105	.049
Raven	26.38 (11.14)	25.33 (7.66)	33.29 (14.22)	30.72 (11.67)	28.85 (8.78)	1.00	.412	.000
Socioeconomic status[a] (SES)	17.54 (7.64)	15.29 (6.68)	29.50 (8.12)	19.44 (7.98)	21.91 (7.39)	2.65	.002	.162
Prenatal exposure								
Alcohol								
AA/day (oz)	1.40 (1.95)	1.10 (.81)	.25 (.22)	.79 (.99)	.00 (.00)	5.78	<.001	.199
AA/occasion (oz)	4.16 (1.90)	4.32 (2.28)	3.68 (4.02)	3.73 (3.52)	.00 (.00)	12.34	<.001	.371
Frequency(days/wk)	.25 (.23)	.26 (.16)	.10 (.06)	.18 (.15)	.00 (.00)	12.39	<.001	.372
Smoking								
Cigarettes/day (smokers only)	6.52 (5.62)	7.83 (5.54)	4.07 (3.49)	7.03 (6.28)	3.17 (5.70)	2.20	.077	.059
Marijuana								
Days/mo (users only)	.13 (.32)	.26 (.89)	.00 (.00)	.47 (1.40)	.10 (.52)	.69	.600	-.016

Note. Values are mean (SD). FAS = fetal alcohol syndrome; PFAS = partial FAS; HE+ = heavily exposed nonsyndromal with subtle facial dysmorphism resembling FAS; HE- = heavily exposed nonsyndromal without facial dysmorphism resembling controls; PPVT-R = Peabody Picture Vocabulary Test-Revised; Raven = Raven's Progressive Matrices; AA = absolute alcohol. [a]Based on Hollingshead Four Factor Index of Social Status (2011).

Two group comparison. Table 3 below presents the socio-demographic and substance use characteristics of the participant mothers according to FAS-dysmorphism grouping. Alpha was set at $\alpha = .01$ for these analyses. The between-group difference for maternal age at delivery was not significant. There was a significant between-group difference for maternal level of education, with mothers of adolescents with FAS-dysmorphism+ having significantly fewer years of education than mothers of those with FAS-dysmorphism-. The size of the effect for between-group difference in maternal education was medium ($\omega^2 = .083$). Between-group differences for mother's scores on the PPVT-R and on the Raven were not significant. Similarly, no between-group difference was found for maternal socioeconomic status (SES) as measured by the Hollingshead Four Factor Index of Social Status (2011).

As expected, there was significant between-group difference for average AA consumed per day across pregnancy (AA/day), average AA consumed per occasion across pregnancy (AA/drinking day), and for the frequency of alcohol consumption (proportion of drinking days; all p's $< .01$). While a medium to large effect size was observed for AA/day ($\omega^2 = .091$), large effect sizes were observed for AA/drinking day and frequency of alcohol consumption ($\omega^2 = .174$, and $\omega^2 = .182$, respectively). While mothers of adolescents with FAS-dysmorphism+ consumed on average 4-5 standard drinks, 1-2 days a week, mothers of FAS-dysmorphism- consumed 1-2 standard drinks, 0-1 days a week, on average. These results demonstrate that although a dose-dependent relationship was not evident between PAE and the FASD diagnosis (as found in the 5-group comparison), a dose-dependent relationship did exist between PAE and FAS-dysmorphism.

No between group difference was found for either prenatal smoking or marijuana use during pregnancy. Given that exposure to cocaine and methaqualone were too infrequent to control for in this sample, the analyses described below were run excluding these cases.

Results detecting significance for both the chi-square and the hierarchical multiple regression analyses remained significant even after excluding these cases.

Table 3.

Maternal Sample Characteristics by FAS-Dysmorphism (*N* = 77)

	FAS-dysmorphic+ (*n* =32)	FAS-dysmorphic- (*n* =45)	*F* or χ^2	*p*	Omega Squared
Age at delivery (years)	28.24 (6.37)	25.62 (5.38)	3.80	.055	.035
Education (years)	7.88 (2.34)	9.29 (2.04)	7.95	.006	.083
PPVT-R	58.53 (17.68)	64.38 (18.55)	1.93	.169	.012
Raven	27.50 (10.86)	29.60 (9.95)	.77	.382	-.003
Socioeconomic status[a] (SES)	19.31 (9.07)	20.92 (7.64)	.71	.402	-.004
Prenatal exposure					
Alcohol					
AA/day (oz)	1.03 (1.38)	.32 (.73)	8.73	.004	.091
AA/occasion (oz)	4.11 (2.54)	1.49 (2.86)	17.20	<.001	.174
Frequency(days/wk)	.22 (.19)	.07 (.13)	18.14	<.001	.182
Smoking					
Cigarettes/day (smokers only)	6.48 (5.24)	4.71 (6.17)	1.73	.193	.009
Marijuana					
Days/mo (users only)	.15 (.58)	.25 (.98)	.26	.615	-.010

Note. Values are mean (SD). FAS-dysmorphic+ = FAS+PFAS+HE-dysmorphic; FAS-dysmorphic- = HE-nondysmorphic+controls; PPVT-R = Peabody Picture Vocabulary Test-Revised; RPM = Raven's Progressive Matrices; AA = absolute alcohol. [a]Based on Hollingshead Four Factor Index of Social Status (2011).

Adolescent Sample Characteristics

Five group comparison. Table 4 below presents the socio-demographic and cognitive characteristics for the adolescents in each of the 5 diagnostic groups. Alpha was set at $\alpha = .01$ for these analyses. A significant between-group difference was observed for age at the adolescent assessment, with a large effect size ($\omega^2 = .165$). Tukey HSD *post-hoc* analyses revealed that adolescents in the PFAS group were significantly older than those in the control group ($p = .001$). No significant between-group differences were observed for gestational age at birth. A chi-square test also demonstrated no significant between-group difference for sex, while the between-group difference for language fell just short of significance. A significant between-group difference was observed for WASI FSIQ, with a large effect size ($\omega^2 = .177$). *Post-hoc* analyses demonstrated that adolescents in both the FAS and PFAS groups performed significantly more poorly than those in the HE+ group ($p = .012$ and $p = .009$, respectively). However, the FAS and PFAS groups did not differ significantly from one another. Interestingly, the FAS and PFAS groups also did not differ significantly from the HE- and control groups.

Table 4.

Adolescent Sample Characteristics by Diagnostic Category Including HE Split (*N* = 77)

Variable	FAS (*n* = 13)	PFAS (*n*= 12)	HE+ (*n*=7)	HE- (*n* =18)	Control (*n* =27)	*F* or χ^2	*p*	Omega Squared/ Cramer's V
Age at assessment (years)	17.68 (.66)	18.24 (.59)	17.98 (.49)	17.65 (.73)	17.32 (.63)	4.80	.002	.165
Gestational age at birth (weeks)	38.90 (2.59)	38.17 (2.21)	39.47 (3.19)	38.81 (2.50)	38.98 (1.59)	.43	.784	-.030
Sex (male:female)	6:7	6:6	3:4	10:8	13:14	.47	.977	.078
Language (English:Afrikaans)	5:8	3:9	6:1	11:7	20:7	12.58	.014	.404
WASI IQ								
FSIQ[a]	68.31 (12.18)	67.42 (10.35)	89.14 (10.95)	81.56 (13.26)	79.19 (15.53)	4.99	.001	.177

Note. Values are mean (SD). FAS = fetal alcohol syndrome; PFAS = partial FAS; HE+ = heavily exposed nonsyndromal with subtle facial dysmorphism resembling FAS; HE- = heavily exposed nonsyndromal without facial dysmorphism resembling controls; WASI = Wechsler Abbreviated Scale of Intelligence; FSIQ = Full Scale IQ. [a]Data missing for 2 adolescents in the HE- group and 1 adolescent in the control group.

Two group comparison. Table 5 below presents the socio-demographic and cognitive characteristics according to FAS-dysmorphism grouping. Once again, alpha was set at $\alpha = .01$ for these analyses. A significant between-group difference was observed for age at the adolescent assessment, with adolescents presenting with FAS-dysmorphism+ being significantly older than those who did not present with dysmorphism. The size of the effect for the between-group difference in age at adolescent assessment was medium to large ($\omega^2 = .113$). No significant between-group difference was observed for gestational age at birth. A chi-square test demonstrated no significant between-group difference sex. The between-group differences for language and WASI FSIQ also fell short of significance.

Table 5.

Adolescent Sample Characteristics by FAS-Dysmorphism ($N = 77$)

Variable	FAS-dysmorphic+ ($n = 32$)	FAS-dysmorphic- ($n = 45$)	F or χ^2	p	Omega Squared/ Cramer's V
Age at assessment (years)	17.96 (.64)	17.45 (.68)	10.79	.002	.113
Gestational age at birth (weeks)	38.75 (2.56)	38.91 (1.98)	.10	.752	-.012
Sex (male:female)	23:22	15:17	.12	.714	.042
Language (English:Afrikaans)	14:18	31:14	4.87	.027	.251
WASI IQ					
FSIQ[a]	72.53 (14.09)	80.10 (14.59)	5.03	.028	.052

Note. Values are mean (SD). FAS-dysmorphic+ = FAS+PFAS+HE-dysmorphic; FAS-dysmorphic- = HE-nondysmorphic+controls; WASI = Wechsler Abbreviated Scale of Intelligence; FSIQ = Full Scale IQ. [a]Data missing for 3 adolescents in the FAS-dysmorphic- group.

Adolescent Attachment by FASD Diagnostic Group

Figure 2.

Percentage of Infants in Each Attachment Classification by Diagnostic Group Including HE Split

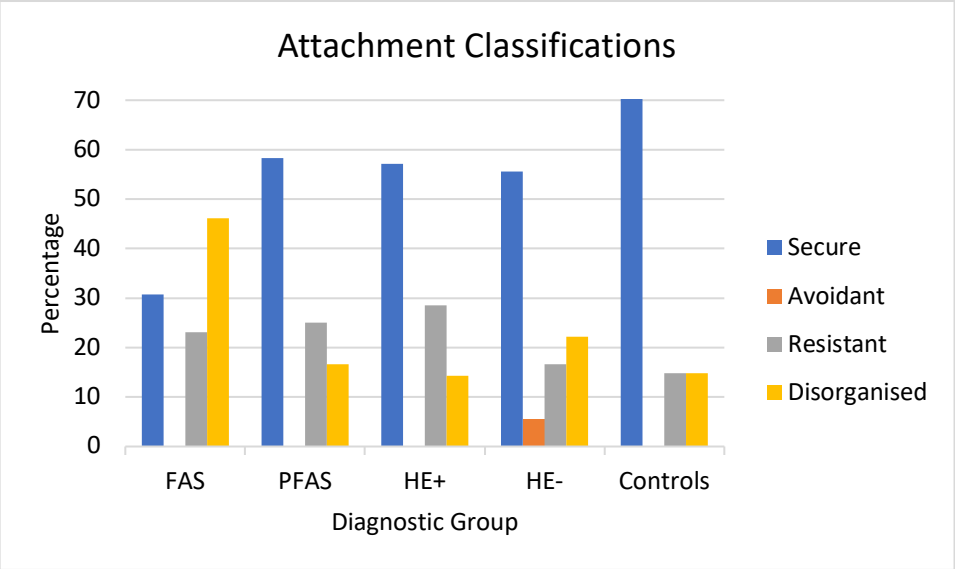
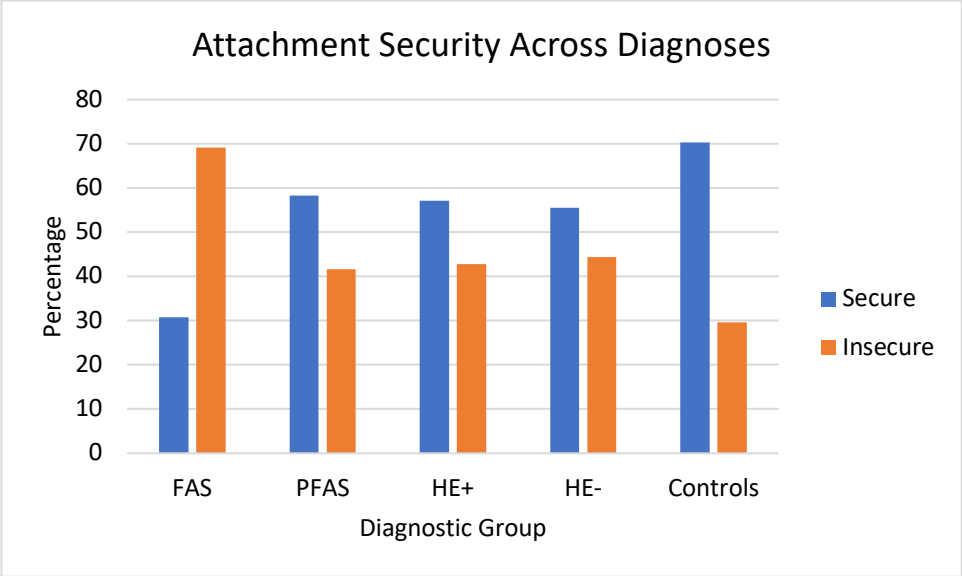


Figure 3.

Percentage of Secure and Insecure Attachment in Infants by Diagnostic Group Including HE Split



Descriptive Statistics for Attachment

Figure 2 presents the percentage of adolescents falling within each attachment classification group according to FASD diagnosis when HE is divided into the two HE split groups. Figure 3 presents the percentage of adolescents falling within the secure and insecure groups according to FASD diagnosis with the HE split groups. These figures, as well as the text below, present the distribution of attachment patterns for the current sample, which are consistent with the distribution of attachment patterns previously found in the larger sample studied by Hay et al. (2004) from which the current subsample was drawn.

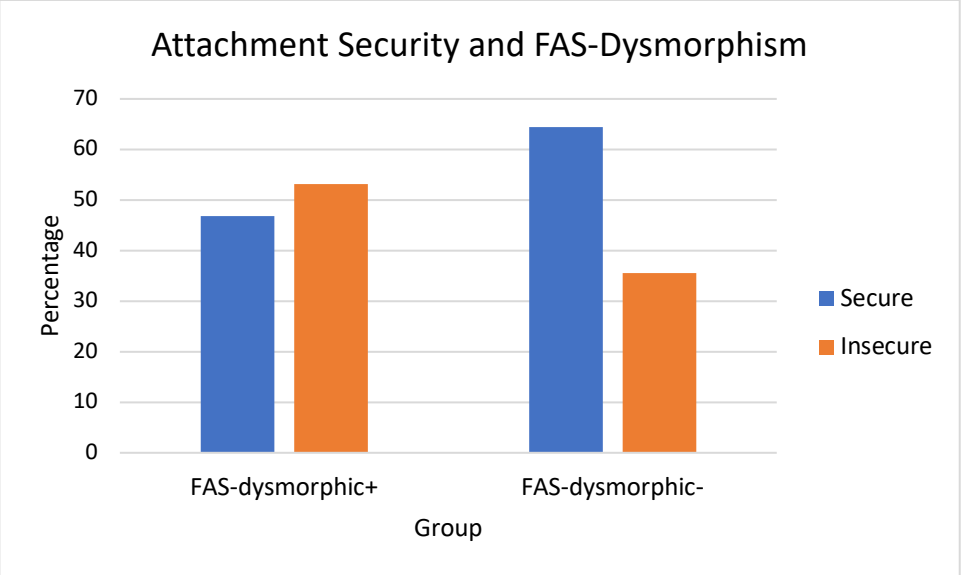
Consistent with the data previously reported by Jacobson and Jacobson (2013) for the larger group of FAS, PFAS, HE, and control infants, the lowest rate of secure attachment is observed for infants with FAS (30.8%) and is accompanied by a high rate of insecure attachment (69.2%). The rates of secure attachment are comparable for PFAS (58.3%), HE-

dysmorphic (57.1%), and HE-nondysmorphic (55.6%) adolescents. The control group appears to have the highest rate of secure attachment (70.4%), and as such, the lowest rate of insecure attachment (29.6%). The rate of resistant attachment was fairly comparable between the FAS (23.1%), PFAS (25.0%), and HE-dysmorphic (28.6%) groups. However, lower rates of resistant attachment were observed in the HE-nondysmorphic (16.7%) and control (14.8%) groups. Also consistent with the findings from the larger sample reported by Jacobson and Jacobson (2013), the FAS (46.2%) group exhibited the highest rate of disorganised attachment. By contrast, the PFAS (16.7%), HE-dysmorphic (14.3%), and HE-nondysmorphic (22.2%) groups exhibited rates of disorganised attachment that were more similar to controls (14.8%). It is interesting to note that only one adolescent from the sample was classified as avoidant in infancy. This adolescent fell into the HE-nondysmorphic group.

Attachment Security and Organisation by FAS-Dysmorphism

Figure 4.

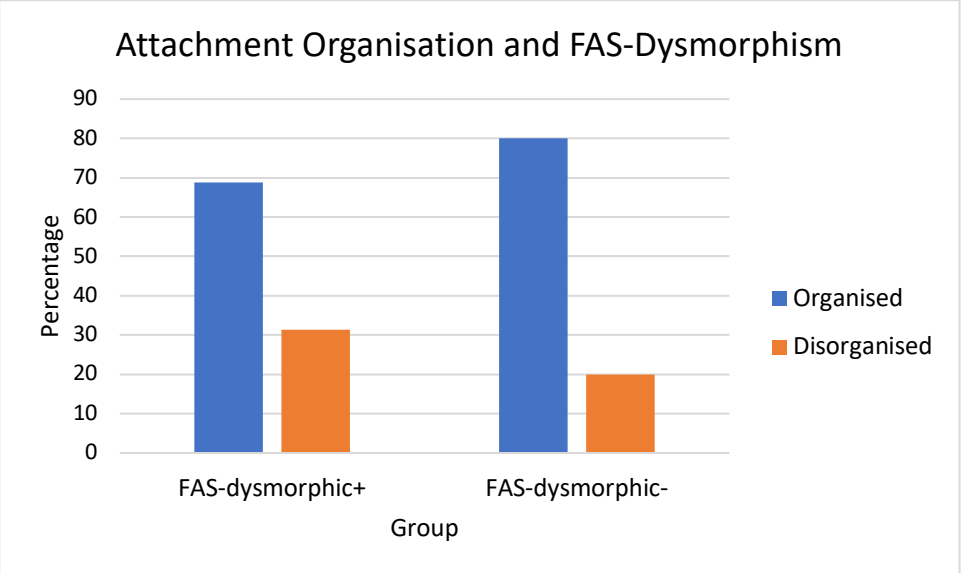
Percentage of Secure and Insecure Attachment in Infants with FAS-Dysmorphism+ and FAS-Dysmorphism-



To test hypothesis 1, infant attachment classifications were collapsed into a dichotomous variable denoting secure attachment versus insecure attachment, the latter of which consisted of avoidant, resistant, and disorganised attachment classifications. As figure 4 shows, higher rates of insecure attachment were found for the FAS-dysmorphic+ group (53,1%) in infancy than those in the FAS-dysmorphic- group (35,6%). However, when a chi-square test was performed, no significant association was found between FAS-dysmorphism+ and insecure attachment in infancy, χ^2 (1) = 2.36, p = .125, with a small effect size, φ = -.18. Rates of insecure attachment were therefore not significantly higher in the infants in the FAS-dysmorphic+ group compared to infants from the FAS-dysmorphic- group.

Figure 5.

Percentage of Organised and Disorganised Attachment in Infants with FAS-Dysmorphism+ and FAS-Dysmorphism-



For the purpose of testing hypothesis 2, an additional variable was computed by collapsing the infant attachment classifications into another dichotomous variable denoting disorganised attachment versus organised attachment, the latter of which consisted of secure, avoidant, and resistant attachment classifications. To test this second hypothesis, a chi-square test was performed, using the attachment variables denoting disorganised and organised attachment. In this comparison, the association between FAS-dysmorphism+ and disorganised attachment in infancy was also not significant χ^2 (1) = 1.27, p = .259, with a small effect size, φ = .13. Hence, rates of disorganised attachment were not significantly higher in the infants in the FAS-dysmorphic+ (31,3%) group compared to infants from the FAS-dysmorphic- group (20%; Figure 5).

Caregiver-reported Adolescent Emotion Regulation

The third hypothesis of the study predicted that adolescents with FAS-related facial dysmorphism would demonstrate poorer caregiver-reported emotion regulation in comparison to adolescents without FAS-related facial dysmorphism, even after controlling for attachment security, as well as other potential covariates. To test this hypothesis, a series of hierarchical multiple regression analyses were conducted.

Descriptive Statistics for Emotion Regulation Measures

To begin, I examined the descriptive statistics for the 5 diagnostic groups on each of the emotion regulation subscales (see Table 6). For these analyses, alpha was set at α = .05. While the subsequent analyses only made use of the dichotomous exposure variable, I was interested to see whether the descriptive statistics suggested any group differences between the 4 exposed groups. Given previous literature on emotion regulation in FASD, I did not expect this to be the case. Next, I went on to examine the descriptive statistics according to FAS-dysmorphism on each of the emotion regulation subscales (see Table 7).

Table 6.

Performance on Emotion Regulation Subscales by Diagnostic Category Including HE Split

		FAS (*n*=10)	PFAS (*n*=10)	HE+ (*n*=4)	HE- (*n*=15)	Control (*n*=22)	*F*	*p*	Omega Squared
ARC									
	Reflection[a]	5.10 (1.91)	5.60 (2.95)	6.50 (1.00)	6.00 (2.07)	5.27 (2.33)	.50	.733	-.034
	Lack of Control[b]	5.60 (3.72)	3.70 (3.20)	4.50 (1.73)	2.27 (2.34)	3.45 (3.26)	1.88	.128	.054
	Suppression of Affect[c]	7.50 (4.14)	6.30 (2.75)	7.50 (3.32)	5.13 (2.95)	4.68 (3.17)	1.82	.137	.051
ERC[f]									
	Adaptive ER[d]	26.44 (3.40)	25.40 (3.41)	24.33 (1.53)	26.33 (3.20)	25.38 (3.73)	.42	.797	-.042
	Lability/ Negativity[e]	32.11 (6.64)	30.90 (8.91)	22.67 (2.89)	24.87 (4.73)	26.86 (8.04)	2.41	.061	.088

Note. Values are mean (SD). FAS = fetal alcohol syndrome; PFAS = partial FAS; HE+ = heavily exposed nonsyndromal with subtle facial dysmorphism; HE- = heavily exposed nonsyndromal without facial dysmorphism. [a]Lower score indicates poorer ability to reflect. [b]Higher score indicates greater lack of control. [c]Higher score indicates greater suppression of affect. [d]Lower score indicates poorer adaptive regulation. [e]Higher score indicates greater lability and negativity. [f]Data missing for 1 adolescent in the FAS group, 1 adolescent in the HE- group, and 1 adolescent in the control group.

Five group comparison. No between group differences were found for the *ARC Reflection*, *ARC Lack of Control*, *ARC Suppression of Affect*, *ERC Adaptive Emotion Regulation*, and *ERC Lability/Negativity* subscales when all 5 diagnostic groups were compared.

Table 7.

Performance on Emotion Regulation Subscales by FAS-Dysmorphism

		FAS-dysmorphic+ (*n*=24)	FAS-dysmorphic- (*n*=37)	*F*	*p*	Omega Squared
ARC						
	Reflection[a]	5.54 (2.28)	5.57 (2.23)	.00	.965	-.017
	Lack of Control[b]	4.62 (3.25)	2.97 (2.95)	4.21	.045	.050
	Suppression of Affect[c]	7.00 (3.39)	4.86 (3.05)	6.54	.013	.083
ERC[f]						
	Adaptive ER[d]	25.68 (3.18)	25.78 (3.51)	.01	.917	-.017
	Lability/Negativity[e]	30.27 (7.84)	26.03 (6.85)	4.70	.034	.060

Note. Values are mean (SD). FAS-dysmorphic+ = FAS+PFAS+HE-dysmorphic; FAS-dysmorphic- = HE-nondysmorphic+controls. [a]Lower score indicates poorer ability to reflect. [b]Higher score indicates greater lack of control. [c]Higher score indicates greater suppression of affect. [d]Lower score indicates poorer adaptive regulation. [e]Higher score indicates greater lability and negativity. [f]Data missing for 2 adolescents in the FAS-dysmorphic+ group, and 1 in the FAS-dysmorphic- group.

Two diagnostic groups. No between-group differences were found for the *ARC Reflection* subscale. A significant between-group difference was found for the ARC *Lack of Control* subscale, with a small to medium effect size (ω^2 = .050). The FAS-dysmorphic+ group were reported to exhibit a greater lack of control than those in the FAS-dysmorphic- groups. A significant between-group difference was also found for *ARC Suppression of Affect* subscale, with the FAS-dysmorphic+ group demonstrating greater suppression of affect. The size of this effect was medium (ω^2 = .083) No significant between-group difference was found for the *ERC Adaptive Emotion Regulation* subscale. A significant between-group difference was found for the *ERC Lability/Negativity* subscale, with a medium effect size (ω^2

= .060). The FAS-dysmorphic+ group were reported by their caregivers as exhibiting greater lability and negativity when compared to the FAS-dysmorphic- group.

Hierarchical Multiple Regression Analyses

Several hierarchical multiple regression analyses were conducted to examine whether FAS-dysmorphism was significantly associated with the scores on the *ARC* and *ERC* subscales (emotion regulation outcome variables) in adolescence, over and above the influence of infant attachment security, as well as other predictor variables. Decisions regarding which variable to add at each step of the model were made based on previous findings in the literature. Each model was then tested in a hierarchical fashion to determine the contributions of each variable in predicting each emotion regulation outcome variable.

To begin, in step 1, I entered age at adolescent assessment. As previously established, there are age differences in the use of different emotion regulation strategies. Whereas these age-related differences have been found to be emotion-specific and the measures used in this study were generalized to all emotions, I was nevertheless interested to examine the association of age with each emotion regulation subscale (Zimmermann & Iwanski, 2014). I entered sex in step 2, as previous literature has suggested differences in the types of regulation strategies most often employed by females and males (Sanchis-Sanchis et al., 2020; Zlomke & Hahn, 2010). Previous research has established the association between a lower household SES and the development of adaptive emotion regulation (Herd et al., 2020; Holmes et al., 2019) and, therefore, I entered SES in step 3. Prenatal smoking was entered in step 4 as prenatal cigarette exposure has been found to be associated with regulation difficulties (Froggatt et al., 2020; Huijbregts et al., 2008). Although research on the association between IQ and emotion regulation has yielded inconsistent findings, these studies have generally made use of samples that have not exhibited the wide range of IQ

scores often found in individuals with FASD (Graziano et al., 2007; Zantinge et al., 2017). As such, I was interested to see whether the impact of FAS-dysmorphism could be seen over and above the effect of IQ. I therefore entered adolescent IQ in step 5. Infant attachment security was entered in step 6. Given the association between attachment pattern, specifically attachment security, and emotion regulation reported in the literature (Mikulincer & Shaver, 2019; Moutsiana et al., 2014), I expected that attachment security in infancy would predict scores on the emotion regulation subscales in adolescence. Lastly, given the third hypothesis of the current study, FAS-dysmorphism was added in the 7th and final step of the regression model in order to ensure that any association observed between FASD- related dysmorphism and the particular emotion regulation outcome was over and above the variance explained by the previous variable, infant attachment security. Each of the models described below was built based on the same rationale.

Model 1: Association Between FAS-Dysmorphism and ARC Reflection Scores. A hierarchical regression analysis was conducted to examine the association between FAS-dysmorphism and scores on the *ARC Reflection* subscale, after controlling for various potential predictors. Table 8 presents the zero-order intercorrelations between the outcome variable and the various potential predictors. As the table shows, age at adolescent assessment, sex, and adolescent IQ correlated significantly with the outcome variable. However, FAS-dysmorphism did not correlate with the outcome variable, suggesting that an association would not emerge. As expected, significant correlations were shown between several of the potential predictors. All the tolerance and the variance inflation factor (VIF) scores for this analysis were determined to be acceptable, indicating no multicollinearity (see Appendix C; Field, 2013).

Table 8.

Intercorrelations Between ARC Reflection Score and Potential Predictors

	1.	2.	3.	4.	5.	6.	7.	8.
1. ARC Reflection	1							
2. Age at adolescent assessment	.25*	1						
3. Sex	.24*	-.13	1					
4. SES[a]	.12	-.06	.08	1				
5. Prenatal smoking	.03	.24*	.07	-.26*	1			
6. Adolescent IQ	.29*	-.10	.14	.27*	-.17	1		
7. Attachment security	.05	-.12	.07	-.07	-.06	.05	1	
8. FAS-dysmorphism	-.02	.36*	.01	-.13	.19	-.18	-.24*	1

Note. [a]Based on Hollingshead Four Factor Index of Social Status (2011).

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

In step 1 of the analysis, I entered age at adolescent assessment to investigate whether it predicted scores on the *ARC-P Reflection* subscale in adolescence. The R^2 change accounted for by age fell just short of significance (*Sig. ΔF* = .057). Introducing adolescent sex into the model at step 2 accounted for an additional 7.3% of variation in scores on the *ARC-P Reflection* subscale. This change in R^2 was significant (*Sig. ΔF* = .035). Adding SES into the model at step 3, showed that SES did not account for a significant change in the amount of variance explained (*Sig. ΔF* = .372). Smoking, which was added at step 4, also did not account for a significant change in the amount of variance explained (*Sig. ΔF* = .817). In step 5, I introduced adolescent IQ into the model, which accounted for an additional 7% of variation in scores on the *ARC-P Reflection* subscale. This change in R^2 was significant (*Sig. ΔF* = .035). Adding infant attachment security at step 6, however, did not account for a significant change in variance explained (*Sig. ΔF* = .652). In the final step, I added FAS-dysmorphism into the model, which did not account for a significant change in the amount of variance explained (*Sig. ΔF* = .517).

As Table 9 shows, in the final model, only age at adolescent assessment and adolescent IQ were significantly associated with scores on the *ARC Reflection* subscale. From the beta values we can see that age was the strongest predictor of scores on this subscale. The positive beta values indicate that being older and having a higher IQ were associated with higher scores on the *ARC Reflection* subscale.

Table 9.

Coefficients for Model 7: FAS-Dysmorphism and ARC Reflection Scores

		Unstandardized coefficients		*Standardized coefficients*		
Model	Predictors	*B*	Std. Error	*β*	*t*	*p*
7	(Constant)	18.82	7.95		-2.37	.022
	Age	1.10	.44	.35	2.51	.015
	Sex	1.07	.58	.24	1.86	.069
	SES	.01	.04	.04	.29	.771
	Prenatal smoking	.00	.06	.01	.05	.961
	IQ	.04	.02	.27	2.02	.049
	Attachment security	.17	.58	.04	.30	.767
	FAS-dysmorphism	-.41	.63	-.09	-.65	.517

Model 2: Association Between FAS-Dysmorphism and ARC Lack of Control Scores. A hierarchical regression analysis was conducted to examine the association between FAS-dysmorphism and scores on the *ARC Lack of Control* subscale, after controlling for various potential predictors. Table 10 presents the zero-order intercorrelations between the outcome variable and the various potential predictors. As the table shows, adolescent IQ,

infant attachment security, and FAS-dysmorphism correlated significantly with the outcome variable. All the tolerance and the variance inflation factor (VIF) scores for this analysis were determined to be acceptable, indicating no multicollinearity (see Appendix C; Field, 2013).

Table 10.

Intercorrelations Between ARC Lack of Control Score and Potential Predictors

	1.	2.	3.	4.	5.	6.	7.	8.
1. ARC Lack of Control	1							
2. Age at adolescent assessment	.13	1						
3. Sex	-.11	-.13	1					
4. SES[a]	-.04	-.06	.08	1				
5. Prenatal smoking	.10	.24*	.07	-.26*	1			
6. Adolescent IQ	-.42**	-.10	.14	.27*	-.17	1		
7. Attachment security	-.31**	-.12	.07	-.07	-.06	.05	1	
8. FAS-dysmorphism	.23*	.36*	.01	-.13	.19	-.18	-.24*	1

Note. [a]Based on Hollingshead Four Factor Index of Social Status (2011).

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

Age at adolescent assessment was entered first in the model to investigate whether it predicted scores on the *ARC Lack of Control* subscale in adolescence. This model was not significant, $F(1,56) = .97$, $p = .328$, and age only explained 1.7% of the variation in scores. At step 2, I added adolescent sex into the model which did not account for a significant change in the amount of variance explained (*Sig.* $\Delta F = .500$). Introducing SES at step 3 also did not account for a significant change in the amount of variance explained (*Sig.* $\Delta F = .844$). At step 4, I added prenatal smoking into the model, which did not account for a significant change in the amount of variance explained (*Sig.* $\Delta F = .639$). Introducing adolescent IQ into the model in the next step, accounted for an additional 16% of variation in scores on the *ARC Lack of Control* subscale (*Sig.* $\Delta F = .002$). The addition of infant attachment security in step 6 accounted for an additional 7.3% in the amount of variance explained (*Sig.* $\Delta F = .029$).

Finally, in step 7, I added FAS-dysmorphism into the model, which did not account for a significant change in the amount of variance explained over and above the variables previously entered into the model (*Sig. ΔF* = .498).

As can be seen in Table 11 below, only adolescent IQ and infant attachment security were found to be significantly associated with scores on the *ARC Lack of Control* subscale in the final model. From the beta values, it appears that adolescent IQ was the strongest predictor of scores on this subscale. The negative beta values indicate that a lower IQ, as well as insecure attachment in infancy, were associated with higher scores on the *ARC Lack of Control* subscale in adolescence.

Table 11.

Coefficients for Model 7: FAS-Dysmorphism and ARC Lack of Control Scores

		Unstandardized coefficients		*Standardized coefficients*		
Model	Predictors	*B*	Std. Error	*β*	*t*	*p*
7	(Constant)	8.47	10.73		.79	.434
	Age	.12	.59	.03	.21	.837
	Sex	-.23	.78	-.04	-.30	.768
	SES	.02	.05	.07	.50	.620
	Prenatal smoking	.00	.07	.00	.01	.995
	IQ	-.08	.03	-.40	-3.09	.003
	Attachment security	-1.59	.78	-.26	-2.03	.048
	FAS-dysmorphism	.59	.86	.09	.68	.498

Model 3: Association Between FAS-Dysmorphism and ARC Suppression of Affect Scores. A hierarchical regression analysis was conducted to examine the association between FAS-dysmorphism and scores on the *ARC Suppression of Affect* subscale, after controlling for various potential predictors. Table 12 below presents the zero-order intercorrelations between the outcome variable and the various potential predictors. As the table shows, infant attachment security and FAS-dysmorphism correlated significantly with the outcome variable. There were also some significant correlations between several of the potential predictors. All the tolerance and the variance inflation factor (VIF) scores for this analysis were determined to be acceptable, indicating no multicollinearity (see Appendix C; Field, 2013).

Table 12.

Intercorrelations Between ARC Suppression of Affect Score and Potential Predictors

	1.	2.	3.	4.	5.	6.	7.	8.
1. ARC Suppression of Affect	1							
2. Age at adolescent assessment	.07	1						
3. Sex	-.03	-.13	1					
4. SES[a]	-.11	-.06	.08	1				
5. Prenatal smoking	.01	.24*	.07	-.26*	1			
6. Adolescent IQ	-.05	-.10	.14	.27*	-.17	1		
7. Attachment security	-.31**	-.12	.07	-.07	-.06	.05	1	
8. FAS-dysmorphism	.30*	.36*	.01	-.13	.19	-.18	-.24*	1

Note. [a]Based on Hollingshead Four Factor Index of Social Status (2011).

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

Age at adolescent assessment was entered into the model first to investigate whether it predicted scores on the *ARC Suppression of Affect* subscale in adolescence. The R^2 change accounted for by age was not significant (*Sig.* $\Delta F = .591$). Adolescent sex, which was introduced into the model at step 2, did not account for a significant change in the amount of

variance explained (*Sig. ΔF* = .891). SES, which was added at step 3, did not account for a significant change in the amount of variance explained (*Sig. ΔF* = .425). At step 4, I added prenatal smoking into the model. This variable also did not account for a significant change in the amount of variance explained (*Sig. ΔF* = .819). Adolescent IQ, which was added in step 5, did not account for any additional variation in scores on the *ARC Suppression of Affect* subscale, (*Sig. ΔF* = .905). Attachment security in inafncy, which was added at step 6, accounted for an additional 10% in the amount of variance explained. This change in R^2 was significant (*Sig. ΔF* = .020). In the final step, FAS-dysmorphism was introduced into the model. FAS-dysmorphism fell short of significance in terms of the amount of variance it explained (*Sig. ΔF* = .091).

In Table 13 below, we can see that only infant attachment security was significantly associated with scores on the *ARC Suppression of Affect* subscale in the final model. The negative beta value for this variable indicates that insecure attachment in infancy was associated with higher scores on the *ARC Suppression of Affect* subscale in adolescence.

Table 13.

Coefficients for Model 7: FAS-Dysmorphism and ARC Suppression of Affect Scores

Model	Predictors	Unstandardized coefficients		*Standardized coefficients*		
		B	Std. Error	*β*	*t*	*p*
7	(Constant)	10.20	12.20		.84	.407
	Age	-.19	.67	-.04	-.28	.784
	Sex	-.04	.89	-.01	-.05	.963
	SES	-.05	.06	-.13	-.94	.354
	Prenatal smoking	-.04	.09	-.07	-.51	.613
	IQ	.01	.03	.03	.21	.838
	Attachment security	-1.80	.89	-.27	-2.02	.049
	FAS-dysmorphism	1.68	.97	.25	1.72	.091

Model 4: Association Between FAS-Dysmorphism and ERC Adaptive Emotion Regulation. A hierarchical regression analysis was conducted to examine the association between FAS-dysmorphism and scores on the *ERC Adaptive Emotion Regulation* subscale, after controlling for various potential predictors. Table 14 presents the zero-order intercorrelations between the outcome variable and the various potential predictors. As the table shows, none of the predictor variables correlated significantly with the outcome variable. The fact that FAS-dysmorphism did not correlate with the outcome variable suggested that an association between FAS-dysmorphism and *ERC Adaptive Emotion Regulation* scores would not emerge. There were, however, some significant correlations between several of the potential predictors. All the tolerance and the variance inflation factor

(VIF) scores for this analysis were determined to be acceptable, indicating no multicollinearity (see Appendix C; Field, 2013).

Table 14.

Intercorrelations Between ERC Adaptive Emotion Regulation Score and Potential Predictors

	1.	2.	3.	4.	5.	6.	7.	8.
1. ERC Adaptive Emotion Regulation	1							
2. Age at adolescent assessment	.03	1						
3. Sex	.18	-.14	1					
4. SES[a]	-.16	-.08	.06	1				
5. Prenatal smoking	.16	.22*	.07	-.28*	1			
6. Adolescent IQ	.18	-.07	.16	.20*	-.15	1		
7. Attachment security	.13	-.16	.07	-.09	-.10	.08	1	
8. FAS-dysmorphism	-.03	.42**	.02	-.11	.23*	-.21	-.20*	1

Note. [a]Based on Hollingshead Four Factor Index of Social Status (2011).

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

Age at adolescent assessment was added at step 1 to investigate whether it predicted scores on the *ERC Adaptive Emotion Regulation* subscale. The R^2 change accounted for by age was not significant (*Sig.* $\Delta F = .850$). Adolescent sex, which was introduced in step 2, did not account for a significant change in the amount of variance explained (*Sig.* $\Delta F = .185$). SES, which was added in step 3 of the model, also did not account for a significant change in the amount of variance explained (*Sig.* $\Delta F = .217$). Prenatal smoking was introduced into the model in step 4. This variable did not account for a significant change in the amount of variance explained (*Sig.* $\Delta F = .493$). At step 5, adolescent IQ was introduced into the model. While adolescent IQ accounted for an additional 5% in the amount of variance explained, this change in R^2 fell short of significance (*Sig.* $\Delta F = .095$). Attachment security in infancy, which was added at step 6, did not account for a significant amount of variance explained (*Sig.* $\Delta F =$

.442). In the final step, I added FAS-dysmorphism into the model. Again, FAS-dysmorphism did not account for a significant amount in the variance of *ERC Adaptive Emotion Regulation* scores (*Sig. ΔF* = .786).

As Table 15 below shows, none of the proposed predictor variables were significantly associated with scores on the *ERC Adaptive Emotion Regulation* subscale in the final model.

Table 15.

Coefficients for Model 7: FAS-Dysmorphism and ERC Adaptive Emotion Regulation Scores

		Unstandardized coefficients		*Standardized coefficients*		
Model	Predictors	*B*	Std. Error	*β*	*t*	*p*
7	(Constant)	17.14	12.99		1.32	.194
	Age	.24	.72	.05	.33	.741
	Sex	.95	.93	.14	1.02	.312
	SES	-.08	.06	-.19	-1.29	.202
	Prenatal smoking	.08	.09	.14	.93	.356
	IQ	.05	.03	.22	1.53	.133
	Attachment security	.67	.93	.10	.72	.474
	FAS-dysmorphism	-.29	1.05	-.04	-.27	.786

Model 5: Association Between FAS-Dysmorphism and ERC Lability/Negativity. A hierarchical regression analysis was conducted to examine the association between FAS-dysmorphism and scores on the *ERC Lability/Negativity* subscale, after controlling for various potential predictors. Table 16 presents the zero-order intercorrelations between the outcome variable and the various potential predictors. As the table shows, SES, prenatal

smoking, adolescent IQ, attachment security, and FAS-dysmorphism all correlated significantly with the outcome variable. There were also some significant correlations between several of the potential predictors. All the tolerance and the variance inflation factor (VIF) scores for this analysis were determined to be acceptable, indicating no multicollinearity (see Appendix C; Field, 2013).

Table 16.

Intercorrelations Between ERC Lability/Negativity Score and Potential Predictors

	1.	2.	3.	4.	5.	6.	7.	8.
1. ERC Lability/Negativity	1							
2. Age at adolescent assessment	.12	1						
3. Sex	-.12	-.14	1					
4. SES[a]	-.32**	-.08	.06	1				
5. Prenatal smoking	.29*	.22*	.07	-.28*	1			
6. Adolescent IQ	-.43**	-.07	.16	.20*	-.15	1		
7. Attachment security	-.35**	-.16	.07	-.09	-.10	.08	1	
8. FAS-dysmorphism	.29*	.42**	.02	-.11	.23*	-.21	-.20*	1

Note. [a]Based on Hollingshead Four Factor Index of Social Status (2011).
*$p < .05$. **$p < .01$.

Age at adolescent assessment was introduced into the model at step 1. This change in R^2 was not significant (*Sig. ΔF* = .370). Adolescent sex, which was introduced in step 2, did not account for a significant change in the amount of variance explained (*Sig. ΔF* = .471). SES, which was introduced in step 3, accounted for an additional 9.3% in the amount of variance explained (*Sig. ΔF* = .023). Prenatal smoking was added in step 4. The addition of prenatal smoking did not account for a significant change in the amount of variance explained (*Sig. ΔF* = .122). Adolescent IQ, which was added in step 5, accounted for an additional 10.4% in the amount of variance explained. This change in R^2 was significant (*Sig. ΔF* = .011). Infant attachment security, which was added in step 6, accounted for an

additional 10.1% in the amount of variance explained. This change in R^2 was also significant (*Sig. ΔF* = .008). In step 7, I added FAS-dysmorphism into the model. FAS-dysmorphism did not account for a significant amount of variance in *ERC Lability/Negativity* scores (*Sig. ΔF* = .298).

As Table 17 below shows, only adolescent IQ and infant attachment security were found to be significantly associated with scores on the *ERC Lability/Negativity* subscale in the final model. The beta values indicate that infant attachment security was the strongest predictor of scores on this subscale. The negative beta values indicate that a lower IQ, as well as insecure attachment in infancy, were associated with higher scores on the *ERC Lability/Negativity* subscale in adolescence.

Table 17.

Coefficients for Model 7: FAS-Dysmorphism and ERC Lability/Negativity Scores

		Unstandardized coefficients		*Standardized coefficients*		
Model	Predictors	*B*	Std. Error	*β*	*t*	*p*
7	(Constant)	55.80	24.87		2.24	.029
	Age	-.65	1.38	-.06	-.47	.642
	Sex	-.85	1.78	-.06	-.47	.638
	SES	-.18	.11	-.21	-1.66	.104
	Prenatal smoking	.19	.17	.14	1.13	.262
	IQ	-.15	.06	-.29	-2.34	.024
	Attachment security	-4.65	1.79	-.31	-2.60	.012
	FAS-dysmorphism	2.12	2.02	.14	1.05	.298

Discussion

The current study aimed to investigate the association between FAS-dysmorphism and attachment classification, as well as to determine the extent to which FAS-dysmorphism was associated with emotion regulation difficulties often reported in this population over and above the impact of infant attachment security. I tested three specific hypotheses. The first of these proposed that children with FAS-dysmorphism+ would exhibit higher rates of insecure attachment than those with FAS-dysmorphism-. The second hypothesis proposed that children with FAS-dysmorphism+ would exhibit higher rates of disorganised attachment than those with FAS-dysmorphism-. My results did not support the hypothesized association between FAS-dysmorphism+ and insecure attachment ($\varphi = -.18$), nor did they support the hypothesized association between FAS-dysmorphism+ and disorganised attachment ($\varphi = .13$) The third hypothesis proposed that FAS-dysmorphism+ would be associated with impairment in emotion regulation over and above what could be explained by attachment security. This hypothesis was also not supported by my results. I found that, when controlling for the effect of attachment security (as well as other potential predictors), FAS-dysmorphism+ was not significantly associated with maladaptive emotion regulation. While individuals with FASD are often reported to have difficulties regulating their emotions, this study's findings appear to suggest that other variables, including IQ and infant attachment security may account for the emotion regulation impairment in individuals with FASD. In the section below, I discuss the findings relating to each of the hypotheses in greater detail, contextualizing them within previously published literature. I then go on to consider the implications of the study's findings, followed by a discussion of the strengths, limitations, and recommendations for future research.

Attachment

Prior to investigating the association between FAS-dysmorphism and attachment patterns in the current sample, I examined the distribution of attachment patterns among the infants in the sample. Only a small body of research has examined the distribution of attachment patterns in South African samples to date (Cooper et al., 2009; Hay et al., 2004; Jacobson & Jacobson, 2013; Tomlinson, 2001; Tomlinson, Cooper, & Murray, 2005). I was therefore interested to determine the extent to which the distribution of attachment patterns in the current sample was consistent with that found in other South African samples, as well as how it compared to that found in international samples.

Consistent with the findings by Hay et al. (2004) in the larger Cape Town Longitudinal Cohort sample of infants from which this subsample is drawn, of the infants in the current sample, 57.1% were classified as secure, while 42.9% were classified as one of the insecure attachment classifications. This rate of secure attachment was fairly consistent with the rate of secure attachment reported in previous studies examining low-income samples (Ijzendoorn et al., 1999; Tomlinson et al., 2005; True et al., 2001). For instance, in a study conducted by Tomlinson, Cooper, and Murray (2005) with a sample of mother-infant dyads living in Khayelitsha, a low-income community in Cape Town, 61.9% of infants were found to be securely attached, while 38.1% were found to be insecure. Furthermore, in a study conducted by True et al. (2001) among mother-infant dyads from the Dogon ethnic group in Mali, 67% of the infants were found to be secure, while 33% were found to be insecure. It is interesting to note here that similar rates of secure attachment have also been found in middle income samples. For instance, in a meta-analysis conducted by Ijzendoorn and colleagues (1999), they found that the average rate of secure attachment in infants from middle income, North American samples was 62%, while the average rate of insecure attachment was 38%. This, however, is not too surprising, given that the rates of secure and,

in turn, insecure attachment have been found to be relatively consistent across several contextual factors such as culture or level of income (Archer et al., 2015; Voges et al., 2019). While secure attachment has generally been found to be the modal attachment pattern regardless of context, the prevalence of the other attachment patterns appears to be impacted by the specific context much more.

Consistent with the distribution in the larger Cape Town Longitudinal Cohort sample (Hay et al., 2004), in the current sample, 1.3% of infants were classified as avoidant, while 19.5% were classified as resistant. The finding that only 1.3% of infants (i.e. 1 infant) were classified as avoidant is well below the 15% reported in the meta-analysis conducted by Ijzendoorn et al. (1999). However, if we compare the rate of avoidant attachment found in this study to that reported by studies conducted with low to middle income countries (LMICs), the current findings are comparable. For instance, in the Tomlinson et al. (2005) study, only 4.1% of infants were classified as avoidant. Furthermore, in a study conducted among mother-infant dyads from the Dogon ethnic group in Mali, none of the infants were classified as avoidant (True et al., 2001). Several explanations have been put forth regarding the low rate of avoidant attachment found in these previous studies of developing and low-income contexts. For instance, Tomlinson et al. (2005) pointed out that in Khayelitsha (the community from which they drew their sample), many homes only comprise of one room. As such, mothers in their sample often conducted the majority of their daily activities in the presence of the infant. Tomlinson et al. (2005) also proposed that the manner in which the infants in their sample were breast fed on demand contributed to their low rate of avoidant attachment. That is, given that the infant's attachment and feeding systems were inextricably connected, the development of avoidant attachment with the mother would be unlikely. In the current sample, one-roomed homes and the tendency for on-demand feeding were similarly

prevalent. It is therefore likely that these factors also contributed to the low rates of avoidant attachment observed in this sample.

Consistent with the findings in the larger Hay et al. (2004) sample, 22.1% of the infants in the current sample were classified were classified as having disorganised attachment to their caregiver. This rate of disorganised attachment was higher than the 15% reported in middle-income samples evaluated in the meta-analysis conducted by Ijzendoorn et al. (1999). However, once again, when we consider studies conducted in LMICs, the rate of disorganised attachment found here is comparable. For instance, Tomlinson et al. (2005) found the rate of disorganised attachment among mother-infant dyads in Khayelitsha to be 25.8%. In the study conducted by True et al. (2001) with mother-infant dyads from the Dogon ethnic group in Mali, the rate of disorganised attachment was similarly high at 25%. The rate of disorganised attachment found among the infants in this sample is therefore comparable to that found in these previous studies conducted in LMICs (Tomlinson et al., 2005; True et al., 2001).

Association Between FAS-Dysmorphism and Insecure Attachment

The first hypothesis of the current study proposed that children with FAS-dysmorphism+ would have a higher rate of insecure attachment in comparison to those with FAS-dysmorphism-. This hypothesis was not supported. A chi-squared analysis revealed that infants with FAS-dysmorphism+ did not demonstrate a significantly higher rate of insecure attachment in comparison to infants with FAS-dysmorphism- ($\varphi = -.18$). Thus, while the rate of insecure attachment in the FAS-dysmorphism+ group (53.1%) was higher than that previously reported in LMICs (38% - Ijzendoorn et al., 1999; 38.1% - Tomlinson et al., 2005; 33% - True et al., 2001) it was not significantly different from that found in the FAS-dysmorphism- group (35.6%).

The finding that FAS-dysmorphism+ was not associated with insecure attachment was consistent with a study conducted by Bergin and McCollough (2009) which failed to find an association between PAE and insecure attachment. Similar to the approach the current study took of grouping all individuals with FAS-dysmorphism+ and comparing them to individuals who did not exhibit FAS-dysmorphism-, Bergin and McCollough (2009) grouped all alcohol-exposed infants together, irrespective of level of exposure, and compared them to unexposed infants. Two previous studies by O'Connor et al. (1987) and O'Connor et al. (2002) found higher rates of insecure attachment particularly in infants born to mothers who drank moderately to heavily during pregnancy (70% and 80%, respectively). Hay et al. (2004) also found that infants with FAS demonstrated a higher rate of insecure attachment compared with the PFAS, HE, and control groups. The findings from these studies reporting higher rates of insecure attachment primarily in infants with heavy PAE suggest that the absence of a relation between FAS-dymorphism+ and insecure attachment in the present study is likely due to the grouping of infants with FAS and PFAS together with less heavily exposed children with facial dysmorphism. Thus, although HE dysmorphism+ is associated with poorer cognitive outcomes (Suttie et al., 2013), it is not related to insecure attachment.

It is important to note here that the finding that infants with FAS-dysmorphism+ did not demonstrate a significantly higher rate of insecure attachment in comparison to infants with FAS-dysmorphism- had a small effect size ($\varphi = -.18$). However, given the small sample size, it is possible that the presence of small effects went undetected due to limited statistical power. It is possible that a larger sample would have been able to detect these small effects.

Association Between FAS-Dysmorphism and Disorganised Attachment

The second hypothesis of the current study proposed that infants with FAS-dysmorphism+ would also demonstrate a higher rate of disorganised attachment in comparison to those with FAS-dysmorphism-. This second hypothesis was not supported. A

chi-squared analysis revealed that infants with FAS-dysmorphism+ did not demonstrate a significantly higher rate of disorganised attachment in comparison to infants with FAS-dysmorphism- ($\varphi = .13$). That is, while the rate of disorganised attachment in the FAS-dysmorphism+ group (31.3%) was higher than that previously reported in LMICs (25% - Ijzendoorn et al., 1999; 25.8% - Tomlinson et al., 2005; 25% - True et al., 2001), it was not significantly different from that found in the FAS-dysmorphism- group (20%).

Previous findings from O'Connor et al. (1987) reported that the infants in their sample who were born to moderate-to-heavy drinking mothers demonstrated a greater incidence of disorganized attachment. Jacobson and Jacobson (2013) also demonstrated that infants with FAS exhibited a higher rate of disorganised attachment compared with the PFAS, HE, and control groups. As noted above with respect to insecure attachment, the findings from these studies reporting higher rates of disorganization in infants with heavy PAE suggest that the absence of a relation between FAS-dymorphism+ and disorganized attachment in the present study is likely due to the grouping of infants with FAS and PFAS together with less heavily exposed children with facial dysmorphism. Therefore, although HE dysmorphism+ is associated with poorer cognitive outcomes (Suttie et al., 2013), it is not related to disorganized attachment.

Once again, it is worthwhile to note here that the finding that infants with FAS-dysmorphism+ did not demonstrate a significantly higher rate of insecure attachment in comparison to infants with FAS-dysmorphism- had a small effect size ($\varphi = .13$). However, given the small sample size, it is possible that the presence of small effects went undetected due to limited statistical power. It is possible that a larger sample would have been able to detect these small effects.

Emotion Regulation

Association Between FAS-Dysmorphism and Emotion Regulation

The third hypothesis of the current study predicted that adolescents with FAS-related facial dysmorphism would demonstrate poorer caregiver-reported emotion regulation in comparison to adolescents without FAS-related facial dysmorphism, even after controlling for attachment security, as well as other covariates. This hypothesis was not supported for the two adaptive emotion regulation outcome variables (i.e. *ARC Reflection* and *ERC Adaptive Emotion Regulation*) nor the three maladaptive emotion regulation outcome variables (i.e. *ARC Lack of Con0trol*, *ARC Suppression of Affect* and *ERC Lability/Negativity*). That is, FAS-dysmorphism+ was not found to be significantly associated with either of the adaptive emotion regulation outcome variables, nor any of the maladaptive regulation outcome variables, after controlling for the effect of attachment and other potential covariates. It is interesting to note, however, that before the influence of other variables were controlled for in the hierarchical multiple regression analyses, between-group differences existed for all three of the maladaptive emotion regulation subscales. This suggests that while adolescents with FAS-dysmorphism+ did exhibit more maladaptive emotion regulation, a third variable likely accounts for this association. This will be discussed further below.

To consider why FAS-dysmorphism did not predict either of the adaptive emotion regulation outcome variables in the sample, one particular unpublished intervention study, which sought to examine the construct of self-awareness with FASD, may provide some insight. In this study it was found that, even before beginning the intervention, adolescents with FASD did not demonstrate any impairment in self-awareness (Joly, 2020). That is, they were able to recognize, and reflect on, their internal arousal states accurately. It is important to note here that the *ARC Reflection* as well as the *ERC Adaptive Emotion Regulation* subscales both tap into the individual's self-awareness/ability to reflect on their emotional

state. The Joly (2020) findings may, therefore, provide a plausible explanation for why the current study did not find FAS-dysmorphism to be associated with the adaptive emotion regulation outcomes.

The finding that FAS-dysmorphism was not associated with any of the three maladaptive emotion regulation outcomes was unexpected. This is because an established body of literature has suggested that individuals with PAE/FASD do indeed present an impaired ability to regulate emotions. Evidence for the notion that individuals with FASD have difficulty regulating their emotions has generally come from studies examining the behaviour of individuals with FASD. For instance, children and adolescents with FASD have been reported to frequently demonstrate greater negative affectivity and mood lability, in addition to a range of other internalizing and externalizing symptoms, in comparison to their typically developing peers (Khoury et al., 2018; Nash et al., 2006; Paley et al., 2005; Tsang et al., 2016). The study of psychiatric conditions often associated with FASD has also provided support for the notion that individuals with FASD have difficulty regulating their emotions. Higher rates of internalizing disorders, such as depression and anxiety, as well as higher rates of externalizing disorders, such as ADHD and conduct disorder, have been reported amongst individuals with FASD (Fryer, McGee, Matt, Riley, & Mattson, 2007; O'Connor & Kasari, 2000; O'Connor et al., 2002; O'Connor & Paley, 2009; Temple et al., 2019). While a higher incidence of behavioural problems as well as an increased prevalence of psychiatric conditions has enabled us to infer the presence of emotion regulation difficulties in individuals with FASD, none of these aforementioned studies have examined emotion regulation directly. As previously mentioned, Shields and Cicchetti (1997) have advocated for the importance of examining the *processes* of emotion regulation that ultimately underlie the emergence of these behavioural problems and psychiatric conditions.

As previously mentioned, only a few studies have sought to interrogate the processes of emotion regulation more directly in FASD. For instance, a small number of studies have made use of the BRI, a subscale of the BRIEF (Gioia et al., 2000) which specifically examines skills related to the regulation of emotions and behaviours. In these previous studies, individuals with FASD have been found to score in the clinical range for the BRI, indicating a significant impairment in the ability to regulate emotions and behaviours (Rasmussen et al., 2007; Schonfeld et al., 2006). Furthermore, an intervention study conducted by Petrenko, Pandolfino, and Robinson (2017) specifically made use of the ERC (Shields & Cicchetti, 1997) to interrogate emotion regulation in their sample. The children in this study exhibited pre-intervention scores on both subscales of the ERC (i.e. *ERC Adaptive Emotion Regulation* and *ERC Lability/Negativity*) that were indicative of difficulties in regulating their emotions (Petrenko et al., 2017). These studies seem to suggest that individuals with FASD present with significant impairment in relation to the processes they use to regulate their emotions. However, as previously mentioned, these studies did not include an unexposed, control group. As a result, these studies were not able to rule out the potential influence of other environmental or individual factors that may be associated with FASD on their participants' ability to regulate emotions. For instance, individuals with FASD often come from a lower SES and present with a lower IQ. Given that both a lower SES, and a lower IQ have been found to be associated with more maladaptive emotion regulation, it is important to control for the potential effect of these factors (Herd et al., 2020; Holmes et al., 2019; Zantinge et al., 2017).

One important consideration that may also explain the null findings regarding the association between FAS-dysmorphism and maladaptive emotion regulation in the current study is that relating to the statistical power of the current study. Specifically, given the small sample size, it is possible that the presence of small effects went undetected due to limited

statistical power. In order to determine whether this may have been the case, I examined the standardized betas for FAS-dysmorphism for each of the models I constructed. The standard betas for FAS-dysmorphism suggest the presence of a small effect for both the *ARC Suppression of Affect* (β = .25) and the *ERC Lability/Negativity* (β = .14) subscales. It is, therefore, possible that a larger sample size would have been able to detect these small effects.

Association of Covariates to Emotion Regulation

Given that FAS-dysmorphism did not account for the variability on the emotion regulation subscales, I was interested to examine which variables did account for some of this variability. Given the importance of attachment in this study, the role of attachment in relation to emotion regulation will be discussed in a separate section below. Age at adolescent assessment, adolescent sex, SES, prenatal smoking, and adolescent IQ were considered as potential covariates in the five hierarchical multiple regression models I ran. Of these potential covariates, only age at adolescent assessment and IQ were found to be associated with any of the emotion regulation subscales.

Age at adolescent assessment was found to account for a significant amount of variance in scores on the *ARC Reflection* subscale, with older adolescents performing better on this subscale. This finding is consistent with previous research by Zimmermann and Iwanski (2014), which demonstrated that adaptive emotion regulation, which includes the ability to reflect on one's emotional state, follows a U-shaped trend. That is, the use of adaptive strategies is high between the ages of 12-14, dips between 15-17, and then increases again after the age of 17. It is interesting to note, however, that age at adolescent assessment was not found to account for a significant amount of variance in scores on the *ERC Adaptive Emotion Regulation* subscale. The *ERC Adaptive Emotion Regulation* subscale taps into multiple aspects of adaptive emotion regulation, including empathy and appropriate social

responsiveness, as well as the ability to reflect on one's emotional state (Shields & Cicchetti, 1997). As such, it is possible that each construct was differentially related to age, which would explain why no uniform association with age emerged for this outcome variable.

Adolescent IQ was also found to account for a significant amount of variability in scores on the *ARC Reflection, ARC Lack of Control,* and *ERC Lability/Negativity* subscales. While a higher IQ was associated with higher scores on the *ARC Reflection* subscale, a lower IQ was associated with a higher score on the *ARC Lack of Control* and the *ERC Lability/Negativity* subscales. As previously mentioned, the few studies that have sought to examine the association between IQ and emotion regulation have yielded inconsistent results. However, it appears that the current study's finding that a higher IQ was associated with the ability to reflect on one's own emotional state, as measured by the *ARC Reflection* subscale, is consistent with findings from Zantinge et al. (2017), demonstrating that individuals with a higher IQ make use of more adaptive emotion regulation. Once again, it is interesting to note that IQ did not account for a significant amount of variance in scores on the *ERC Adaptive Emotion Regulation* subscale. However, as previously mentioned, the *ERC Adaptive Emotion Regulation* subscale taps into multiple aspects of adaptive emotion regulation. It is, therefore, possible that each construct was differentially related to IQ, which could explain why no uniform association with IQ emerged for this outcome variable.

In terms of the maladaptive emotion regulation subscales, the current study found an association between lower IQ and higher scores on the *ARC Lack of Control* and *ERC Lability/Negativity* subscales. It is important to note here that both of these subscales tap into emotional lability. However, this finding is inconsistent with previous findings. For instance, in a study conducted by Sobanski et al. (2010), it was determined that IQ was not associated with increased lability. However, this study excluded participants who had an IQ of below 70. Given that the current study included participants with an IQ as low as 52, it is possible

that the truncated range of IQ scores in the Sobanski et al. (2010) study prevented an association between IQ and lability from being detected.

An important issue to note regarding the use of IQ as a predictor in this study is the fact that FASD is almost always associated with lower IQ (Carmichael-Olson, Feldman, Streissguth, Sampson, & Bookstein, 1998; Carter et al., 2016; Jacobson et al., 2004; Mattson, Riley, Gramling, Delis, & Jones, 1997; Streissguth et al., 1991; Streissguth, Barr, Sampson, Darby, & Martin, 1989). It is, therefore, difficult to disentangle the effects of FASD from the effects of a lower IQ. Thus, although the current study included these two variables as separate predictors, and the multicollinearity statistics were found to be acceptable, the fact that IQ and FASD are not truly independent of one another makes it difficult to examine the FAS-dysmorphism effects over and above those of IQ. Future studies may, therefore, seek to consider IQ as a mediator of the effect of FAS-dysmorphism on emotion regulation.

Association of Attachment and Emotion Regulation

As previously mentioned, the attachment relationship has been proposed as an important role player in the development of emotion regulation (Cassidy, 1994; Kochanska, Philibert, & Barry, 2009; Moutsiana et al., 2014; Orehek, Vazeou-Nieuwenhuis, Quick, & Weaverling, 2017; Sroufe, Carlson, Levy, & Egeland, 1999; Waters et al., 2010). Given that the current study failed to find an association between FAS-dysmorphism and emotion regulation after controlling for attachment security, it is of interest to examine the extent to which infant attachment security was associated with adolescent emotion regulation in this sample.

Attachment security in infancy was not significantly associated with the two adaptive emotion regulation subscales (i.e. *ARC Reflection* and *ERC Adaptive Emotion Regulation*). That is, adolescents classified as secure in infancy were no more likely to make use of

adaptive emotion regulation strategies in adolescence than were adolescents who had been classified as insecure in infancy.

Importantly, however, this study found that infant attachment security was a significant predictor for the three maladaptive emotion regulation subscales (i.e. *ARC Lack of Control, ARC Suppression of Affect,* and *ERC Lability/Negativity*). As such, infants classified as insecure in infancy were more likely to make use of maladaptive emotion regulation strategies in adolescence than were adolescents who had been classified as secure in infancy. These findings are consistent with studies among typically developing individuals that have found insecure attachment to be associated with the use of more maladaptive emotion regulation strategies. This association between insecure attachment and maladaptive emotion regulation has been demonstrated over multiple timespans in an individual's development - in the toddler/preschool years (e.g. NICHD Early Child Care Research, 2004), in later childhood (e.g. Borelli et al., 2010; Kerns et al., 2007), in adolescence (e.g. Hershenberg et al., 2011; Kobak et al., 2016; Scharf et al., 2004; Zimmermann, 1999), and in adulthood (e.g. Girme et al., 2020; Moutsiana et al., 2014).

One explanation for why attachment security significantly predicted the maladaptive emotion regulation outcomes but did not predict the adaptive emotion regulation outcomes in this study is proposed by Cracco and colleagues (2017). Specifically, they suggest that, irrespective of attachment classification in infancy, adolescents are likely to still make an attempt to implement an adaptive emotion regulation strategy. However, adolescents who are classified as insecurely attached in infancy often experience their emotions as overwhelming. As such, their attempts at implementing adaptive regulation strategies are unsuccessful, and they are, therefore, forced to compensate by using maladaptive emotion regulation strategies.

Another important point to note regarding attachment is that, as with IQ, insecure attachment has been consistently reported to be a consequence of PAE (Bergin &

McCollough, 2009; Hay et al., 2004; O'Connor et al., 1987; O'Connor et al., 2002). As such, it is difficult to distinguish the effects of PAE from the effects of insecure attachment in infancy on emotion regulation in adolescence. Thus, although the current study included both FAS-dysmorphism and attachment security as separate predictors in the regression analyses, and the multicollinearity statistics were found to be acceptable, the fact that attachment security and FAS-dysmorphism are not truly independent of one another made it more difficult to examine the FAS-dysmorphism effects over and above those of attachment security. Future studies may, therefore, also seek to consider attachment security as a mediator of the effect of FAS-dysmorphism on emotion regulation.

FAS-Dysmorphism Grouping

While not explicitly examined as a hypothesis in the current study, the use of FAS-dysmorphism to group participants in this study warrants some discussion. In this study, the HE category was split into those that exhibited even subtle facial dysmorphism consistent with FAS and those exhibited facial features consistent with controls. It is important to note that the level of PAE in these two HE groups (HE-dysmorphic and HE-nondysmorphic) did not differ. The HE-dysmorphic group was then clustered with the FAS and PFAS groups, forming the FAS-dysmorphic+ group, while the HE-nondysmorphic group was clustered with the controls, forming the FAS-dysmorphic- group.

In terms of cognitive performance according to FAS-dysmorphism, while the between group difference for adolescent IQ fell just short of significance in this study, it must be noted that alpha was adjusted down to $\alpha = .01$ for these confirmatory-type analyses. However, if I had used a conventional alpha of $\alpha = .05$, a significant between-group difference for adolescent IQ would exist. Nevertheless, in this sample the FAS-dysmorphism+ adolescents demonstrated an average IQ in the low borderline range while FAS-dysmorphism- adolescents demonstrated an average IQ that was in the low average range. These results are,

therefore, consistent with the findings reported by Suttie et al. (2013) on the larger Cape Town Longitudinal Cohort, from which this sample was drawn. Specifically, Suttie et al. (2013) found that HE children for which the signature analyses of 3D facial images detected even subtle dysmorphism (HE-dysmorphic) performed more poorly on neurocognitive measures, such as the WISC-IV Verbal Comprehension Index (Wechsler, 2004) as well as the CVLT-C (Delis et al., 1994) when compared to HE children for which the signature analyses of 3D facial images did not detect subtle dysmorphism (HE-nondysmorphic). Moreover, average performance of HE-dysmorphic children was comparable to those with FAS, while the performance of HE-nondysmorphic children was comparable to controls. The results of the current study therefore also suggest that the presence of even subtle facial dysmorphism (i.e. that which can only be detected by the analyses of 3D facial images) could be a potential indicator of cognitive performance.

The grouping of individuals as FAS-dysmorphic+ versus FAS-dysmorphic- in the current study was informed by the findings presented by Suttie et al. (2013) that even subtle facial dysmorphism could be a useful indicator of cognitive performance. This grouping was also informed by previous studies which have suggested that emotion regulation difficulties are commonly reported in individuals across the FASD spectrum, including individuals who do not present with the more noticeable facial dysmorphism associated with the more severe phenotypes (i.e. FAS and PFAS). While this way of grouping was important as it was informed by previous findings, as well as useful given the issue of statistical power due to the small sample size in this study, it did have its disadvantages. Specifically, by not examining the effects for each diagnostic category individually, it is possible that certain effects may have been concealed or obscured by grouping the categories together. However, in order to detect effects across individual diagnostic categories, a much larger sample would have been necessary (this issue is examined in greater depth below).

Clinical Significance

While the current study demonstrated that FAS-dysmorphism+ was not associated with higher rates of insecure and disorganised attachment, it did demonstrate the importance of infant attachment security in predicting emotion regulation in adolescents with FAS-dysmorphism+. While previous research has examined the emotion regulation difficulties often reported among individuals with FASD (Petrenko et al., 2017; Rasmussen et al., 2007; Schonfeld et al., 2006), none have considered the contribution of the early attachment relationship to these difficulties. This finding therefore also provides a novel contribution to the body of research on emotion regulation in FASD.

A number of intervention studies have sought to address the self-regulation and emotion regulation difficulties often reported among individuals with FASD (Kable et al., 2016; Petrenko et al., 2017; Wells et al., 2012). These interventions have been found to be effective in improving self- and emotion-regulation skills in children and adolescents with FASD. However, given the findings of the current study implicating the early attachment relationship in the emotion regulation difficulties evidenced by individuals with FASD, it may be that earlier intervention is advisable. For instance, these interventions could focus on promoting secure attachment by working to improve factors such as maternal sensitivity, and teaching caregivers techniques to soothe their infant (Carmichael-Olson et al., 2007). Thus, consideration of the contribution of attachment insecurity to emotion regulation difficulties could enable the design of interventions with more far-reaching impact on emotion regulation and the associated behavioural and psychiatric outcomes for individuals with FASD.

Strengths, Limitations, and Future Directions

The current study has several strengths which improve upon some of the limitations of previous studies investigating attachment or emotion regulation in the context of PAE/FASD. Firstly, the use of a prospectively recruited cohort in the current study

constitutes an important strength of the study. In this study, the level of PAE could be established based on maternal reports obtained throughout the pregnancy. Data collected antenatally have been found to be more accurate than retrospective reports of alcohol consumption during pregnancy (Jacobson, Chiodo, Sokol, & Jacobson, 2002). Furthermore, the timeline follow-back interview used here has been validated against data regarding the levels of fatty acid ethyl esters found in meconium samples in this population (Bearer et al., 2003).

A second strength of the current study is the use of measures directly assessing emotion regulation. With the exception of a few studies (Petrenko et al., 2017; Rasmussen et al., 2007; Schonfeld et al., 2006), most previous research investigating emotion regulation in children and adolescents with FASD has employed measures assessing internalizing and externalizing behaviours, as well as those assessing psychopathology (for example, Fagerlund, Autti-Rämö, Hoyme, Mattson, & Korkman, 2011; Mukherjee, Cook, Norgate, & Price, 2018; O'Connor & Paley, 2009; Streissguth et al., 1996). While it could be argued that the existence of difficulties in the regulation of emotion can be inferred in these cases, these measures do not assess emotion regulation processes directly. The current study, on the other hand, made use of two measures specifically designed to assess emotion regulation, thus allowing us to access the processes relating to poor emotion regulation more directly.

The third strength of this study is its longitudinal nature. Many studies examining the association between attachment and emotion regulation have been cross-sectional, with the assessment of attachment being conducted using the Adult Attachment Interview (AAI; George, Kaplan, & Main, 1996) or the AQS (Waters, 1995), for instance. Hay et al. (2004) assessed attachment patterns in the Cape Town Longitudinal Cohort during infancy using the SSP (Ainsworth, 1979), which is considered to be the 'gold standard' observational measure

of infant attachment to their caregiver. Drs. Sandra and Joseph Jacobson then followed the infants up through adolescence, at which point I assessed their emotion regulation abilities.

The current study also has several limitations which are necessary to mention. It is recommended that future studies that aim to interrogate the associations between FASD, attachment, and emotion regulation improve upon the current study's methodology in order to address these limitations. As Jacobson and Jacobson (2005) point out, there is a growing concern regarding the incidence of Type II errors in developmental teratology research. Given this, along with the small estimates of effect size found in the current study, it is advised that future studies seek to replicate the findings reported here.

The first major limitation of the current study is the fact that it comprised of a relatively small sample size (n = 77), which limited the statistical power to detect effects. The sample size was restricted by the number of infants who were administered the Strange Situation Procedure (n = 115). Sample size was further restricted as only 77 of the initial 115 infants were available for the adolescent follow-up assessment. With the current sample size, only moderate to large effects could be detected. As such, it is possible that the presence of small effects went undetected due to limited statistical power. Future research with a greater sample size is therefore recommended in an effort to replicate the current findings and perhaps even detect further effects not observed here due to a lack of statistical power.

It is worthwhile to note that even by collapsing the FASD diagnostic categories into the two groups (i.e. FAS-dysmorphic+ and FAS-dysmorphic-), the statistical power was still limited. Furthermore, sometimes the grouping of diagnostic categories may conceal effects seen for specific categories. However, in order to examine effects across diagnostic categories a much larger sample size would be needed; this can be difficult to accomplish with clinical samples. It would therefore be worthwhile for future research to conduct studies with multi-site cohorts in an effort to obtain a larger sample size. Given the growing

emphasis of open science and open data repositories, the sharing of data may also be a valuable avenue to consider in order to address the issue of sample size in clinical samples.

The second major limitation is the use of caregiver-report measures to evaluate emotion regulation. While caregivers' observations may provide valuable information regarding their adolescent's ability to regulate their emotions, it must be noted that the use of a caregiver-report measure of emotion regulation here may have resulted in an overestimation of the association between insecure attachment and emotion regulation. That is, reports by caregivers of adolescents classified as insecure in infancy may continue to be biased as a result of the unfavourable dynamic experienced within the early attachment relationship (Waters et al., 2010).

Another limitation of the use of caregiver-report measures, specifically to measure a construct like emotion regulation, concerns the extent to which caregivers are able to report on their adolescent's internal processes. As a result, a caregiver's report of their adolescent's ability to regulate their emotions may not be accurate. However, given that adolescents with FASD often demonstrate cognitive impairment, which may impact their ability to reflect upon the ways in which they regulate their emotions, it was decided that caregiver-reports would be most informative. Nevertheless, it cannot be ignored that caregiver-reports may be missing vital information. In order to address this, Taylor and Enns (2019) suggest collecting evidence of impairment across multiple different contexts. Teacher-reports may be particularly insightful in this regard. Given their extensive experience with adolescents of a similar age, teachers may be more equipped to evaluate an individual, given their superior knowledge of age-related norms and ability to draw comparisons (Antrop et al., 2002; Sims & Lonigan, 2013). Future research might, therefore, seek to employ multiple report measures across multiple environments in order to produce a more comprehensive account of the individual's emotion regulation abilities.

While the use of caregiver-, teacher-, and self-report measures to assess emotion regulation is helpful, the direct assessment of emotion regulation is also highly recommended (Adrian et al., 2011). For instance, some studies have examined emotion regulation by observing adolescents and their caregivers as they interact in order to resolve a given conflict (Beijersbergen et al., 2008; Yap et al., 2010) or solve a challenging puzzle (Eisenberg et al., 2005). Other studies have also sought to study emotion regulation by examining an individual's physiological responses to emotion induction. For instance, some studies have assessed emotion regulation via respiratory sinus arrhythmia (RSA; Butler, Wilhelm, & Gross, 2006; Vasilev, Crowell, Beauchaine, Mead, & Gatzke-Kopp, 2009) and vagal tone (Calkins, 1997; Movahed Abtahi & Kerns, 2017) during tasks designed to induce emotions. In order to measure emotion regulation as comprehensively as possible, it is suggested that future research employ both report, behavioural/observational, and physiological measures of emotion regulation. Furthermore, it is important to understand how emotion regulation processes may change across developmental periods as well as how the various measurements of emotion regulation may relate to one another (Adrian et al., 2011). Future studies, with longitudinal cohorts specifically, provide a unique opportunity to employ these multiple methods as we as examine changes over time.

The third limitation of the current study is the fact that it likely did not include all the potential confounding variables that may have influenced the effects of FASD on infant attachment and emotion regulation. A complex range of psychosocial risks exist for families living in LMICs, especially for those living in particularly low-income communities such as that from which the current sample is drawn (Howell, 2019). The Drakenstein Child Health Study (DCHS), a birth cohort study in the peri-urban Drakenstein area of Paarl, South Africa, has sought to examine the various psychosocial risk factors that influence child outcomes (Donald et al., 2019; Stein et al., 2015). These risk factors include low levels of maternal

education and employment, food insecurity (Barnett et al., 2019), high levels of unplanned pregnancies, as well as high levels of antenatal psychological distress, depression, and substance use. Many of the mothers in these studies have also been subjected to neglect or abuse in childhood and have experienced intimate partner violence in adulthood (Donald et al., 2019; Stein et al., 2015). Such psychosocial risk factors are also likely to be present in the sample examined in the current study and, therefore, may have impacted the results found by this study.

In terms of the significant associations between attachment security and the three maladaptive emotion regulation outcome variables found in this study specifically, there are likely other variables that influence the mechanism through which the attachment relationship supports the development of later emotion regulation skills that this study did not consider. For example, negative life events, such as a loss of a caregiver (Feigelman et al., 2017), physical or sexual abuse (Gruhn & Compas, 2020; Hébert et al., 2018), and exposure to violence and trauma (Herringa, 2017) have all been found to influence the development of emotion regulation. Given that such negative life events are prevalent in the population examined in this study (Howell, 2019), it would be important for future studies to determine whether the association between attachment security and emotion regulation holds up after considering the influence of these factors. It is important to note here, however, that given the small sample size of the current study, the statistical analyses used would not have been able to manage the level of complexity if all these additional factors were to be considered. Future studies with larger sample sizes should therefore aim to account for the impact of these additional variables on the relationship between attachment security and emotion regulation.

Another consideration is the issue of stability of attachment patterns over time. For instance, in studies of low-risk populations, attachment was found to be constant from infancy through to adulthood (Hamilton & Hamilton, 2000; Waters, Merrick, Treboux,

Crowell, & Albersheim, 2000). However, in high-risk populations where negative life events are often more frequent, individuals did not necessarily maintain the same attachment classification into adulthood (Weinfield et al., 2000). It is therefore recommended that future studies consider the addition of an adolescent/adult measure of attachment to determine whether attachment classification remains stable from infancy to adolescence. Once this has been established, the relationship of both infant and adolescent attachment classification to future competencies, such as emotion regulation, can be examined.

Conclusion

The current study demonstrated that children with FAS-dysmorphism (even subtle, i.e. only detectable via signature analyses of 3D facial images) are not more likely to demonstrate insecure or disorganised attachment in infancy than those without FAS-dysmorphism. However, this study is the first to establish the longitudinal nature of the relationship between attachment insecurity in infancy and the emotion regulation difficulties often reported in individuals with FASD. It also confirmed previous findings from studies among typically developing individuals that demonstrated the association between insecure attachment in infancy and maladaptive emotion regulation. Given the importance of emotion regulation for an individual's social functioning and psychological adjustment, the mechanism underlying the emotion regulation difficulties exhibited by individuals with FASD warrants more investigation. Furthermore, as attachment insecurity in infancy has been shown to contribute to emotion regulation difficulties, it is suggested that interventions targeting the early attachment relationship be developed. Such early intervention could result in a further-reaching impact on the emotion regulation abilities, as well as the associated behavioural and psychiatric outcomes, of individuals with FASD.

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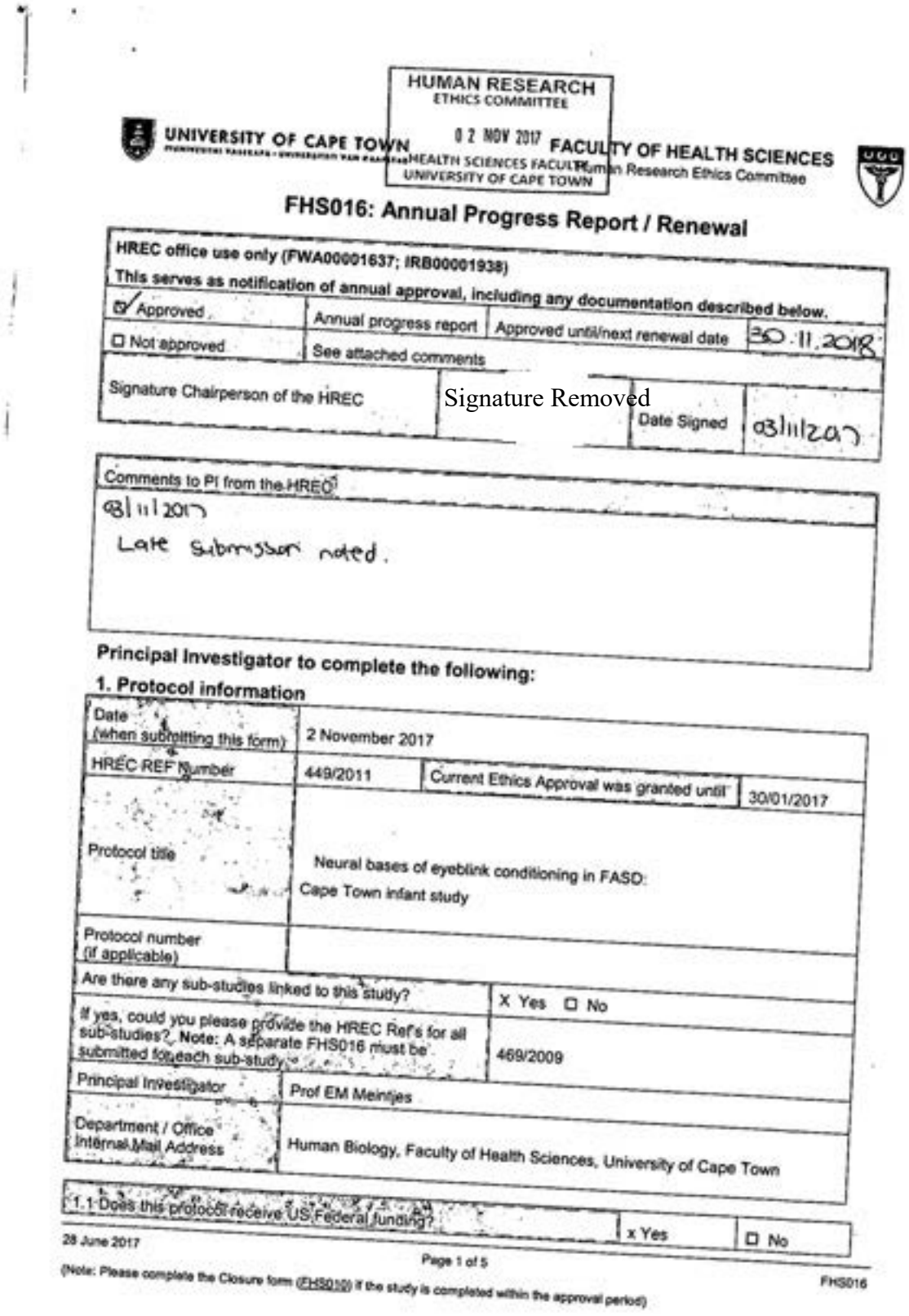
Appendix A

University of Cape Town Ethics Approval



HUMAN RESEARCH ETHICS COMMITTEE
02 NOV 2017
HEALTH SCIENCES FACULTY
UNIVERSITY OF CAPE TOWN

UNIVERSITY OF CAPE TOWN
FACULTY OF HEALTH SCIENCES
Human Research Ethics Committee

FHS016: Annual Progress Report / Renewal

HREC office use only (FWA00001637; IRB00001938) This serves as notification of annual approval, including any documentation described below.			
☑ Approved	Annual progress report	Approved until/next renewal date	30.11.2018
☐ Not approved	See attached comments		
Signature Chairperson of the HREC	Signature Removed	Date Signed	03/11/2017

Comments to PI from the HREC
03/11/2017 Late submission noted.

Principal Investigator to complete the following:

1. Protocol information

Date (when submitting this form)	2 November 2017		
HREC REF Number	449/2011	Current Ethics Approval was granted until	30/01/2017
Protocol title	Neural bases of eyeblink conditioning in FASD: Cape Town infant study		
Protocol number (if applicable)			
Are there any sub-studies linked to this study?	X Yes ☐ No		
If yes, could you please provide the HREC Ref's for all sub-studies? Note: A separate FHS016 must be submitted for each sub-study	469/2009		
Principal Investigator	Prof EM Meintjes		
Department / Office Internal Mail Address	Human Biology, Faculty of Health Sciences, University of Cape Town		
1.1 Does this protocol receive US Federal funding?	x Yes	☐ No	

(Note: Please complete the Closure form (FHS010) if the study is completed within the approval period)

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FACULTY OF HEALTH SCIENCES
Human Research Ethics Committee

1.2 If the study receives US Federal Funding, does the annual report require full committee approval? **Note:** Any annual approvals for **Full Committee** review MUST be submitted on the monthly HREC submission dates.	☐ Yes	x No

If yes in 1.2 please complete section 1.3 below for invoicing purposes	
1.3 Annual Approval for **full committee** review	- R 3420 (inclusive of vat)
For invoicing purposes, please provide:	
Sponsor's name	
Contact person	
Address	
Telephone number	
Email Address	

2. List of documentation for approval

3. Protocol status (tick ✓)

☐	Open to enrolment
✓	Closed to enrolment (tick ✓)
☐	Research-related activities are ongoing
☐	Research-related activities are complete, long-term follow-up only
✓	Research-related activities are complete, data analysis only
☐	Main study is complete but sub-study research-related activities are ongoing
☐	Study is closed → Please submit a Study Closure Form (FHS010)

4. Enrolment

Number of participants enrolled to date	151
Number of participants enrolled, since last HREC Progress report (continuing review)	0
Additional number of participants still required	0

5. Refusals

(Note: Please complete the Closure form (FHS010) if the study is completed within the approval period)

Total number of refusals (participants invited to join the study, but refused to take part)	0

6. Cumulative summary of participants

Total number of participants who provided consent	158
Number of participants determined to be ineligible (i.e. after screening)	0
Number of participants currently active on the study	0
Number of participants completed study (without events leading to withdrawal)	151
Number of participants withdrawn at participants' request (i.e. changed their mind)	0
Number of participants withdrawn by PI due to toxicity or adverse events	0
Number of participants withdrawn by PI for other reasons (e.g. pregnancy, poor compliance)	0
Number of participants lost to follow-up. Please comment below on reasons for loss of follow-up.	0
Number of participants no longer taking part for reasons not listed above. Please provide reasons below:	7
NONE SINCE LAST SUBMISSION. three stillbirths; one infant died due to gastroenteritis; one SIDS; 1 infant excluded from future analyses due to very low birthweight (all deaths unrelated to study procedures)	

7. Progress of study

Please provide a brief summary of the research to date including the overall progress and the progress since the last annual report as well as any relevant comments/issues you would like to report to the HREC:
All infants have completed neurobehavioral testing and neuroimaging. Study activities now are limited to data analysis.

8. Protocol violations and exceptions (tick ✓ all that apply)

x	No prior violations or exceptions have occurred since the original approval
☐	Prior violations or exceptions have been reported since the last review and have already been acknowledged or approved
☐	Unreported minor violations that have occurred since the last review, as well as significant deviations not yet reported, are attached for review

9. Amendments (tick ✓ all that apply)

☐	No prior amendments have been made since the original approval
x	Prior amendments have been reported since the last review and have already been approved
☐	New protocol changes/ amendments are requested as part of this continuing review (See note below)

Note: If new protocol changes are being requested in this review, please complete an amendment form (FHS006).
Specific changes in the amended protocol and consent/assent forms must be **bolded**, *italicised* or tracked and all changes must include a rationale.

10. Adverse events

10.1 Please provide below or attach a narrative summary of serious adverse events and/ or unanticipated problems since the last progress report. Please indicate changes made to the protocol and informed consent document(s) as a result (if not already reported to the HREC). Please comment on whether causality to any study procedure or intervention could be established.

None

10.2 Have participants received appropriate treatment/ follow-up/ referral when indicated (e.g. in the case of abnormal or incidental clinical findings, distress or anxiety)?

☐ Yes	☐ No	x Not applicable

If yes, please describe:

11. Summary of Monitoring and Audit Activities (tick ✓)

11.1 Was this study monitored or audited by an external agency (e.g. MCC, FDA)?

☐ Yes	x No	☐ Not applicable

11.2 Did a Data and Safety Monitoring Board publish a report?

☐ Yes	x No	☐ Not applicable

11.3 If yes, please identify the agency and attach a summary of the findings.

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Human Research Ethics Committee

Agency Name		Report attached	☐ Yes	☐ No	☐ Not applicable
		DSMB report attached	☐ Yes	☐ No	☐ Not applicable

11.4 Has there been any agency, institutional or other inquiry into non-compliance in this study, or any finding of non-compliance concerning a member of the research team?	
☐ Yes	x No
If yes, please explain:	

12. Level of risk (tick ✓)

12.1 In light of your experience of this research, please indicate whether the level of risk to participants has:	
☐	Increased
☐	Decreased
x	Shown no change
If there has been a change, please explain:	

12.2 Please provide a narrative summary of recent relevant literature that may have a bearing on the level of risk.

13. Statement of conflict of interest

Has there been any change in the conflict of interest status of this protocol since the original approval? (tick ✓)	
☐ Yes	x No
If yes, please explain and if necessary attach a revised conflict of interest statement (Section #7 in the New Protocol Application Form FHS013):	

14. Signature

My signature certifies that the above is complete and correct.			
Signature of PI	Signature Removed	Date	2 November 2017

(Note: Please complete the Closure form (FHS010) if the study is completed within the approval period)

UNIVERSITY OF CAPE TOWN

FACULTY OF HEALTH SCIENCES
Human Research Ethics Committee

Form FHS006: Protocol Amendment

HREC office use only (FWA00001637; IRB00001938)		
☐ Approved	☑ Type of review: Expedited	☐ Full committee
This serves as notification that all changes and documentation described below are approved.		
Signature Chairperson of the HREC	Signature Removed	Date 29/9/2017

Note: All major amendments must include a local PI Synopsis justifying the changes for the amendment.
Please note that incomplete amendment submissions will not be reviewed.

Comments from the HREC to the Principal Investigator:
Note: The approval of this protocol amendment does not grant annual approval. Please complete the FHS016 / FHS017 form for annual approval at least one month before study expiration.

Principal Investigator to complete the following:

1. Protocol information

Date (when submitting this form)	29 Aug 2017
HREC REF Number	471/2015
Protocol title	Neural Bases of cognitive and behavioural effects of FASD Addendum 2: Collection of urine and saliva samples
Protocol number (if applicable)	
Principal Investigator	Prof EM Meintjes
Department / Office Internal Mail Address	Division of Biomedical Engineering, Human Biology Room 5.14, Anatomy Building

1.1 Is this a major or a minor amendment? (see FHS006hlp) Major (tick box) Minor (tick box)	☐ Major	☒ Minor
1.2 Does this protocol receive US Federal funding?	☒ Yes	☐ No
1.3 If the amendment is a major amendment and receives US Federal Funding, does the amendment require full committee approval?	☐ Yes	☐ No

2. List of Proposed Amendments with Revised Version Numbers and Dates

Please itemise on the page below, all amendments with revised version numbers and dates, which need approval.
This page will be detached, signed and returned to the PI as notification of approval. Please add extra pages if necessary.

Enclosed please find the following consent and assent forms in both English and Afrikaans as both clean and tracked documents:

- Preschool Assessment Consent (English and Afrikaans, dated 08/03/2017)
- Adolescent Assessment Consent (English and Afrikaans, dated 08/03/2017)
- Adolescent Assent Form (English and Afrikaans, dated 08/03/2017)

We have also enclosed the 'Notice of full board amendment approval' from Wayne State University for IRB# 062914B3F

3. Protocol status (tick ✓)

■	Open to enrolment
□	No participants have been enrolled
□	Closed to enrolment (tick ✓)
□	Research-related activities are ongoing
□	Research-related activities are complete, long-term follow-up only
□	Research-related activities are complete, data analysis only

4. Proposed changes will affect: (tick ✓ all the categories that apply)

	Protocol
□	Study objectives, design (including investigator's brochure, clinical activities, study length)
□	Study instruments, questionnaires, interview schedules
□	Sample size
□	Recruitment methods
□	Eligibility criteria (inclusion and exclusion criteria)
□	Drug/device (composition, amount, schedule, route of administration, combination with other drugs/devices, safety information)
■	Data collection/ analysis
□	Principal Investigator. (Please attach revised conflict of interest and PI declaration statements. Refer: sections 7 and 8.4 in the New Protocol Application Form FHS013)
■	Consent form and information sheet
□	Recruitment materials (e.g. advertisements)
□	Administrative (e.g. change in sponsor's name, change in contact information)

☐	Other. Please specify:

4.1 In your opinion, will there be any **increase** in risk, discomfort or inconvenience to participants?	☐ Yes	☒ No
If yes, please provide a detailed justification/explanation:		

4.2 What follow-up action do you propose for participants who are already enrolled in the study?

☐	Inform current participants as soon as possible
☒	Re-consent current participants with revised consent/assent forms (append)
☐	No action required
☐	Other. Please describe:

5. Detailed description of the change(s)

Please attach, for each amendment, a summary of all changes which clearly indicates:

i. Old wording (e.g. ~~strikethrough~~ text, CHANGED FROM and CHANGED TO)

ii. New wording (e.g. *italicized*, **bold**, tracked)

iii. Detailed rationale/ justification/ explanation for each change

6. Signature

My signature certifies that I will maintain the anonymity and/ or confidentiality of information collected in this research. If at any time I want to share or re-use the information for purposes other than those disclosed in the original approval, I will seek further approval from the HREC.

Signature of PI	Signature Removed	Date	29 Aug 2017

UNIVERSITY OF CAPE TOWN
Faculty of Health Sciences
Human Research Ethics Committee

Room E52-24 Old Main Building
Groote Schuur Hospital
Observatory 7925
Telephone [021] 406 6338 • **Facsimile** [021] 406 6411
Email: nosi.tsama@uct.ac.za
Website: www.health.uct.ac.za/fhs/research/humanethics/forms

24 August 2015

HREC REF: 471/2015

Prof E Meintjes
Biomedical Engineering
Human Biology
Anatomy Building

Dear Prof Meintjes

PROJECT TITLE: NEURAL BASES OF COGNITIVE AND BEHAVIOURAL EFFECTS OF FASD

Thank you for your response to the Faculty of Health Sciences Human Research Ethics Committee dated 21 August 2015.

It is a pleasure to inform you that the HREC has **formally approved** the above-mentioned study.

Approval is granted for one year until the 30th August 2016.

Please submit a progress form, using the standardised Annual Report Form if the study continues beyond the approval period. Please submit a Standard Closure form if the study is completed within the approval period.
(Forms can be found on our website: www.health.uct.ac.za/fhs/research/humanethics/forms)

Please quote the HREC REF in all your correspondence.

Please note that the ongoing ethical conduct of the study remains the responsibility of the principal investigator.

Yours sincerely

Signature Removed

PROFESSOR M BLOCKMAN
CHAIRPERSON, FHS HUMAN RESEARCH ETHICS COMMITTEE
Federal Wide Assurance Number: FWA00001637.
Institutional Review Board (IRB) number: IRB00001938
This serves to confirm that the University of Cape Town Human Research Ethics Committee complies to the Ethics Standards for Clinical Research with a new drug in patients, based on the Medical Research Council (MRC-SA), Food and Drug Administration (FDA-USA), International Convention on Harmonisation Good Clinical Practice (ICH GCP), South African Good Clinical Practice Guidelines (DoH 2006), based on the Association of the British Pharmaceutical Industry Guidelines (ABPI),and Declaration of Helsinki guidelines.

The Human Research Ethics Committee granting this approval is in compliance with the ICH Harmonised Tripartite Guidelines E6: Note for Guidance on Good Clinical Practice (CPMP/ICH/135/95) and FDA Code Federal Regulation Part 50, 56 and 312.

HREC 471/2015

Wayne State University Ethics Approval

WAYNE STATE UNIVERSITY

IRB Administration Office
87 East Canfield, Second Floor
Detroit, Michigan 48201
Phone: (313) 577-1628
FAX: (313) 993-7122
http://irb.wayne.edu

NOTICE OF FULL BOARD CONTINUATION APPROVAL

To: Sandra Jacobson
Psychiatry
Department of Psychiatry and B

From: Dr. Deborah Ellis or designee D. Ellis, PhD/em
Chairperson, Behavioral Institutional Review Board (B3)

Date: February 15, 2018

RE:

IRB #:	062914B3F
Protocol Title:	Neural Bases of Cognitive and Behavioral Effects of FASD
Funding Source:	Sponsor: National Institute on Alcohol Abuse and Alcoholism Sponsor: National Institutes of Health Award: 019328-001
Protocol #:	1406013123
Expiration Date:	February 14, 2019
Risk Level / Category:	45 CFR 46.404 - Research not involving greater than minimal riskResearch not involving greater than minimal risk

Continuation for the above-referenced protocol and items listed below (if applicable) were **APPROVED** following Full Board review by the Wayne State University Institutional Review Board (B3) for the period of 02/15/2018 through 02/14/2019. This approval does not replace any departmental or other approvals that may be required.

- Actively accruing participants.
- Behavioral Research Screening Consents (dated 8/19/2015) (1 Screening) - English and Afrikaans versions (2)
- Behavioral Research Informed Consents (dated 3/1/2016) (five year assessment) - English and Afrikaans versions (2)
- Behavioral Research Informed Consents (dated 1/31/2018) (Adolescent Assessment) - English and Afrikaans versions (2)
- Adolescent Assent Forms - Ages 13-17 Years (dated 1/31/2018) (2 Adolescent) - English and Afrikaans versions (2)
- Parental Permission/Research Informed Consent - 3D Photo & Diagnostic Clinic Consent (young adult) - English and Afrikaans versions (2) (dated 7/19/2017)
- Parental Permission/Research Informed Consent - 3D Photo & Diagnostic Clinic Consent (child /adolescent) - English and Afrikaans versions (dated 7/19/2017)
- Parental Permission/Research Informed Consent - 3D Photo & Diagnostic Clinic Consent (child) - English and Afrikaans versions (2) (dated 7/19/2017)
- Adolescent Assent Form - Ages 13-17 - English and Afrikaans versions (2) (revision dated 7/19/2017)
- Cape Town 2016 Dysmorphology Assent Script - English and Afrikaans versions (2)

* Federal regulations require that all research be reviewed at least annually. You may receive a *"Continuation Renewal Reminder"* approximately two months prior to the expiration date; however, it is the Principal Investigator's responsibility to obtain review and continued approval ***before*** the expiration date. Data collected during a period of lapsed approval is unapproved research and can never be reported or published as research data.

* All changes or amendments to the above-referenced protocol require review and approval by the IRB **BEFORE** implementation.

* Adverse Reactions/Unexpected Events (AR/UE) must be submitted on the appropriate form within the timeframe specified in the IRB Administration Office Policy (http://www.irb.wayne.edu/policies-human-research.php).

NOTE:

WAYNE STATE UNIVERSITY

IRB Administration Office
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NOTICE OF EXPEDITED AMENDMENT APPROVAL

To: Sandra Jacobson
Psychiatry
Department of Psychiatry and B

From: Dr. Deborah Ellis or designee M. Tarcan, MD/sm
Chairperson, Behavioral Institutional Review Board (B3)

Date: February 01, 2018

RE:

IRB #:	062914B3F
Protocol Title:	Neural Bases of Cognitive and Behavioral Effects of FASD
Funding Source:	Sponsor: National Institute on Alcohol Abuse and Alcoholism Sponsor: National Institutes of Health Award: 019328-001
Protocol #:	1406013123
Expiration Date:	March 15, 2018
Risk Level / Category:	45 CFR 46.404 - Research not involving greater than minimal riskResearch not involving greater than minimal risk

The above-referenced protocol amendment, as itemized below, was reviewed by the Chairperson/designee of the Wayne State University Institutional Review Board (B3) and is APPROVED effective immediately.

- Parental Consent Form and Assent Form (revisions dated 1/31/2018) - Parental Consent Form and Assent Form modified to reflect an added sentence to inform the participants that s/he will be voice recorded while s/he provides a narrative to the pictures. The detail was inadvertently not included but is part of the procedure previously approved, obtaining a voice recording of the participants narrative is necessary for the purpose of transcribing and coding of the responses. (I) Adolescent Assent Form - Ages 13-17 years (English &Afrikaans) (II) Behavioral Research Informed Consent (English &Afrikaans)

Notify the IRB of any changes to the funding status of the above-referenced protocol.

WAYNE STATE UNIVERSITY

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FAX: (313) 993-7122
http://irb.wayne.edu

NOTICE OF FULL BOARD AMENDMENT APPROVAL

To: Sandra Jacobson
Psychiatry
Department of Psychiatry and B

For:
From: Dr. Deborah Ellis or designee D. Ellis, PhD/sm
Chairperson, Behavioral Institutional Review Board (B3)

Date: April 21, 2016

RE:

IRB #:	062914B3F
Protocol Title:	Neural Bases of Cognitive and Behavioral Effects of FASD in Adolescence
Funding Source:	Sponsor: NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM Sponsor: NATIONAL INSTITUTES OF HEALTH
Protocol #:	1406013123
Expiration Date:	April 20, 2017
Risk Level / Category:	45 CFR 46.404 - Research not involving greater than minimal riskResearch not involving greater than minimal risk

The above-referenced protocol amendment, as itemized below, was reviewed by the Wayne State University Institutional Review Board (B3) and is **APPROVED** effective immediately.

- Protocol - Data Collection Methods modified to reflect updated language, the addition of a saliva collection that was previously approved but inadvertently left out, and the addition of the collection of a urine sample.
- Consent Forms (revision dated 3/1/2016) - Consent Forms modified to reflect adding missing language regarding a saliva sample, the addition of a urine test to assess the use of drugs that could affect performance on the behavioral assessments, and a pregnancy test to ensure that none of the women are pregnant during the MRI scan. (I) Behavioral Research Informed Consents (five year assessment) - English & Afrikaans versions (2). (II) Behavioral Research Informed Consents (2 Adolescent Assessment) - English & Afrikaans versions (2). (III) Adolescent Assent Forms - Ages 13-17 years (2 Adolescent) - English & Afrikaans versions (2).
- Receipt of New Protocol Summary Form Appendix H:The Use of Biological Specimens.

Notify the IRB of any changes to the funding status of the above-referenced protocol.

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NOTICE OF FULL BOARD CONTINUATION APPROVAL

To: Sandra Jacobson
Psychiatry
Department of Psychiatry and B

From: Dr. Deborah Ellis or designee D. Ellis/BB.
Chairperson, Behavioral Institutional Review Board (B3)

Date: May 21, 2015

RE:

IRB #:	062914B3F
Protocol Title:	Neural Bases of Cognitive and Behavioral Effects of FASD in Adolescence
Funding Source:	Sponsor: NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM Sponsor: NATIONAL INSTITUTES OF HEALTH
Protocol #:	1406013123
Expiration Date:	May 20, 2016
Risk Level / Category:	45 CFR 46.404 - Research not involving greater than minimal riskResearch not involving greater than minimal risk

Continuation for the above-referenced protocol and items listed below (if applicable) were **APPROVED** following Full Board review by the Wayne State University Institutional Review Board (B3) for the period of 05/21/2015 through 05/20/2016. This approval does not replace any departmental or other approvals that may be required.

- Actively accruing participants.
- Behavioral Research Screening Consent (dated 9/11/2014, 1 Screening) - English and Afrikaans versions
- Behavioral Research Informed Consent [dated 2/24/2015, Protocol Version #:3 (five year assessment)] - English and Afrikaans versions
- Behavioral Research Informed Consent (dated 2/4/2015, Protocol Version #: 2) - English and Afrikaans versions
- Adolescent Assent Form for Ages 13-17 Years (dated 1/5/2015, Protocol Version #: 2 Adolescent) - English and Afrikaans versions

- Federal regulations require that all research be reviewed at least annually. You may receive a *"Continuation Renewal Reminder"* approximately two months prior to the expiration date; however, it is the Principal Investigator's responsibility to obtain review and continued approval **before** the expiration date. Data collected during a period of lapsed approval is unapproved research and can *never* be reported or published as research data.
- All changes or amendments to the above-referenced protocol require review and approval by the IRB **BEFORE** implementation.
- Adverse Reactions/Unexpected Events (AR/UE) must be submitted on the appropriate form within the timeframe specified in the IRB Administration Office Policy (http://www.irb.wayne.edu/policies-human-research.php).

NOTE:

1. Upon notification of an impending regulatory site visit, hold notification, and/or external audit the IRB Administration Office must be contacted immediately.
2. Forms should be downloaded from the IRB website at **each** use.

Appendix B

Informed Consent - English

Title of Study: Neural Bases of Cognitive and Behavioral Effects of FASD (Adolescent Assessment)

We are pleased to invite you and your child _____ to continue to take part in our research study that many of you have been in since you were pregnant and your child was born. Please read this form and ask us any questions you have before agreeing to be in the study. The people conducting this study are doctors and scientists: Christopher Molteno, M.D., and Ernesta Meintjes, Ph.D., from the Faculty of Health Sciences of the University of Cape Town; Nadine Gaab, Ph.D., from Boston Children's Hospital/Harvard University, United States; Andrea Berger, Ph.D., from Ben Gurion University in the Negev, Israel; R. Colin Carter, M.D., Columbia University College of Physicians and Surgeons; and Sandra W. Jacobson, Ph.D., and Joseph L. Jacobson, Ph.D., from Wayne State University School of Medicine in the United States. The study is being paid for by the National Institute on Alcohol Abuse and Alcoholism in the United States. The study will be conducted according to the ethical guidelines and principles of the international Declaration of Helsinki, as well as South African research ethics guidelines, as documented by the Department of Health in "Ethics in health research: Principles, processes and structures", Second edition, 2015.

Purpose

You and your child are being asked to be in a research study so that we can learn whether some aspects of a child's learning and behavior are affected by the mother's diet, alcohol, smoking or drug use during pregnancy and how these exposures affect the developing brain. Some of the children in the study will have fetal alcohol syndrome (FAS), some will have attention deficit hyperactivity disorder (ADHD), and others will have neither. We want to see if arithmetic and reading and brain activity while doing arithmetic and reading are similar or different for those with and without ADHD who were or were not exposed to alcohol during pregnancy. Other purposes of the study are to see whether your child's abilities when s/he was a baby and at 5 or 10 years old predict how he or she is doing now as an adolescent. The study is being conducted at the University of Cape Town. We expect that there will be about 220 study participants seen in our laboratory. To help decide whether or not to agree to take part with your child in this study, a project staff member has talked with you about the risks and benefits of the study. This consent form summarizes the information given to you by the project staff member during this informed consent process. **Please read this form and ask any questions you may have before agreeing to be in the study.**

In this research study, we will use new methods for studying the brain called event related potentials (ERPs) to better understand how diet, alcohol, smoking, or drug use during pregnancy can affect a child's development, especially his or her reading, arithmetic, attention, memory, behavior, and growth. ERP is a new noninvasive way of studying the electrical activity of the brain. We will also use methods for studying the brain called magnetic resonance imaging (MRI), which were used in your earlier visits to the Cape Universities Brain Imaging Center (CUBIC) at Tygerberg Hospital. During the MRI assessment, your child lies as still as possible in a scanner that uses magnets to take pictures

of the brain. We will take these pictures in the scanner while your child watches a video and then does some reading, memory, arithmetic, and other cognitive and behavioral tasks.

Study Procedures

If you agree to have your child take part in this study, we will bring you and your child to our laboratory at the University of Cape Town for two visits that will each take about 3-4 hours for a total of about 6-8 hours. About 3-4 months later you and your child will come back to our laboratory for two more visits that will also each take about 3-4 hours for a total of about 6-8 hours.

- We will begin by introducing you and your child to the research staff and will give you both breakfast each day before the assessment begins.
- During the visits, your child will do tasks involving attention, learning, memory, social/emotional function, arithmetic, and reading. Several of these are computer tasks.
- Then we will study how his/her brain reacts when working on arithmetic and reading problems and related cognitive/behavioral tasks using ERP. To do so, we will examine brain activity while s/he is looking at a videotape.
- Before starting the ERP tasks, we will soak a special net with small sensors in warm water before putting it on his/her head. We then will place the net on your child's head to record the brain activity while s/he looks at the videotape—this takes less than 5 min to do.
- Each ERP task takes 15-20 min. Your child will complete two ERP tasks on each day of the first two visits, one in the early morning and the other later during the visit. Your child will be videotaped performing these tasks. The videotapes will be viewed only by research team members and will be destroyed after 10 years.
- In another task, we will show your child some pictures (drawings) and ask him/her to tell us a story about what is going on in the picture, what led up to the scene, what are the people thinking or feeling, and what might happen next. Your child's voice will be recorded while he/she tells these stories.
- We will also test your child's hearing and vision and handedness.
- We will weigh and measure your child and take a photograph to look for facial features that often relate to alcohol exposure during pregnancy.
- We will ask your child about his/her diet, alcohol and drug use, and behavior.
- You will be present in a room nearby during all of your child's assessments and will be present with your child during the physical growth examination.
- During this visit, we will ask you some questions about your child's attention and behavior, his/her school and health history, and any medications that s/he is taking. We will also ask you to complete a form asking your child's teacher to fill out the DBD, which gives us information about your child's behavior at school.
- We will ask you to update us about your family's employment, your current drinking, smoking, and drug use, attention problems you may have had as a child (and stressful feelings that you experience, including sadness, anxiety, and distress).
- At one visit our research nurse may ask you to provide about 10 ml (about 2 teaspoons) of saliva (spit) from you and your child to study genes (characteristics that you inherit from your parents) and changes in genes caused by diet and alcohol exposure (epigenetic differences) that have been found to be related to differences in alcohol use, nutrition, depression, attachment, or child attention/behavior and development. These samples will be stored and used for future genetic and/or epigenetic analyses.

- We would also like your child to give us a small urine sample (about 3 table spoons), which may be used to look at chemicals that tell us about your child's diet and how your child's body uses what your eats. We would also look at chemicals in the urine that result from use of drugs, such as marijuana (dagga) and methamphetamine (tik). We will also perform a urine pregnancy test for the girls. To maintain confidentiality, we cannot share results of these tests with the parents, but we will encourage your child to share this information with you.
- You and your child always have the option of not answering some of the questions and will still be able to remain in the study.
- Your and your child's name will not appear on any of our interviews or testing material. Instead you will be identified by a code number (see Confidentiality below).
- We will use the findings from this study for research purposes only.

On a different day, our research nurse and driver will drive you and your child to the University of Cape Town campus of CUBIC, which is located at Groote Schuur Hospital, for one visit that should take about 3 hours in total.

- We will begin by showing your child what will happen in the scanner by practicing in a mock scanner in which s/he will hear the kinds of sounds and practice with a button press like the one used during the scan.
- During the scan, your child will lie on a padded plastic bed that slides into the scanner. We will ask your child to lie as still as possible while the scans are being taken. Taking these pictures of the brain does not hurt and is used every day by many people in the hospital. During some of the time in the scanner, your child will watch videos and during some of the time s/he will do reading or other related cognitive or behavioral tasks.
- There will be two sessions in the scanner on the same day; each will last no longer than 45-60 minutes for a total of about 3-4 hours (including meals).
- Children with the following may not have an MRI but will take part in the rest of the visits: implanted medical devices, such as aneurysm clips in the brain, heart pacemakers, and cochlear (inner ear) implants; lead-based tattoos; or pieces of metal close to or in an important organ (such as, the eye); claustrophobia or fear of being in a small space.

During each of the visits to our laboratory at the University of Cape Town and to CUBIC, you and your child will be given breakfast, a snack, and lunch. In all, the five visits (two to our laboratory now and two in 3-4 months and one to CUBIC) will take a total of about 15-20 hours.

Benefits

As a participant in this research study, there may be no direct benefits for you; however, information from this study may help other people now or in the future. The possible benefits to you for taking part in this research study are that we will give you information about your child's development at this age. If a serious problem is found, we will tell you and refer your child to a doctor and/or someone who can help, if you would like us to do so. If your child is suffering from any major illness, we will send you to Red Cross Children's Hospital or Groote Schuur Hospital. No information about your child will be given to any doctors, hospitals, or schools unless you ask us and allow us to do so in writing.

Risks
There are no known risks at this time to participation in this study. There are no known harmful effects of ERP or MRI used in this study. During the MRI neuroimaging assessments, certain metal objects, such as, watches, credit cards, hairpins, and writing pens, may be damaged by the MRI scanner or pulled away from the body by the magnet. For these reasons, we will ask your child to remove these before going into the scanner. When the scanner makes the pictures, the bed may shake, and your child will hear loud banging noises. S/he will be given earplugs or headphones to protect the ears. Also, some people feel nervous in a small closed space, such as when they are in the scanner. Your child will be able to see out of the scanner at all times, and we will not start until s/he tells us that s/he is comfortable. You may feel sad or upset when asked to talk about sadness, anxiety, or stressful events in your life. A referral can be made for help with these feelings, if you would like. No names are recorded on the interview or assessment forms. All names are kept in locked files in our locked offices at UCT and WSU. There is a small risk that your identity may be revealed since we need to keep this information for purposes of contacting you to take part in this research study and to transport you to our laboratory. There is little risk that anything you tell us will be told to people outside the study, and we will do everything we can to keep this information secret, as described below, except that the following information must be reported to the appropriate authorities, if at any time during the study there is concern that:

- child abuse or elder abuse has possibly occurred

There may also be risks involved from taking part in this study that are not known to researchers at this time.

Study Costs
Participation in this study will be of no cost to you or your child, and you and your child will be transported to the laboratory at University of Cape Town by our study driver.

Compensation
For taking part in this research study, we will pay you R150 for each visit for a total of R750 and give you a photo of your child, and we will give your child a small gift. You and your child will also be given breakfast, a snack, and lunch at each visit.

What if Something Goes Wrong?
The University of Cape Town undertakes that in the event that you or your child suffer any significant deterioration in health or well-being, or from any unexpected sensitivity or toxicity, that is caused by your participation in the study, it will provide immediate medical care. The University of Cape Town has appropriate insurance cover to provide prompt payment of compensation for any research-related injury according to the guidelines outlined by the Association of the British Pharmaceutical Industry, ABPI 1991. Broadly-speaking, the ABPI guidelines recommend that the insured company (University of Cape Town), without legal commitment, should compensate you without you having to prove that University of Cape Town is at fault. An injury is considered research-related if, and to the extent that, it is caused by study activities. You must notify the study doctor immediately of any side effects and/or injuries during the research study, whether they are research-related or other related complications. If you think that you or your child has suffered a research-related injury, please contact Dr. Christopher Molteno right away at 021-406-6212 (Department of Human Biology, Anatomy Building Suite 7.02, Anzio Road, Observatory 7925, Cape Town).

The University of Cape Town reserves the right not to provide compensation if, and to the extent that, your or your child's injury came about because you chose not to follow the instructions that you were given while you were taking part in the study. Your right in law to claim compensation for injury where you prove negligence is not affected. Copies of these guidelines are available on request. No reimbursement, compensation, or free medical care is offered by Wayne State University.

Confidentiality
All information collected about you and your child during the course of this study will be kept secret to the extent permitted by law. You will be identified in the research records by a code number. Information that identifies you personally will not be released without your written permission. However, the study sponsor, the Institutional Review Board (IRB) at Wayne State University and the University of Cape Town Research Ethics Committee, or government agencies with appropriate regulatory oversight may review your records. The list linking names and code numbers will be stored in locked file cabinets in the research laboratory. Only project staff members who need to contact you by telephone or in person will be allowed to look in these files.

Information from this study may be presented in scientific meetings or journals or for teaching purposes, but your and your child's names will be kept secret. We have added a signature line to the Consent asking for your permission to sometimes use a photo or video for research or educational purposes. We will only use them if we have your written consent. Permission to use the photos or videos is not required for you to participate in the study.

Voluntary Participation/Withdrawal
Taking part in this study is voluntary. You have the right to choose not to take part in this study. If you decide to have your child take part in the study, you can later change your mind and quit the study at any time. Also, you and your child are free not to answer any questions or to stop any task before it is finished. Your decisions will not change any present or future relationship with the University of Cape Town or Wayne State University or other services you are entitled to receive.

The researcher or the sponsor may also stop your child's taking part in this study without your consent. The PI will make the decision and let you know if it is not possible for you to continue. The decision that is made is to protect your health and safety or because you did not follow the instructions to take part in the study

Questions
If you have any questions about this study now or in the future, you may contact Prof. Christopher Molteno or one of his research team members at 021-406-6212. If you have questions or concerns about your rights as a research participant, you can contact the Chair of the University of Cape Town Research Ethics Committee (021-406-6338).

Consent to Participate in a Research Study
To voluntarily agree to have you and your child take part in this study, you must sign on the line below. If you choose to take part in this study with your child, you or your child may withdraw at any time. You are not giving up any of your or your child's legal rights by signing this form. Your signature shows that you have read, or had read to you, this whole

consent form, including the risks and benefits, and that we have answered all of your questions. We will give you a copy of this consent form to take home.

_____ _____
Signature of Parent or Legally Authorized Guardian Date

_____ _____
Printed Name of Parent or Authorized Guardian Time

_____ _____
Signature of Witness* Date

_____ _____
Printed Name of Witness* Time

_____ _____
Signature of Person Obtaining Consent Date

_____ _____
Printed Name of Person Obtaining Consent Time

I hereby give my permission to use and retain a photo or video of my child and/or me for research or educational purposes:

Yes____ No_____

_____ _____
Signature of Parent or Legally Authorized Guardian Date

_____ _____
Printed Name of Parent or Authorized Guardian Time

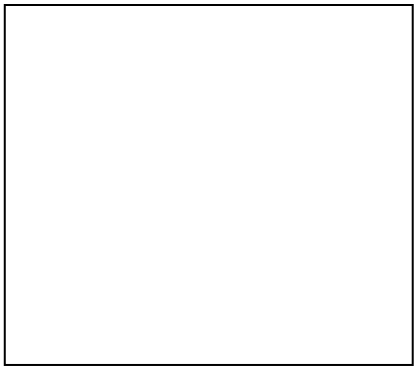
May we contact you again to be a part of future studies?

_____ Yes
_____ No

_____ _____
Signature of Parent or Legally Authorized Guardian Date

_____ _____
Printed Name of Parent or Authorized Guardian Time

***** Use when parent has had consent form read to them (i.e., illiterate, legally blind, translated into foreign language).



Informed Assent – English

Adolescent Assent Form

(Ages 13-17 years)

Title: Neural Bases of Cognitive and Behavioral Effects of FASD

Study Investigators: Sandra W. Jacobson, Nadine Gaab, Andrea Berger, Ernesta M. Meintjes,
Joseph L. Jacobson, Christopher D. Molteno, and R. Colin Carter

Why am I here?

This is a research study. Only people who choose to take part are included in research studies. You are being asked to take part in this study because many of you are part of a large group of children who have been taking part in this study since you were born and in visits as an infant and at 5 and 10 years of age. Some of you are new to this study. We are inviting all of you to take part in the next phase of this study. Please take time to make your decision. Talk to your family about it and be sure to ask questions about anything you don't understand.

Why are they doing this study?

- This study is being done to study how adolescents solve arithmetic problems and to study your reading. We want to see how your brain responds when you do arithmetic problems and read.
- In this research study, we will use new methods for studying the brain called event related potentials (ERPs) to better understand how diet, alcohol, smoking, or drug use during pregnancy can affect development, including reading, arithmetic, attention, memory, and behavior. ERP is a new noninvasive way of studying the electrical activity of the brain.
- We also want to see if arithmetic and reading and brain activity while doing arithmetic and reading are similar or different for those with and without attention deficit/hyperactivity disorder (ADHD).
- We will also use methods for studying your brain called magnetic resonance imaging (MRI), which many of you took part in during earlier visits to the Cape Universities Brain Imaging Center (CUBIC). During the MRI assessment, you lie as still as possible in the scanner that uses magnets to take pictures of your brain. We will take these pictures in the scanner while you watch a video and then do some reading, memory, arithmetic, and other learning and behavioral tasks.

What will happen to me?

Here at University of Cape Town, we will study how you do on arithmetic and reading problems. Then we will study how your brain reacts when you do arithmetic and reading problems using ERP. To do so, we will examine brain activity while you are looking at a videotape. Before starting the task, we will soak a special net with small sensors in warm water before putting it on your head. We then will place the net on your head to record your brain activity while you look at the videotape. You will be videotaped while doing these tasks. In another task we will ask you to tell a story about what is happening in some pictures that we show you, what are the people thinking or feeling, and what might happen next. Your voice will be recorded while you tell these stories. You will also complete some other tasks involving learning, memory, and attention. Some of these will be paper and pencil tasks, and

others will be computer tasks. We will also weigh you, measure how tall you are, take a photo of you, and check how well you can hear and see. We will ask you about your current alcohol, smoking, and drug use, and ask your current classroom teacher about your school work and behavior.

You will spend this morning here and will come back to University of Cape Town another day now and then again for 2 days in a few months to do more of the brain activity and other tasks.

On a different day, our research nurse and driver will drive you and your parent to the University of Cape Town campus of CUBIC, which is located at Groote Schuur Hospital, for one visit that should take about 3 hours in total. During this visit we will show you what will happen in the scanner by practicing in a mock scanner in which you will hear the kinds of sounds and practice with a button press like the one used during the scan. During the scan, you will lie on a padded plastic bed that slides into the scanner. We will ask you to lie as still as possible while the scans are being taken. Taking these pictures of the brain does not hurt and is used every day by many people in the hospital. During some of the time in the scanner, you will watch videos and during some of the time you will do reading or other related cognitive or behavioral tasks.

We will also ask you to give us a small urine sample (about 3 table spoons), which may be used to look at chemicals that tell us about your diet; how your body uses what you eat; that result from use of drugs, such as marijuana (dagga) and methamphetamine (tik). We will also perform a urine pregnancy test for the girls before you take part in the scanner visit. To maintain confidentiality, we will not share results of these tests with parents but will encourage you to do so.

We may also ask you for a small saliva (spit) sample (about 2 teaspoons) to study genes (characteristics that you inherit from your parents) and changes in genes caused by diet and alcohol exposure that have been found to be related to differences in alcohol use, nutrition, depression, attachment, or child attention/behavior and development. These samples will be stored and used for future genetic and/or epigenetic analyses.

How long will I be in the study?

You will be in the study for this phase on 2 days now, for about 3-4 hours each time, at our laboratory at the University of Cape Town (including breakfast, a snack, and lunch) and again for 2 days, again for about 3-4 hours each time, in a few months. There will also be two sessions in the CUBIC scanner on one day; each session will last no longer than 45-60 minutes for a total of about 3-4 hours (including breakfast, a snack, and lunch).

Will the study help me?

You will not benefit from being in this study; however information from this study may help other people in the future better understand how the brain performs different tasks and whether diet, alcohol, smoking, or drug exposure during pregnancy affects how the brain performs.

Will anything bad happen to me?

There is a chance that some of the questions might make you sad or upset. If you feel that way, we can get someone to talk with you. We do not expect there to be any other risks from any of the tasks we will do with you in our laboratory at University of Cape Town or in the scanner.

Do my parents or guardians know about this?
This study information has been given to your parent/guardian and s/he said that you could take part in the study. You can talk this over with him/her before you decide.

Research Related Injuries
In the event that this research related activity results in an injury, treatment will be made available including first aid, emergency treatment, and follow-up care, as needed. Care for such will be billed in the ordinary manner to you or your insurance company/South African public assistance. No reimbursement, compensation, or free medical care is offered by Wayne State University or the University of Cape Town. If you think that you have suffered a research related injury, please contact the Cape Town Principal Investigator Prof. Christopher Molteno right away at 021-406-6212.

What about confidentiality?
Every reasonable effort will be made to keep your records (medical or other) and/or your information confidential, however we do have to let some people look at your study records.

We will keep your records private unless we are required by law to share any information. The law says we have to tell someone if you might hurt yourself or someone else. The study doctor can use the study results as long as you cannot be identified.

The following information must be released/reported to the appropriate authorities if at any time during the study there is concern that:

- child abuse or elder abuse has possibly occurred

Compensation
While taking part in this phase of the research study, we will give you a small gift and a photo taken of you. We will provide breakfast, a snack, and lunch each time you come to our laboratory at University of Cape Town.

What if I have any questions?
For questions about the study please call Dr. Christopher Molteno at 021-406-6212. If you have questions or concerns about your rights as a research participant, you can contact the Chair of the University of Cape Town Research Ethics Committee at 021-406-6338.

Do I have to be in the study?
You don't have to be in this study if you don't want to or you can stop being in the study at any time. Please discuss your decision with your parents and researcher. No one will be angry if you decide to stop being in the study.

AGREEMENT TO BE IN THE STUDY

Your signature below means that you have read the above information about the study and have had a chance to ask questions to help you understand what you will do in this study. Your signature also means that you have been told that you can change your mind later and withdraw if you want to. By signing this assent form you are not giving up any of your legal rights. You will be given a copy of this form.

_____ _____

Signature of Participant (13 yr & older) Date

_____
Printed name of Participant (13 yr & older)

_____ _____
**Signature of Witness (When applicable) Date

** Use when participant has had consent form read to them (i.e., illiterate, legally blind, translated into foreign language).

_____
Printed Name of Witness

_____ _____
Signature of Person who explained this form Date

_____
Printed Name of Person who explained form

Informed Consent - Afrikaans

Titel van Studie: Neurale Basis van Kognitiewe en Gedragseffekte van FASD Adolessente Assessering

Ons wil u en u kind _____ graag uitnooi om verder deel te neem aan ons navorsingstudie waarin baie van julle betrokke was sedert u swanger was en u baba gebore is. Lees asseblief hierdie vorm deur en vra enige vrae wat u mag hê voordat u instem om in die studie te wees. Die persone wat hierdie studie onderneem is dokters en wetenskaplikes: Christopher Molteno, M.D. en Ernesta Meintjes, Ph.D., van die Fakulteit Gesondheidswetenskappe aan die Universiteit van Kaapstad in Suid Afrika; Nadine Gaab, Ph.D., van Boston Kinderhospitaal/Harvard Universiteit, Verenigde State van Amerika; Andrea Berger, Ph.D., van Ben Gurion Universiteit in die Negev, Israel; R. Colin Carter, M.D., van Columbia Universiteit Kollege van Interniste en Chirurge; en Sandra W. Jacobson, PhD. en Joseph L. Jacobson, Ph.D., van Wayne Staatsuniversiteit se Mediese Skool in die Verenigde State. Die studie word geborg deur die Nasionale Instituut oor Alkohol Misbruik en Alkoholisme in die Verenigde State. Die studie sal uitgevoer word volgens die etiese riglyne en beginsels van die internasionale Verklaring van Helsinki, asook die Suid-Afrikaanse navorsings etiek riglyne soos weergegee deur die Departement van Gesondheid in “Etiek in gesondheids navorsing: beginsels, prosesse en structure”, tweede uitgawe, 2015.

Doel

U en u kind word gevra om in 'n navorsingstudie te wees sodat ons kan leer of sommige aspekte van hoe ‘n kind leer en optree beïnvloed word deur die moeder se dieet, alkohol, rook of dwelmgebruik tydens swangerskap en hoe hierdie blootstellings die ontwikkelende brein beïnvloed. Sommige van die kinders in die studie sal fetale alkoholsindroom (FAS) hê, ander sal aandag tekort hiperaktiwiteit versteuring (ADHD) hê en ander sal nie een van hierdie twee hê nie. Ons wil sien of rekenkunde en lesery en breinaktiwiteit gedurende rekenkunde en lesery soortgelyk of verskillend is vir diegene met en sonder ADHD wat tydens swangerskap aan alkohol blootgestel was of nie. Ander doeleindes van die studie is om te sien of u kind se vermoëns wanneer hy/sy 'n baba was en op 5- of 10-jarige ouderdom voorspel hoe hy of sy nou doen as 'n jeugdige. Die studie word gedoen by die Universiteit van Kaapstad. Ons verwag dat daar ongeveer 220 studiedeelnemers in ons laboratorium gesien sal word. Om u te help besluit of u wil instem om met u kind aan hierdie studie deel te neem of nie, het 'n projek personeellid met u gepraat oor die risiko's en voordele van die studie. Hierdie toestemmingsvorm is ‘n opsomming van die inligting wat aan u gegee is deur die projek personeellid tydens hierdie ingligte toestemmingsproses. **Lees asseblief hierdie vorm deur en vra enige vrae wat u mag hê voordat u instem om in die studie te wees.**

In hierdie navorsingstudie, sal ons nuwe metodes vir die bestudering van die brein gebruik, genoem gebeurtenis-verwante potensiale (ERPs), om beter te verstaan hoe dieet, alkohol, rook, of dwelmgebruik tydens swangerskap 'n kind se ontwikkeling kan beïnvloed, veral sy of haar lesery, rekenkunde, aandag, geheue, gedrag en groei. ERP is 'n nuwe nie-invallende manier om die elektriese aktiwiteit van die brein te bestudeer. Ons sal ook gebruik maak van metodes om die brein te bestudeer genoem magnetiese resonansiebeelding (MRB), wat gebruik was in u vorige besoeke aan die Kaapse Universiteite Breinbeeldingsentrum (CUBIC) by Tygerberg Hospitaal. Gedurende die MRB assessering, lê u kind so stil as moontlik in 'n skandeerder wat magnete gebruik om foto's van die brein te neem. Ons sal

hierdie foto's in die skandeerder neem terwyl u kind 'n video kyk en dan 'n paar lees-, geheue- en rekenkundige take, asook ander kognitiewe en gedragstake doen.

Studie Prosedures

Indien u instem om u kind aan hierdie studie te laat deelneem, sal ons u en u kind na ons laboratorium bring by die Universiteit van Kaapstad (UK) vir twee besoeke wat elk ongeveer 3-4 ure sal duur vir 'n totaal van ongeveer 6-8 ure. Omtrent 3-4 maande later sal u en u kind terug kom na ons laboratorium toe vir nog twee besoeke wat elk ook ongeveer 3-4 ure sal duur, vir ‘n total van omtrent 6-8 ure.

- Ons sal begin deur u en u kind aan die navorsingspersoneel voor te stel en sal elke dag voordat die assessering begin vir julle albei ontbyt gee.
- Tydens die besoeke, sal u kind take doen wat kyk na aandag- en leervermoë, geheue, sosiale/emosionele funksionering, rekenkunde en lesery. Verskeie van hierdie is rekenaartake.
- Dan sal ons bestudeer hoe sy/haar brein reageer wanneer hy/sy aan rekenkunde en leesprobleme en verwante kognitiewe/gedragstake werk, met behulp van ERP. Om dit te doen, sal ons breinaktiwiteit ondersoek terwyl hy/sy na 'n video kyk.
- Voordat die ERP take begin, sal ons 'n spesiale net met klein sensors in warm water week voordat ons dit op sy/haar kop sit. Ons sal dan die net op u kind se kop plaas om die breinaktiwiteit aan te teken, terwyl hy/sy na die video kyk - dit neem minder as 5 min om te doen.
- Elke ERP-taak neem 15-20 min. U kind sal twee ERP-take op elke dag van die eerste twee besoeke voltooi, een in die vroeë oggend en die ander een later tydens die besoek. Daar sal 'n video-opname van u kind gemaak word terwyl hy/sy elke taak uitvoer. Die video-opnames sal slegs deur lede van ons navorsingsspan gesien word en sal na 10 jaar vernietig word.
- In 'n ander taak, sal ons vir u kind 'n paar prentjies wys en hom/haar vra om vir ons 'n storie te vertel oor wat in die prentjie aan die gang is, wat gelei het tot die toneel, wat die mense dink of voel en wat dalk volgende kan gebeur. U kind se stem sal opgeneem word terwyl hy/sy hierdie stories vertel.
- Ons sal ook u kind se gehoor en sig toets en toets of hy/sy regs-of linkshandig is.
- Ons sal u kind weeg en meet en ‘n foto neem om te kyk vir gesigskenmerke wat dikwels verbandhou met alkohol blootstelling tydens swangerskap.
- Ons sal u kind vra oor sy/haar dieet, alkohol- en dwelmgebruik en gedrag.
- U sal in 'n kamer in die omgewing teenwoordig wees tydens al u kind se assesserings en sal saam met u kind wees tydens die fisiese groei ondersoek.
- Tydens hierdie besoek, sal ons u 'n paar vrae vra oor u kind se aandagvermoë en gedrag, sy/haar skool- en gesondheidsgeskiedenis en enige medikasie wat hy/sy neem. Ons sal u ook vra om 'n vorm te voltooi wat u kind se onderwyser vra om die DBD-skaal in te vul, wat vir ons inligting gee oor u kind se gedrag by die skool.
- Ons sal u vra om ons op hoogte te bring oor die werk wat u gesin doen, u huidige drank- en dwelmgebruik en rookpatrone, aandagprobleme wat u as 'n kind kon gehad het (en stresvolle gevoelens wat u ervaar, insluitend hartseer, angs en nood).
- Tydens een besoek sal ons navorsingsverpleegster u dalk vra om omtrent 10ml (omtrent 2 teelepels) speeksel (spoeg) van u en u kind te verskaf om gene (eienskappe wat jy van jou ouers erf) en veranderinge in gene wat deur dieet en alkohol blootstelling veroorsaak word (epigenetiese verskille) te bestudeer wat gevind is verband hou met verskille in alkohol gebruik, voeding, depressie, gehegtheid, of kinders se aandag/gedrag en ontwikkeling. Hierdie monsters sal gestoor word en vir toekomstige genetiese en/of epigenetiese analises gebruik word.

- Ons wil ook graag hê dat u kind vir ons 'n klein urinemonster (ongeveer 3 eetlepels) gee, wat gebruik mag word om te kyk na chemikalieë wat ons vertel van u kind se dieet en hoe u kind se liggaam gebruik maak van wat hy/sy eet. Ons sal ook na chemikalieë in die urine kyk wat die gevolg is van die gebruik van dwelms, soos marijuana (dagga) en metamfetamien (tik). Ons sal ook 'n urine swangerskap toets vir die meisies doen. Om vertroulikheid te behou, kan ons nie die resultate van hierdie toetse met ouers deel nie, maar sal u kind aanmoedig om hierdie informasie met u te deel.
- U en u kind het altyd die opsie om sommige van die vrae nie te beantwoord nie en sal steeds in staat wees om in die studie te bly.
- U en u kind se name sal nie op enige van ons onderhoude of toetsmateriaal verskyn nie. In plaas daarvan sal u geïdentifiseer word deur 'n kode nommer (sien Vertroulikheid hieronder).
- Ons sal die bevindinge van hierdie studie slegs gebruik vir navorsingsdoeleindes.

Op 'n ander dag, sal ons navorsingsverpleegster en bestuurder u en u kind na die Universitiet Kaapstad kampus van CUBIC toe neem, wat by Groote Schuur Hospitaal geleë is, vir een besoek wat ongeveer 3 ure in totaal behoort te neem.

- Ons sal begin deur u kind te wys wat in die skandeerder gaan gebeur deur in 'n kamma-skandeerder te oefen waarin hy/sy dieselfde soort klanke sal hoor en sal oefen om 'n knoppie te druk soos die een wat tydens die skandeering gebruik word.
- Gedurende die skandering, sal u kind op 'n opgestopte plastiek bed lê wat in die skandeerder inskuif. Ons sal u kind vra om so stil as moontlik te lê terwyl die skanderings geneem word. Die neem van hierdie foto's van die brein maak nie seer nie en word elke dag deur baie mense in die hospitaal gebruik. Vir 'n gedeelte van die tyd in die skandeerder, sal u kind na videos kyk en vir 'n gedeelte van die tyd sal hy/sy leestake of ander verwante kognitiewe of gedragstake doen.
- Daar sal twee sessies in die skandeerder op dieselfde dag wees; elkeen sal nie langer as 45-60 minute neem nie, vir 'n totaal van ongeveer 3-4 ure (insluitend etes).
- Kinders met die volgende mag nie 'n MRB ondergaan nie, maar sal deelneem aan die res van die besoeke: ingeplante mediese toestelle soos aneurisme knippies in die brein, hart pasaangeërs en kogleêre (binne-oor) inplantings; lood-gebasseerde tattoeërmerke; of stukkies metaal naby aan of binne-in 'n belangrike orgaan (soos die oog); engtevrees of die vrees om binne 'n klein ruimte beperk te wees.

Gedurende elkeen van die besoeke aan ons laboratorium by die Universiteit van Kaapstad en aan CUBIC, sal u en u kind ontbyt, 'n peuselhappie en middagete gegee word. Alles tesaam, sal die vyf besoeke (twee aan ons laboratorium nou en twee in 3-4 maande en een aan CUBIC) 'n totaal van ongeveer 15-20 ure neem.

Voordele

As 'n deelnemer in hierdie navorsingstudie, mag daar dalk geen direkte voordele vir u wees nie; inligting van hierdie studie kan egter ander mense nou of in die toekoms help. Die moontlike voordele aan u vir deelname aan hierdie navorsingstudie is dat ons vir u inligting sal gee oor u kind se ontwikkeling op hierdie ouderdom. Indien 'n ernstige probleem gevind word, sal ons vir u sê en u kind verwys na 'n dokter en/of iemand wat kan help, as u wil hê ons moet dit doen. Indien u kind aan enige ernstige siekte ly, sal ons u na Rooikruis Kinderhospitaal of Groote Schuur Hospitaal stuur. Geen inligting oor u kind sal aan enige dokters, hospitale, of skole gegee word nie, tensy u dit skriftelik versoek en toelaat.

Risiko's
Daar is tans geen bekende risiko's tot deelname aan hierdie studie nie. Daar is geen bekende skadelike gevolge van ERP of MRB wat in hierdie studie gebruik word nie. Gedurende die MRB neurobeelding assesserings, mag sekere metaalvoorwerpe, soos horlosies, kredietkaarte, haarknippies en skryfpenne, beskadig word deur die MRB skandeerder of van die liggaam af weggetrek word deur die magneet. Vir hierdie redes, sal ons u kind vra om hierdie voorwerpe te verwyder voordat hy/sy die skandeerder binnegaan. Wanneer die skandeerder die foto's maak, mag die bed skud en u kind sal harde kapgeluide hoor. Hy/sy sal oorpluisies of oorfone gegee word om die ore te beskerm. Sommige mense voel ook senuweeagtig in 'n klein geslote ruimte, soos wanneer hulle in die skandeerder is. U kind sal te alle tye by die skandeerder kan uitsien, en ons sal nie begin voordat hy/sy vir ons sê dat hy/sy gemaklik is nie. U mag dalk hartseer of ontsteld voel wanneer u gevra word om te praat oor hartseer, angs, of stresvolle gebeurtenisse in u lewe. 'n Verwysing kan gemaak word om te help met hierdie gevoelens, as u dit wil hê. Geen name word op die onderhoud of assesseringsvorms aangeteken nie. Alle name word in geslote lêers gehou in ons kantore wat gesluit word by die UK en WSU. Daar is 'n klein risiko dat u identiteit geopenbaar kan word, omdat ons hierdie inligting moet hou sodat ons u kan kontak om deel te neem aan hierdie navorsing en u kan vervoer na ons laboratorium. Daar is min risiko dat enigiets wat u vir ons vertel vir mense buite die studie gesê sal word en ons sal alles doen wat ons kan om hierdie inligting geheim te hou, soos hieronder beskryf, behalwe dat die volgende inligting aan die toepaslike owerhede gerapporteer moet word, indien daar ter enige tyd tydens die studie kommer is dat:

- kindermishandeling of mishandeling van bejaardes moontlik plaasgevind het.

Daar mag dalk ook risiko's betrokke wees in deelname aan hierdie studie wat nie op hierdie stadium aan navorsers bekend is nie.

Studiekostes
Daar sal geen koste vir u of u kind wees om aan hierdie studie deel te neem nie en u en u kind sal deur ons bestuurder na die laboratorium by die Universiteit van Kaapstad vervoer word.

Vergoeding
Vir u deelname aan hierdie navorsingstudie, sal ons u R150 vir elke besoek gee, vir 'n totaal van R750, asook ‘n foto van u kind en ons sal vir u kind ‘n klein geskenkie gee. Ons sal ook vir u en u kind ontbyt, 'n peuselhappie en middagete gee met elke besoek.

Wat as Iets Verkeerd Gaan?
Die Universiteit van Kaapstad (UK) onderneem dat indien u of u kind enige noemenswaardige agteruitgang in gesondheid of welstand ondervind, of ly aan enige onverwagte sensitiwiteit of newe effek, wat veroorsaak is deur u deelname aan die studie, UK onmiddelike mediese sorg sal voorsien. UK het die toepaslike versekering om vinnig vergoeding te betaal vir enige navorsingsverwante besering volgens die riglyne van die “Association of the British Pharmaceutical Industry”, ABPI 1991. Basies beveel die ABPI riglyne aan dat die versekerde besigheid (UK), sonder wederregterlike voorbehoud, u moet vergoed sonder dat u hoef te bewys dat dit UK se fout of skuld was. ‘n Besering word beskou as navorsingsverwant indien, en tot die mate wat, dit veroorsaak is deur die aktiwiteite van die studie. U moet die studie doktor onmiddelik in kennis stel van enige newe effek en/of besering gedurende die studie, nieteenstaande of dit navorsingsverwant is of aan ‘n ander komplikasie verwant is nie. Indien u dink dat u of u kind 'n navorsingsverwante besering opgedoen het, kontak asseblief dadelik Dr. Christopher Molteno by 021-406-6291

(Departement van Menslike Biologie, Anatomie Gebou Suite 7.02, Anzioweg, Observatory 7925, Kaapstad).

UK behou die reg om nie vergoeding te voorsien nie as, en tot die mate wat, u of u kind se besering te weeg gebring is omdat u gekies het om nie die instruksies wat u gegee is terwyl u deelgeneem het aan die studie te volg nie. U wettiglike reg om vergoeding te vereis vir ‘n besering waar u nalatigheid bewys word nie hierdeur beïnvloed nie. Afskrifte van hierdie riglyne is beskikbaar indien u dit versoek. Geen terugbetaling, vergoeding, of gratis mediese sorg word deur Wayne Staatsuniversiteit verskaf nie.

Vertroulikheid
Alle inligting wat deur die loop van hierdie studie oor u en u kind versamel word, sal geheim gehou word tot die mate wat die wet dit toelaat. Julle sal in die navorsingsrekords deur 'n kodenommer geïdentifiseer word. Ons sal nie inligting uitgee wat julle persoonlik identifiseer nie tensy u ons skriftelike toestemming gee. U rekords mag egter hersien word deur die studieborg, die Oorsigkomitee (IRB) by Wayne Staatsuniversiteit en die Universiteit van Kaapstad se Navorsingsetiek-komitee, of regeringsliggame met toepaslike regulatoriese oorsig. Die lys wat deelnemers se identifikasienommers met hul name verbind sal gestoor word in geslote kabinette in die navorsingslaboratorium. Slegs personeellede wat nodig het om u telefonies of persoonlik te kontak sal toegelaat word om na hierdie leêrs te kyk.

Inligting uit hierdie studie mag aangebied word in wetenskaplike vergaderings of joernale, of vir opleidingsdoeleindes gebruik word, maar u en u kind se name sal geheim gehou word. Ons het 'n handtekening-lyntjie in die toestemmingsvorm bygevoeg wat toestemming vra om soms 'n foto of video vir navorsing of opvoedkundige doeleindes te gebruik. Ons sal dit slegs gebruik as ons u skriftelike toestemming het. Toestemming om die foto's of video's te gebruik is nie nodig vir u om deel te neem aan die studie nie.

Vrywillige Deelname/Onttrekking
Deelname aan hierdie studie is vrywillig. U het die reg om te kies om nie deel te neem aan hierdie studie nie. As u besluit om u kind aan die studie te laat deelneem, mag u later van besluit verander en die studie los. U en u kind het ook die reg om enige vrae nie te beantwoord nie, of om enige taak te stop voordat dit klaar is. U besluite sal nie enige huidige of toekomstige verhouding verander met die Universiteit van Kaapstad of Wayne Staatsuniversiteit of ander dienste waartoe u geregtig is nie.

Die navorser of die borg mag ook u kind se deelname aan hierdie studie stop sonder u toestemming. Die hoofnavorser sal die besluit maak en u laat weet as dit nie vir u moontlik is om voort te gaan nie. Die besluit wat gemaak word, is vir die beskerming van u gesondheid en veiligheid of omdat u nie die instruksies gevolg het om deel te neem aan die studie nie.

Vrae
Indien u nou of in die toekoms enige vrae oor hierdie studie het, kan u Prof. Christopher Molteno of een van sy navorsingspanlede kontak by 021-406-6212. Indien u vrae of bekommernisse het oor u regte as ’n navorsingsdeelnemer, kan u die Voorsitter van die Universiteit van Kaapstad se Navorsingsetiek-komitee skakel (021-406-6338).

Toestemming om aan ‘n Navorsingstudie Deel te Neem
Om vrywilliglik in te stem om saam met u kind aan hierdie studie deel te neem, moet u op die lyntjie hieronder teken. Indien u kies om saam met u kind aan hierdie studie deel te neem,

mag u of u kind op enige stadium julle deelname stop. U gee nie enige van u of u kind se wettiglike regte op deur hierdie vorm te teken nie. U handtekening wys dat u hierdie vorm heeltemal deurgelees het, of dat dit aan u voorgelees is, insluitende die dele wat die risiko's en voordele verduidelik, en dat ons al u vrae beantwoord het. Ons sal vir u 'n afskrif van hierdie toestemmingsvorm gee om huis toe te vat.

_____ _____
Handtekening van Ouer of Wetlike Voog Datum

_____ _____
Naam van Ouer of Wetlike Voog in Drukskrif Tyd

_____ _____
Handtekening van Getuie* Datum

_____ _____
Naam van Getuie in Drukskrif* Tyd

_____ _____
Handtekening van Persoon wat Toestemming Verkry Datum

_____ _____
Naam van Persoon wat Toestemming Verkry in Drukskrif Tyd

Ek gee hiermee toestemming dat 'n foto of video van my kind en/of van my gebruik en behou mag word vir navorsing of opvoedkundige doeleindes:

Ja____ Nee_____

_____ _____
Handtekening van Ouer of Wetlike Voog Datum

_____ _____
Naam van Ouer of Wetlike Voog in Drukskrif Tyd

Mag ons u weer kontak om aan toekomstige studies deel te neem?

_____ Ja _____ Nee

_____ _____
Handtekening van Ouer of Wetlike Voog Datum

_____	_____
Naam van Ouer of Wetlike Voog in Drukskrif	Tyd

***** Gebruik wanneer die toestemmingsvorm aan die ouer voorgelees is (m.a.w. in gevalle van ongeletterdheid, blindheid, vertaling in ‘n ander taal)

Informed Assent – Afrikaans

Adolessente Instemmingsvorm

(ouderdomme 13-17 jaar)

Titel: Neurale Basis van Kognitiewe en Gedragseffekte van FASD

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Hoekom is ek hier?

Hierdie is ‘n navorsingstudie. Slegs mense wat kies om deel te neem word ingesluit by navorsingstudies. Jy word gevra om deel te neem aan hierdie studie omdat baie van julle deel is van 'n groot groep kinders wat aan die studie deelgeneem het vandat julle gebore is en aan besoeke as 'n baba en op 5- en 10-jarige ouderdom. Sommige van julle was nog nie deel van hierdie studie nie. Ons nooi julle almal uit om deel te neem aan die volgende fase van hierdie studie. Vat asseblief jou tyd om ‘n besluit te neem. Gesels met jou familie daaroor en maak seker om vrae te vra oor enige iets wat jy nie verstaan nie.

Hoekom doen hulle hierdie studie?

- Hierdie studie word gedoen om te bestudeer hoe tieners rekenkundige probleme oplos en om jou lesery te bestudeer. Ons wil sien hoe jou brein reageer wanneer jy rekenkundige probleme doen en lees.
- In hierdie navorsingstudie, sal ons nuwe metodes vir die bestudering van die brein gebruik, genoem gebeurtenis-verwante potensiale (ERPs), om beter te verstaan hoe dieet, alkohol, rook, of dwelmgebruik tydens swangerskap ontwikkeling kan beïnvloed, insluitende lees, rekenkunde, aandag, geheue, en gedrag. ERP is 'n nuwe nie-invallende manier om die elektriese aktiwiteit van die brein te bestudeer.
- Ons wil ook sien of rekenkunde en lesery en breinaktiwiteit gedurende rekenkunde en lesery soortgelyk of verskillend is vir diegene met en sonder aandag tekort hiperaktiwiteit versteuring (ADHD).
- Ons sal ook gebruik maak van metodes om die brein te bestudeer genoem magnetiese resonansiebeelding (MRB), waaraan baie van julle deelgeneem het tydens vroeër besoeke aan die Kaapse Universiteite Breinbeeldingsentrum (CUBIC). Gedurende die MRB assessering, lê jy so stil as moontlik in die skandeerder wat magnete gebruik om foto's van jou brein te neem. Ons sal hierdie foto's in die skandeerder neem terwyl jy 'n video kyk en dan 'n paar lees-, geheue-, rekenkundige en ander leer- en gedragstake doen.

Wat sal met my gebeur?

Hier by die Universiteit van Kaapstad, sal ons kyk hoe jy rekenkunde en leesprobleme doen. Dan sal ons kyk hoe jou brein reageer wanneer jy rekenkunde en leesprobleme doen met behulp van ERP. Om dit te doen, sal ons jou breinaktiwiteit ondersoek terwyl jy na 'n video kyk. Voordat die taak begin, sal ons 'n spesiale net met klein sensors in warm water week voordat ons dit op jou kop sit. Ons sal dan die net op jou kop sit om jou breinaktiwiteit aan te teken terwyl jy na die video kyk. Daar sal 'n video-opname van jou gemaak word terwyl jy hierdie take doen. In 'n ander taak sal ons jou vra om 'n storie te vertel oor wat aangaan in sommige prentjies wat ons vir jou wys, wat die mense besig is om te dink of voel en wat dalk volgende kan gebeur. Jou stem sal opgeneem word terwyl jy hierdie stories vertel. Jy sal ook 'n paar ander take voltooi wat te doen het met hoe jy leer, onthou, en aandag gee. Sommige

van hierdie sal potlood en papier take wees en ander sal rekenaar take wees. Ons sal jou ook weeg, meet hoe lank jy is, 'n foto van jou neem en kyk hoe goed jy kan sien en hoor. Ons sal jou vra oor jou huidige rookpatrone, drank- en dwelmgebruik en ons sal jou huidige klasonderwyser vra oor jou skoolwerk en gedrag.

Jy sal vanoggend hier spandeer en sal weer terugkom na die Universiteit van Kaapstad op 'n ander dag nou, en dan weer vir twee dae in ‘n paar maande om nog van die breinaktiwiteit en ander take te doen.

Op 'n ander dag, sal ons navorsingsverpleegster en bestuurder jou en jou ouer na die Universiteit Kaapstad kampus van CUBIC toe neem, wat by Groote Schuur Hospitaal geleë is, vir een besoek wat omtrent 3 ure in totaal behoort te neem. Gedurende hierdie besoek sal ons jou wys wat in die skandeerder gaan gebeur deur in 'n kamma-skandeerder te oefen waarin jy die soorte klanke sal hoor en sal oefen om 'n knoppie te druk soos die een wat tydens die skandeering gebruik word. Gedurende die skandering, sal jy op 'n opgestopte plastiek bed lê wat in die skandeerder inskuif. Ons sal jou vra om so stil as moontlik te lê terwyl die skanderings geneem word. Die afneem van hierdie foto's van die brein maak nie seer nie en word elke dag deur baie mense in die hospitaal gebruik. Vir 'n gedeelte van die tyd in die skandeerder, sal jy na videos kyk en vir 'n gedeelte van die tyd sal jy leestake of ander verwante kognitiewe of gedragstake doen.

Ons sal jou ook vra om vir ons 'n klein urinemonster (ongeveer 3 eetlepels) te gee, wat gebruik mag word om te kyk na chemikalieë wat ons vertel van jou dieet; hoe jou liggaam gebruik maak van wat jy eet; wat die gevolg is van die gebruik van dwelms, soos marijuana (dagga) en metamfetamien (tik). Ons sal ook 'n urine swangerskap toets vir die meisies doen voordat julle deelneem aan die skandeerder besoek. Om vertroulikheid te behou, sal ons nie die resultate van hierdie toetse met ouers deel nie, maar sal julle aanmoedig om dit te doen.

Ons sal jou dalk ook vra vir 'n klein speeksel(spoeg)-monster (omtrent 2 teelepels) om gene (eienskappe wat jy van jou ouers erf) te bestudeer en veranderinge in gene wat deur dieet en alkohol blootstelling veroorsaak word (epigenetiese verskille) wat gevind is verband hou met verskille in alkohol gebruik, voeding, depressie, gehegtheid, of kinders se aandag/gedrag en ontwikkeling. Hierdie monsters sal gestoor word en vir toekomstige genetiese en/of epigenetiese analises gebruik word.

Hoe lank sal ek in die studie wees?

Jy sal vir hierdie fase in die studie wees vir twee dae nou, vir omtrent 3-4 ure elke keer, by ons laboratorium by die Universiteit van Kaapstad (wat insluit ontbyt, 'n peuselhappie en middagete), en dan weer in ‘n paar maande vir 2 dae, weer vir ongeveer 3-4 ure elke keer. Daar sal ook twee sessies in die CUBIC skandeerder wees op een dag; elke sessie sal nie langer as 45-60 minute neem nie, vir 'n totaal van ongeveer 3-4 ure (wat insluit ontbyt, 'n peuselhappie en middagete).

Sal die studie my help?

Jy sal nie daarby baat om in hierdie studie te wees nie, maar inligting uit hierdie studie kan ander mense in die toekoms help om beter te verstaan hoe die brein verskillende take verrig en of dieet, alkohol, rook, of blootstelling aan dwelms gedurende swangerskap beïnvloed hoe die brein werk.

Sal enige iets sleg met my gebeur?
Daar is ‘n moontlikheid dat sommige van die vrae jou hartseer of ongemaklik sal laat voel. As jy so voel, kan ons iemand kry wat met jou daaroor kan gesels. Ons verwag geen ander risiko's verbonde aan enige van die take wat ons met jou sal doen in ons laboratorium by die Universiteit van Kaapstad of in die skandeerder nie.

Weet my ouers of voogde hiervan?
Hierdie studie-inligting is aan jou ouer/voog gegee en hy/sy het gesê dat jy mag deelneem aan die studie. Jy kan met hom/haar hieroor praat voordat jy besluit.

Navorsingsverwante Beserings
Indien hierdie navorsingsverwante aktiwiteite lei tot 'n besering, sal behandeling beskikbaar gemaak word, insluitend eerstehulp, noodbehandeling, en opvolg-sorg, soos benodig. Sulke sorg sal betaalbaar wees in die gewone manier, deur jou of jou versekeringsmaatskappy/Suid-Afrikaanse openbare hulp. Geen terugbetaling, vergoeding, of gratis mediese sorg word verskaf deur Wayne Staats Universiteit of die Universiteit van Kaapstad nie. As jy dink dat jy 'n navorsingsverwante besering opgedoen het, kontak asseblief dadelik die Kaapstad hoofnavorser Prof. Christopher Molteno by 021-406-6212.

Wat van vertroulikheid?
Elke redelike poging sal aangewend word om jou rekords (mediese of ander) en/of jou inligting konfidensieel te hou, maar ons moet sommige mense na jou studierekords laat kyk.

Ons sal jou rekords geheim hou tensy ons deur die wet vereis word om enige inligting te deel. Die wet sê dat ons iemand moet vertel as jy dalk jouself of iemand anders mag seer maak. Die studiedokter mag die studie resultate gebruik so lank as wat jy nie geïdentifiseer kan word nie.

Die volgende inligting moet vrygelaat/gerapporteer word aan die toepaslike owerhede indien daar te eniger tyd gedurende die studie kommer is dat:

- kindermisbruik of mishandeling van bejaardes moontlik plaasgevind het.

Vergoeding
Terwyl jy in hierdie fase van die navorsingstudie deelneem, sal ons vir jou 'n klein geskenkie gee en 'n foto wat van jou geneem is. Ons sal ontbyt, 'n peuselhappie en middagete voorsien elke keer as jy na ons laboratorium toe kom by die Universiteit van Kaapstad.

Wat as ek enige vrae het?
Vir vrae oor die studie kontak asseblief vir Dr. Christopher Molteno by 021-406-6212. As jy enige vrae of bekommernisse het oor jou regte as ‘n deelnemer aan die navorsing, kan jy die Voorsitter van die Universiteit van Kaapstad se Navorsingsetiek-komitee kontak by 021-406-6338.

Moet ek in die studie wees?
Jy hoef nie in hierdie studie te wees as jy nie wil nie of jy kan ophou om in die studie te wees op enige stadium. Bespreek asseblief jou besluit met jou ouers en navorser. Niemand sal kwaad wees as jy besluit om op te hou om in die studie te wees nie.

INSTEMMING OM IN DIE STUDIE TE WEES

Jou handtekening hieronder beteken dat jy die bogenoemde inligting oor die studie gelees het en dat jy kans gekry het om vrae te vra om jou te help verstaan wat jy in hierdie studie gaan doen. Jou handtekening beteken ook dat daar aan jou verduidelik is dat jy later van besluit mag verander en onttrek as jy wil. Jy gee nie enige van jou regte op deur hierdie vorm te teken nie. Ons sal vir jou ‘n kopie van hierdie toestemmingsvorm gee.

_____ _____

Handtekening van Deelnemer (13 j. & ouer) Datum

_____

Naam van Deelnemer in Drukskrif (13 j. & ouer)

_____ _____

** Handtekening van Getuie (Wanneer van toepassing) Datum

** Gebruik wanneer instemmingsvorm aan deelnemer voorgelees is (bv. wanneer ongeletterd, wetlik blind, vertaal in ‘n vreemde taal).

_____

Naam van Getuie in Drukskrif

_____ _____

Handtekening van Persoon wat vorm verduidelik het Datum

_____

Naam van Persoon wat vorm verduidelik het in drukskrif

Appendix C

Collinearity Statistics for Hierarchical Regression Analyses

Table C1.

Final Model Collinearity Statistics for ARC Reflection

Model		Correlations			Collinearity Statistics	
		Zero-order	Partial	Part	Tolerance	VIF
1	Age	.25	.25	.25	1.00	1.00
2	Age	.25	.29	.28	.98	1.02
	Sex	.24	.28	.27	.98	1.02
3	Age	.25	.30	.29	.98	1.02
	Sex	.24	.27	.26	.98	1.02
	SES	.12	.12	.11	.99	1.01
4	Age	.25	.30	.29	.92	1.09
	Sex	.24	.27	.26	.96	1.04
	SES	.12	.11	.10	.92	1.09
	Prenatal smoking	.03	-.03	-.03	.87	1.15
5	Age	.25	.32	.30	.92	1.09
	Sex	.24	.25	.23	.95	1.06
	SES	.12	.04	.04	.87	1.15
	Prenatal smoking	.03	-.00	-.00	.86	1.17
	IQ	.29	.29	.27	.90	1.11
6	Age	.25	.32	.30	.91	1.10
	Sex	.24	.25	.22	.94	1.06
	SES	.12	.05	.04	.86	1.16
	Prenatal smoking	.03	.00	.00	.86	1.17
	IQ	.29	.29	.26	.90	1.12
	Attachment security	.05	.06	.06	.97	1.03
7	Age	.25	.34	.31	.82	1.22
	Sex	.24	.25	.23	.94	1.07
	SES	.12	.04	.04	.86	1.17
	Prenatal smoking	.03	.01	.01	.85	1.17
	IQ	.29	.27	.25	.88	1.13
	Attachment security	.05	.04	.04	.92	1.09
	FASD-related dysmorphism	-.02	-.09	-.08	.80	1.26

Table C2.

Final Model Collinearity Statistics for ARC Lack of Control

Model		Correlations			Collinearity Statistics	
		Zero-order	Partial	Part	Tolerance	VIF
1	Age	.13	.13	.13	1.00	1.00
2	Age	.13	.12	.12	.98	1.02
	Sex	-.11	-.09	-.09	.98	1.02
3	Age	.13	.12	.12	.98	1.02
	Sex	-.11	-.09	-.09	.98	1.02
	SES	-.04	-.03	-.03	.99	1.01
4	Age	.13	.10	.10	.92	1.09
	Sex	-.11	-.10	-.10	.96	1.04
	SES	-.04	-.01	-.01	.92	1.09
	Prenatal smoking	.09	.07	.06	.87	1.15
5	Age	.13	.09	.08	.92	1.09
	Sex	-.11	-.05	-.04	.95	1.06
	SES	-.04	.09	.08	.87	1.15
	Prenatal smoking	.09	.03	.02	.86	1.17
	IQ	-.42	-.41	-.40	.90	1.11
6	Age	.13	.06	.05	.91	1.10
	Sex	-.11	-.03	-.03	.94	1.06
	SES	-.04	.06	.05	.86	1.16
	Prenatal smoking	.09	.01	.01	.86	1.17
	IQ	-.42	-.41	-.39	.90	1.12
	Attachment security	-.31	-.30	-.27	.97	1.03
7	Age	.13	.03	.03	.82	1.22
	Sex	-.11	-.04	-.04	.94	1.07
	SES	-.04	.07	.06	.86	1.17
	Prenatal smoking	.09	.00	.00	.85	1.17
	IQ	-.42	-.40	-.37	.88	1.13
	Attachment security	-.31	-.28	-.25	.92	1.09
	FASD-related dysmorphism	.23	.10	.08	.80	1.26

Table C3.

Final Model Collinearity Statistics for ARC Suppression of Affect

Model		Correlations			Collinearity Statistics	
		Zero-order	Partial	Part	Tolerance	VIF
1	Age	.07	.07	.03	1.00	1.00
2	Age	.07	.07	.07	.98	1.02
	Sex	-.03	-.02	-.02	.98	1.02
3	Age	.07	.06	.06	.98	1.02
	Sex	-.03	-.01	-.01	.98	1.02
	SES	-.11	-.11	-.11	.99	1.01
4	Age	.07	.07	.07	.92	1.09
	Sex	-.03	-.01	-.01	.96	1.04
	SES	-.11	-.11	-.11	.92	1.09
	Prenatal smoking	.01	-.03	-.03	.87	1.15
5	Age	.07	.07	.07	.92	1.09
	Sex	-.03	-.00	-.00	.95	1.06
	SES	-.11	-.11	-.11	.87	1.15
	Prenatal smoking	.01	-.03	-.03	.86	1.17
	IQ	-.05	-.02	-.02	.90	1.11
6	Age	.07	.04	.04	.91	1.10
	Sex	-.03	.02	.02	.94	1.06
	SES	-.11	-.15	-.14	.86	1.16
	Prenatal smoking	.01	-.06	-.05	.86	1.17
	IQ	-.05	.00	.00	.90	1.12
	Attachment security	-.31	-.32	-.32	.97	1.03
7	Age	.07	-.04	-.04	.82	1.22
	Sex	-.03	-.01	-.01	.94	1.07
	SES	-.11	-.13	-.12	.86	1.17
	Prenatal smoking	.01	-.07	-.07	.85	1.17
	IQ	-.05	.03	.03	.88	1.13
	Attachment security	-.31	-.27	-.26	.92	1.09
	FASD-related dysmorphism	.30	.24	.22	.80	1.26

Table C4.

Final Model Collinearity Statistics for ERC Adaptive Emotion Regulation

Model		Correlations			Collinearity Statistics	
		Zero-order	Partial	Part	Tolerance	VIF
1	Age	.12	.12	.12	1.00	1.00
2	Age	.12	.12	.11	.98	1.02
	Sex	-.12	-.10	-.10	.98	1.02
3	Age	.12	.09	.09	.98	1.03
	Sex	-.12	-.09	-.08	.98	1.02
	SES	-.32	-.31	-.31	.99	1.01
4	Age	.12	.04	.04	.93	1.08
	Sex	-.12	-.12	-.11	.96	1.04
	SES	-.32	-.25	-.24	.92	1.09
	Prenatal smoking	.29	.22	.20	.87	1.15
5	Age	.12	.04	.04	.93	1.08
	Sex	-.12	-.07	-.06	.94	1.06
	SES	-.32	-.17	-.15	.86	1.17
	Prenatal smoking	.29	.20	.17	.86	1.16
	IQ	-.43	-.35	-.32	.88	1.14
6	Age	.12	-.01	-.01	.91	1.10
	Sex	-.12	-.05	-.04	.94	1.07
	SES	-.32	-.23	-.19	.84	1.20
	Prenatal smoking	.29	.18	.14	.86	1.17
	IQ	-.43	-.34	-.29	.87	1.15
	Attachment security	-.35	-.37	-.32	.94	1.06
7	Age	.12	-.07	-.05	.78	1.28
	Sex	-.12	-.07	-.05	.93	1.08
	SES	-.32	-.23	-.19	.84	1.20
	Prenatal smoking	.29	.16	.13	.85	1.18
	IQ	-.43	-.32	-.27	.85	1.18
	Attachment security	-.35	-.35	-.30	.92	1.08
	FASD-related dysmorphism	.29	.15	.12	.75	1.33

Table 22.

Final Model Collinearity Statistics for ERC Lability/Negativity

Model		Correlations			Collinearity Statistics	
		Zero-order	Partial	Part	Tolerance	VIF
1	Age	.03	.03	.03	1.00	1.00
2	Age	.03	.05	.05	.98	1.02
	Sex	.18	.18	.18	.98	1.02
3	Age	.03	.04	.04	.98	1.03
	Sex	.18	.19	.19	.98	1.02
	SES	-.16	-.17	-.17	.99	1.01
4	Age	.03	.02	.02	.93	1.08
	Sex	.18	.18	.18	.96	1.04
	SES	-.16	-.14	-.14	.92	1.09
	Prenatal smoking	.16	.10	.09	.87	1.15
5	Age	.03	.02	.02	.93	1.08
	Sex	.18	.15	.14	.94	1.06
	SES	-.16	-.20	-.19	.86	1.17
	Prenatal smoking	.16	.12	.11	.86	1.16
	IQ	.18	.23	.23	.88	1.14
6	Age	.03	.04	.03	.91	1.10
	Sex	.18	.14	.13	.94	1.07
	SES	-.16	-.18	-.17	.84	1.20
	Prenatal smoking	.16	.13	.12	.86	1.17
	IQ	.18	.23	.22	.87	1.15
	Attachment security	.13	.11	.10	.94	1.06
7	Age	.03	.05	.05	.78	1.28
	Sex	.18	.15	.14	.93	1.08
	SES	-.16	-.18	-.17	.84	1.20
	Prenatal smoking	.16	.13	.13	.85	1.18
	IQ	.18	.22	.21	.85	1.18
	Attachment security	.13	.10	.10	.92	1.08
	FASD-related dysmorphism	-.03	-.04	-.04	.75	1.33