DADS MATTER TOO: THE ROLE OF PATERNAL FACTORS ON BIRTH OUTCOMES

By

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DECLARATION

I, **Tadiwa Melinda Mashonganyika**, declare that all the work presented in the current dissertation is original and represents my own body of knowledge. It is being submitted for the degree of Masters of Science in Medicine in the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at this or any other university.

Signature: _____

_____day of_____2019

DEDICATION

I would like to dedicate this dissertation and entire MSc research over the past three years to my beloved parents; Oswald and Eusebia Mashonganyika. Thank you to my siblings, Anesu and Thandeka Mashonganyika. Your support and patience throughout the course of my studies is truly appreciated.

ABSTRACT

Background

Vast evidence exists illustrating the role of maternal factors on key birth outcomes and adverse birth events in low to middle-income countries. Little attention is given to investigating the role of paternal factors, and their independent contribution after adjusting for maternal factors and other known covariates. More importantly, little is known of these associations in African populations undergoing significant health transitions.

Aim

The aim of the study was to investigate and explore the independent associations between paternal, maternal factors and neonatal outcomes namely; birth weight (BW), birth length (BL), head circumference (HC) and ponderal index (PI).

Methods

A cross-sectional study design including a secondary data analysis approach was used. The data were primarily collected by the Soweto first 1000 days (S1000) and Father of the Baby (FOB) studies conducted in Soweto, South Africa in 2013-2015. Paternal, maternal and other key data were used in the analysis. Descriptive statistics were conducted to summarise paternal, maternal and neonatal characteristics. Furthermore, to explore independent and adjusted associations of paternal and maternal factors with birth outcomes, multivariate linear regression models were computed. All statistical significance was defined set at $p<0.05$.

Results

In the current study, paternal factors, namely Body Mass Index (BMI) and waist circumference (WC) were significantly associated with BW (β: -41.00; 95%CI: -79.77; -2.30; p = 0.038), (β: 19.32; 95%CI: 2.21; 36.42; p = 0.027), respectively. However, after adjusting for neonatal factors, self-reported marital status where fathers of the baby were cohabitating with their partners was significantly associated with BW (β: 194.00; 95%CI: 14.71; 373.26; p = 0.034). Once maternal factors were added to the model, all the paternal factors and neonatal BW associations became insignificant. Paternal BMI was significantly associated with HC (β: -0.15; 95%CI: -0.28;-0.01; p =

0.042), however the association diminished once neonatal and maternal factors were added to the model. Maternal BMI and age were independent predictors for BW, but after adjusting for paternal and neonatal factors only maternal BMI remained a significant predictor of BW (β: 21.43; 95%CI: 9.39; 33.48; p = 0.001). Paternal factors were not significantly associated with BW, HC and PI when maternal and neonatal factors were held constant.

As expected, in the full models, gestational age (GA) was a consistent intermediary predictor for BW (β: 132.54; 95%CI: 95.08; 170.0; p = 0.001), BL (β: 0.91; 95%CI: 0.06; 1.23; p = 0.001) and HC (β: 0.42; 95%CI: 0.27; 0.56; p = 0.001). Whilst sex at birth consistently had a significant association with only BL (β: -1.52; 95%CI: -2.80; -0.23; p = 0.021).

Conclusion

Paternal measures of adiposity (BMI and WC) may play an important role in birth outcomes in addition to social support indexed by marital status and these factors may be mediated by maternal and intermediate neonatal factors like GA and sex. Findings affirm the important role of maternal, paternal and neonatal factors in relation to birth outcomes. The current evidence needs to be further researched and strengthened by using a larger sample size.

Keywords: Paternal factors, Maternal factors, Birth Weight, Birth length, Head circumference, Ponderal index, DoHAD, South Africa

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LIST OF ABBREVIATIONS

β:	Beta
BL:	Birth length
BMI:	Body Mass Index
BP:	Blood pressure
BW:	Birth weight
CI:	Confidence interval
cm:	Centimetre
CRP:	C - reactive protein
DEXA:	Dual-Energy X-ray Absorptiometry
DNA:	Deoxyribonucleic Acid
DoHAD:	Developmental origins of Health and Disease
DPHRU:	Developmental Pathways for Health Research Unit
EDC:	Expected dates of confinement
FOB:	Father of the baby
GA:	Gestational age
GDM:	Gestational diabetes
HbA1c:	glycated haemoglobin
HBW:	High birth weight
HC:	Head circumference
HIV:	Human Immunodeficiency Virus
IQ:	Intelligence Quotient
kg:	Kilogram
LBW:	Low birth weight
LIC:	Low Income Country
LMIC:	Low to Middle Income Countries
mmHg:	Millimetres of mercury
NCD:	Non-communicable disease
OR:	Odds ratio
PI:	Ponderal index
PTB:	Preterm birth
R:	Risk
RR:	Relative Risk

S1000:	Soweto first 1000 days
SA:	South Africa
SD:	Standard deviation
SES:	Socio-economic status
SGA:	Small for gestational age
WC:	Waist circumference
WHO:	World Health Organization
Wits:	University of Witwatersrand

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CHAPTER 1: INTRODUCTION

Chapter 1 presents the background information and problem statement related to selected birth outcomes. Additionally, it covers the following, aims and objectives of the study, related conceptual frameworks, structure of the dissertation and contribution of the candidate to the MSc to the study.

1.1 Background and Problem Statement

Globally, the prevalence of infants born before 37 completed weeks of gestation and with a low birth weight (LBW) -weight less than 2500g- continues to increase despite extensive prevention and screening programmes (Altenhöner, Köhler and Philippi, 2016). On a worldwide scale, early infant deaths (deaths that occur during the first week of life) are accountable for at least 75% of all of all neonatal deaths and 40% of them are due to premature births (Blencowe *et al.*, 2013). Newborns that are born prematurely or small for gestational age (SGA) are at a higher risk of perinatal death and severe disability (Colella *et al.*, 2018), this is due to the fact that LBW infants are more susceptible to infectious diseases as compared to an infant born with a normal and healthy birth weight (BW) (Muchemi, Echoka and Makokha, 2015). Additionally, they stand a higher chance of developing chronic diseases such as Type II Mellitus Diabetes and cardiovascular diseases in their later stages of life (Barker *et al.*, 2005) as well as poor mental and psychological development during school-going age (Hack *et al.*, 2009).

Usually, the lower the weight is at birth, the higher a baby's risk of infant mortality, making pre-term births (PTB), SGA and LBW a major cause of infant mortality (Darmstadt *et al.*, 2009). In early childhood, PTB babies who survived usually experience a variety of health and developmental issues including, motor delay, cerebral palsy, poor vision, educational deficits such as low Intelligence Quotient (IQ), limited academic skills, poor social adaptive skills and respiratory illnesses such as asthma (Mayor, 2005; McCormick *et al.*, 2011).

The Developmental origins of health and disease (DoHaD) concept sheds light on how early life environment can impact the long-term risk of non-communicable diseases (NCDs) from childhood to adulthood (Suzuki, 2018). The DoHad theory postulates that if a developing foetus is exposed to a hostile environment (caused by

insults such as infections, poor nutrition, chemicals, metabolite or hormonal distresses), the foetus responds by developing adaptations that foster its immediate viability and its survival if a similar environment is encountered in life (Osmond *et al.*, 1993). Some of the short term adaptations include the down regulation of endocrine and/or metabolic function and the decrease of specific organ function in order to match the low nutrient supply in the deprived uterine development (Mandy and Nyirenda, 2018).

The long term effects of adverse environment are usually subtle, irreversible changes that take place in the development stages of a foetus, have an effect on the structure and functions of some tissues and vital organs as a result of disruptions in gene expression, cell differentiation and proliferation (Barker *et al.*, 2001). Conversely, if the individual then grows up in an extra-uterine environment the opposite of that experienced in the utero would predispose that individual to a higher risk of NCDs (Gluckman *et al.*, 2008) such as diabetes, hypertension and cardiovascular diseases (Goldenberg and Culhane, 2007). The risk of developing NCDs is further exacerbated by excessive weight gain in adult life and by the ageing process (Vickers *et al.*, 2000; Mandy and Nyirenda, 2018). High birth weight (HBW) and obesity may not be directly linked to infant mortality; however it is related to long term health issues such as obesity and cancer (Holman, Buchanan and Cancer Prevention During Early Life Expert Group, 2016).

A study conducted by Fyker and colleagues (2019) supports the statement that African American and Latina women have a higher risk of spontaneous PTB due to social determinants of health and this could be the by linked with maternal stress ((Fryer, Vines and Stuebe, 2019). Long-time stressors such as social and racila discrimination lead to an inclinded rate of of higher premature activation and parturition. A current study also indicates that mothers who received a higher dosage of folic acid gave birth to an infant with a 1.18 larger head circumference compared to the mothers who received a higher dose (Yusuf *et al*., 2019)

Robust literature that investigates paternal factors and birth outcomes is quite sparse; however a study conducted in Taiwan reported that low paternal education (≤ 12 years) had an influence on the neurodevelopmental outcomes of PTB (Lin *et*

al., 2019). The study associated low paternal education levels with low socio-economic status which affects the wellbeing of the family. Another cohort study conducted in Netherlands reported similar findings (Franz *et al.*, 2009). In a study conducted by Tian *et.al* (2019) reported that paternal nutrition had an effect on epigenetic modification (Tian *et al.*, 2019)

Globally, approximately 20 million infants are LBW babies and 96.5% of these births take place in developing countries whilst 15% of these births take place in sub-Saharan Africa (World Health Organization, 2014). The survival gap between infants born in high and low income countries (LICs) are quite significant and continue to widen (Chou *et al.*, 2015). Complications related to premature births and LBW are the single largest cause of neonatal deaths and deaths amongst children under the age of 5 years (Blencowe *et al.*, 2013) – one of the public health issues that the United Nations Sustainable Development Goals aims to alleviate.

Despite South Africa's commendable efforts in trying to decrease neonatal and maternal mortality, premature births and LBW babies; neonatal and maternal mortality and morbidity continue to be a national issue due to social and biological determinants (Sartorius *et al.*, 2011). In South Africa, an estimated 15 neonatal deaths occur per 1000 live births, 34 child mortality (death of children under the age of 5) per 1000 live births and 233 maternal deaths per 100 000 lives (Alabi *et al.*, 2015). Of those deaths that occur amongst children under the age of 5 years, approximately 40% of deaths transpire in the early neonatal period (Kassebaum *et al.*, 2014). This is quite alarming in comparison with developed countries which have much lower rates – 3 neonatal deaths per 1000 live births and 12 maternal deaths per 100 000 lives (Alabi *et al.*, 2015).

Premature births and infant mortality are an important measurement and indicator of a community's health status and life expectancy which have long term effects on an individual's quality of life and are unequally globally distributed (Kim and Saada, 2013a); especially amongst westernized countries. Premature births not only affect the health of the neonate; however they also have detrimental effects on the mother's wellbeing which is also one of the main predictors of maternal mortality – highly preventable deaths (Ruiz *et al.*, 2015).

Foetal growth and development is significantly influenced by the genetic factors inherited by both parents as well as the maternal in utero environment (Hillman, Peebles and Williams, 2013). An increasing amount of literature suggests that LBW and preterm births are not solely a result of individual-level determinants; however they are also a result of harmful social factors that are presented during critical stages of foetal development (Meng, Thompson and Hall, 2013).

While there is an abundance of research that investigates maternal factors that have been identified to influence LBW and have an adverse effect on the development and outcome of the new-born, there appears to be a lack of research that explores the associations between paternal factors and birth outcomes (Misra *et al.*, 2010). Studies rarely delve into the risk factors that fathers contribute towards their infant's adverse birth outcomes (Shah, 2010) and as a result most public health interventions are solely focused on maternal factors.

1.2 Conceptual Framework

To better understand the prominent increase of adverse birth outcomes, a conceptual framework for the societal and individual level determinants of birth outcomes was adopted by Kayode *et.al* (2014). The framework illustrates how parental social and biological factors, amongst others, have a direct effect on the birth outcome of an infant. This research study aims to further investigate the association between paternal factors and neonate outcomes, which is what this research study aims to further investigate.

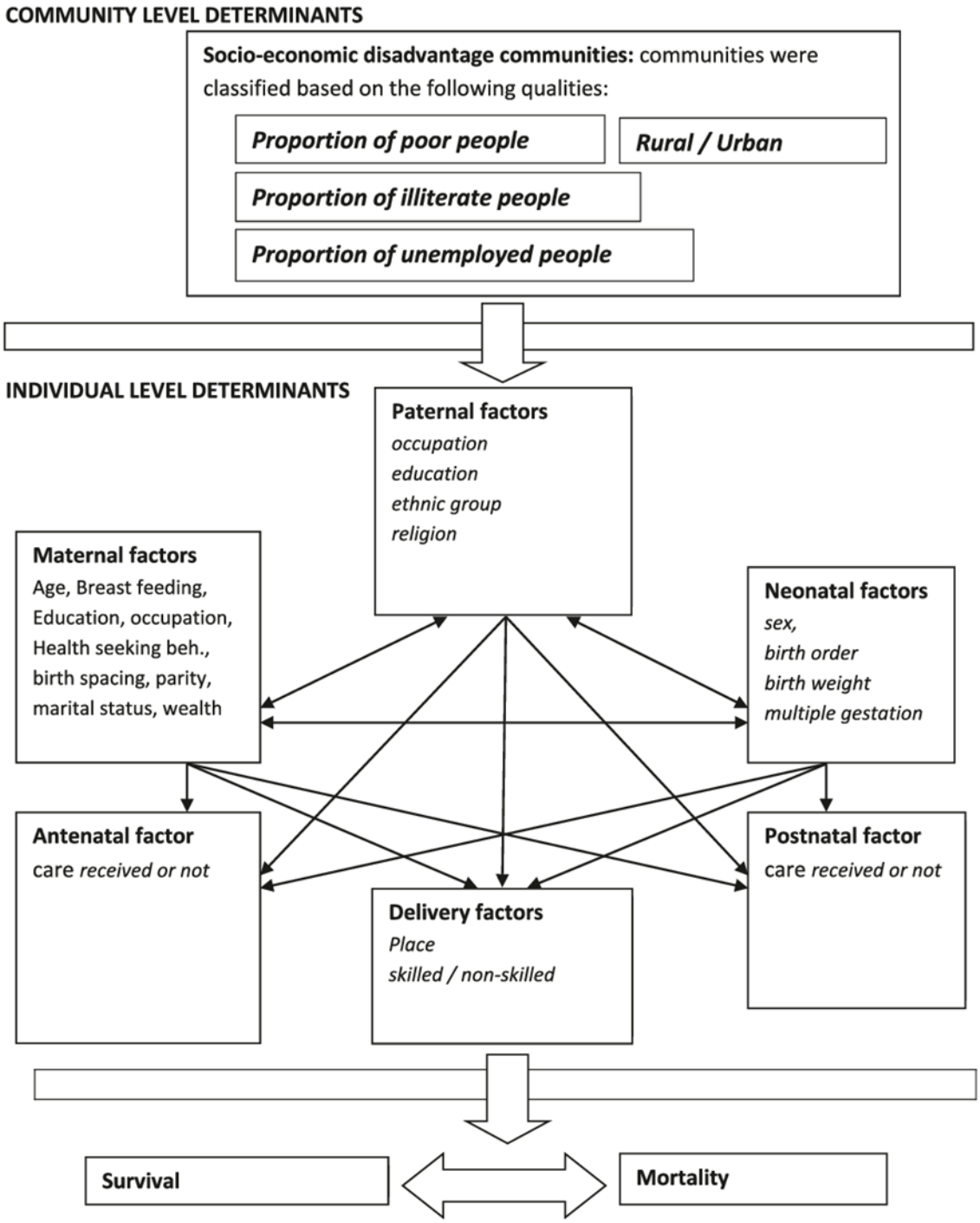

Figure 1: Individual and community determinants of neonatal mortality in Ghana: a multilevel analysis (Kayode *et al.*, 2014)

The social factors include an individual's socio-economic status (SES) which commonly comprises of education, occupation, income, race and ethnicity among others (Meng, Thompson and Hall, 2013)**.** The scientific study of the relationship between socioeconomic status and health dates back at least as far as the 19th

century (Cutler, Lleras-Muney and Vogl, 2008). Since then, a great deal of studies have been developed to measure and describe the relationships between parental socioeconomic status and the effects on child development as research suggests that effects begin prior to birth and continue into adulthood (Bradley and Corwyn, 2002); however most research focuses on the maternal factors and barely investigate the paternal factors. As indicated on the conceptual framework above, intermediary determinants such as biological factors, health seeking behaviours and psychosocial factors also play a crucial role in an infant's birth outcomes.

In figure 2, a theoretical framework was constructed to illustrate what this research study aims to investigate. The theoretical framework takes into consideration one of the gaps that the conceptual framework (figure 1) contains which are associations that paternal socio-biological determinants have on neonatal outcomes (BW, BL, HC and PI).

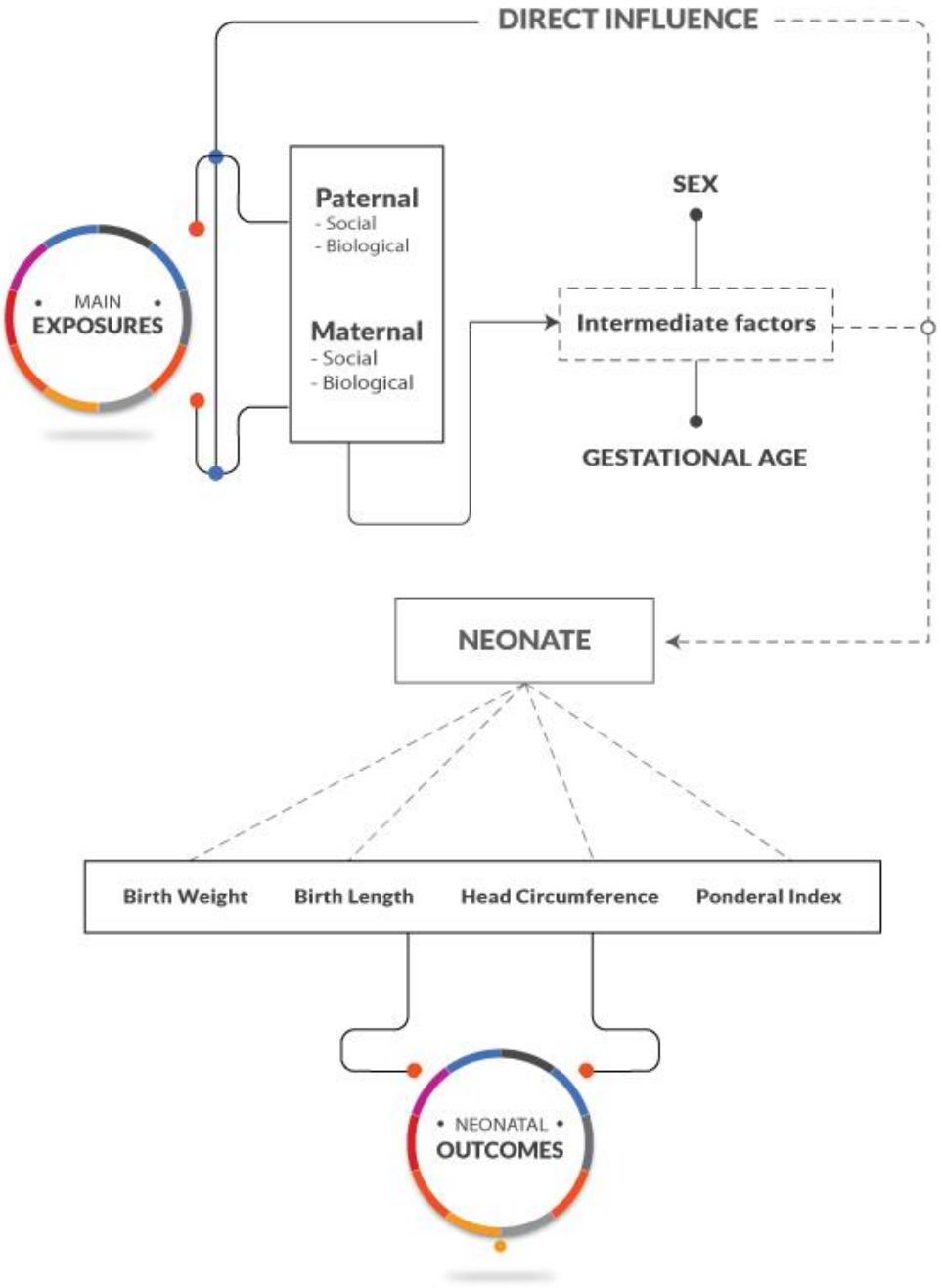

Figure 2: Research Based Theoretical Framework

In light of the conceptual framework (figure 1) that highlights the social determinants of health, an extensive literature review was conducted on paternal and maternal factors related to neonatal outcomes to identify gaps in research and summarized in Table 1 - 4.

1.3 Aim and study objectives

The aim of the study is to investigate and explore the independent associations between paternal factors with birth outcomes namely; birth weight, birth length, head circumference and ponderal index.

Study Objectives

- To explore the role of paternal factors on BW, BL, HC and PI in an African population undergoing a significant epidemiological health transition
- To explore the role of maternal factors on BW, BL, HC and PI in an African population undergoing a significant epidemiological health transition
- To explore the role of paternal factors on BW, BL, HC and PI independent of maternal factors in an African population undergoing a significant epidemiological health transition

  Kindly refer to the methods section where a description of the variables is provided.

1.4 Structure of dissertation

This dissertation is presented in block format. Following this introductory chapter:

Chapter 2: Literature review chapter that showcases the current findings on issues related the dissertation

Chapter 3: Methods chapter that presents an in-depth discussion of all the methods that have been used in carrying out this research. This includes the design, data collection and statistical methods used in the study

Chapter 4: Results chapter that presents detailed tables and narratives of the results

Chapter 5: Discussion chapter is an integrating narrative that summarizes all the results, the public health implications related with adverse birth outcomes and the conclusion of the whole dissertation.

Other annexures including data collection tools and ethical clearance certificate are consolidated at the end of the dissertation.

1.5 MSc candidate contributions to dissertation

The research reported in this dissertation was planned and executed by the MSc candidate, Tadiwa Melinda Mashonganyika. The following contributions were made:

- Obtaining ethical and regulatory approvals
- Conducting all data analysis
- Interpretation of the results and,
- Drafting the dissertation.

CHAPTER 2: LITERATURE REVIEW

2.1 Introduction

Chapter 2 presents a condensed overview of literature related to the role of both paternal and maternal factors on the following birth outcomes: BW, BL, HC and PI. The chapter presents the importance of having optimum neonatal BW, BL, HC and PI, and additionally presents the literature demonstrating the association with either paternal or maternal factors. The literature review section is presented by key outcomes, followed by a highlight of research gaps and structure of the dissertation.

2.2 Importance of key birth outcomes and associations including a lack of consensus with paternal and maternal factors

a. Birth weight

i. Public health significance of birth weight

The following sections summarize the literature on the public health significance of paternal and maternal factors on BW. Being born with an optimum weight is of great importance as infants who have an extremely high or LBW increases the risk of various short and/or long-term adverse health issues. An infant with a LBW is more susceptible to developing chronic conditions such as cerebral palsy, asthma, poor motor skills, and visual disability, poor cognitive ability, academic achievement and social adaptive skills (Mayor, 2005). Like LBW, foetal macrosomia (weight ≥ 4,000 g) also possesses short- and long-term detrimental effects on an infant's health. The long term effects include increased predisposition of developing obesity, type 2 diabetes, hypertension and other NCDs later on in life (KC, Shakya and Zhang, 2015). Table 1 summarizes the literature on the various paternal and maternal factors that have an influence on the neonate's weight at birth. The same table reflects inconsistent literature on the various factors (both paternal and maternal) and their contribution to BW.

ii. Associations between paternal and maternal factors with BW

Table 1: Summary of the literature review – paternal and maternal factors related to BW

Study Title, Author, Year	Study Area	Study Design	Sample Size: N	Paternal/Maternal Exposures	Results
Paternal factors and low birth weight, preterm, and small for gestational age births: a systematic review (Shah *et al.*, 2010)	-	A systematic review	38 reviewed studies	Paternal age, anthropometry, paternal birth weight, occupation, and educational background	Advanced paternal age (41 to 50 years) was associated with higher risk for LBW. Among infants who were born to tall fathers, birth weight was approximately 125-150 g higher compared with infants who were born to short fathers. Paternal LBW was associated with lower birth weight of the offspring. In conclusion, paternal characteristics including age, height, and birth weight are associated with LBW. Paternal occupational exposure and low levels of education may be associated with LBW; however, further studies are needed.
The association between parity, infant gender, higher level of paternal education and preterm birth in Pakistan: a cohort study (Shaikh *et al.*, 2011)	Pakistan	Prospective cohort study	N = 132	Paternal stress, depression and blood cortisol	Almost 20% of pregnant women (19·7%, 95% CI 13·3-27·5) experienced a high level of stress and nearly twice as many (40·9%, 95% CI 32·4-49·8%) experienced depressive symptoms. The median of cortisol level was 27·40 ug/dl (IQR 22·5-34·2). The preterm birth rate was 11·4% (95% CI 6·5-18). There was no relationship between cortisol values and stress scale or depression. There was a significant positive relationship between maternal depression and stress. Preterm birth was associated with higher parity, past delivery of a male infant, and higher levels of paternal education
Paternal age and adverse birth outcomes: teenager	United States of America	Retrospective cohort	N = 2 614 966	Paternal age	Compared with infants born to fathers aged 20-29 years, infants fathered by teenagers (<20 years old) had an increased risk of preterm birth [odds ratio (OR) = 1.15,

Study Title, Author, Year	Study Area	Study Design	Sample Size: N	Paternal/Maternal Exposures	Results
or 40+, who is at risk? (Chen *et al.*, 2008)					95% confidence interval (CI): 1.10, 1.20], low birth weight (OR = 1.13, 95% CI: 1.08, 1.19), small-for-gestational-age births (OR = 1.17, 95% CI: 1.13, 1.22), low Apgar score (OR = 1.13, 95% CI: 1.01, 1.27), neonatal mortality (OR = 1.22, 95% CI: 1.01, 1.49) and post-neonatal mortality (OR = 1.41, 95% CI: 1.09, 1.82). Advanced paternal age (> or =40 years) was not associated with the risk of adverse birth outcomes
Paternal factors to the offspring birth weight: the 829 birth cohort study (Fan *et al.*, 2015)	China	Cohort study	N = 829	Socioeconomic and obstetric factors (BMI)	Fathers living in the rural area had offspring with higher risk of low birth weight when compared with fathers who live in the capital city. Maternal lower education, lower gestational weight gain, being primipara and shorter gestational age were risk factors for low birth weight. In addition, Mothers with the history of chronic disease had higher risk to deliver a low birth weight baby. On the contrary, women who increased non-staple food consumption during pregnancy had higher risk to have a macrocosmic pregnancy. However, lifestyle factors including diet, exercise, screen time, drinking and smoking from both maternal and paternal exhibited little influence on foetal birth weight
Offspring Birth Weight and Cardiovascular Risk in Parents—A Population-based HUNT 2 Study (Myklestad *et al.*, 2012)	Norway	Population based study	N = 14079 pairs	Parental BMI, waist circumference, blood pressure, glucose, and serum lipids	Low birth weight was associated with maternal (odds ratio (OR) = 1.9, 95% confidence interval (CI): 1.5, 2) and paternal (OR = 1.2, 95% CI: 1.0, 1.6) smoking, but the paternal association was fully attenuated after adjustment for maternal smoking (OR = 1.0, 95% CI: 0.8, 1.3)

Study Title, Author, Year	Study Area	Study Design	Sample Size: N	Paternal/Maternal Exposures	Results
Birth weight of offspring and paternal insulin resistance and paternal diabetes in late adulthood: cross sectional survey (Wannamethee *et al.*, 2004)	Britain	Cross-sectional survey	N = 4252	Paternal insulin resistance defined by the homeostasis model assessment (HOMA) score and with Type 2 diabetes in late adulthood	Fathers of offspring in the highest quartile of sex-standardized birth weight SD scores had a 34% reduction in odds of having a high HOMA insulin resistance score (OR=0.66, 95% CI: 0.47 to 0.92) compared with fathers of offspring in the lowest quartile after adjustment for potential confounders. A stronger inverse association was seen between offspring birth weight and risk of paternal diabetes (adjusted OR=0.59, 95% CI: 0.39 to 0.88 top quartile vs. lowest quartile). For each increase of offspring-birth weight SD score the odds of high HOMA scores decreased by 13% (OR=0.87, 95% CI: 0.78 to 0.98) and the odds for diabetes by 17% (OR=0.83, 95% CI: 0.72 to 0.95), after full adjustment.
The Relevance of Maternal Socioeconomic Characteristics for Low Birth Weight – a Case-Control Study (Altenhöner, Köhler and Philippi, 2016)	Germany	A case-control study	N = 454 (131 cases and 323 mothers)	Maternal socioeconomic status, health behaviour and stress in the workplace	Independent of medical diagnosis and health behaviour, women with lower level education (OR [95% CI] = 2.24 [1.12; 4.51]) and those who were not working (OR [95% CI] = 1.82 [1.10; 3.00]) were more likely to have an LBW baby. No effect was shown for immigrant background (OR [95% CI] = 1.14 [0.59; 2.21]) or stress in the workplace (OR [95% CI] = 1.17 [0.90; 1.51])

Study Title, Author, Year	Study Area	Study Design	Sample Size: N	Paternal/Maternal Exposures	Results
Paternal contribution to birth weight (Magnus *et al.*, 2001)	Norway	A family design, using trios of father-mother-firstborn child	N = 67 795 families	Paternal birth weight	The BW correlations were 0.126 for father-child. The RR of LBW for the first born are 8.2 if both parents were both LBWs infants as well. The estimated heritability for BW is 0.25
Differential parental weight and height contributions to offspring birthweight and weight gain in infancy (Griffiths, Dezateux and Cole, 2007)	United Kingdom	Cohort study	N = 6 811	Parental weight and height	Paternal height had less of an influence compared to maternal height – coefficient for the difference between parents is as follows: ((95% CI): 0.15 (0.100.20). The maternal and paternal weight and height equally contributed to infant weight gain [difference coefficients -0.03 (-0.09 to 0.02) and 0.02 (-0.03 to 0.07), respectively]
Maternal characteristics influencing birth weight and infant weight gain in the first 6 weeks post-partum: A cross-sectional study of a post-natal clinic population (Yilgwan, Utoo and	Nigeria	A cross-sectional descriptive study	N = 318	Maternal weight, age, socio-economic status	Mothers from the Northern side of Nigerian had multiparity and systolic and/or diastolic hypertension factors that were associated with low birth weight. Similarly, 37.0% of infants born to mothers with some tertiary education showed slow weight gain compared with those who had secondary (19.2%) or primary (14.7%) education, *P*=0.03. Maternal weight at delivery positively correlated with birth weight of the infant (r=0.357, *P*<0.001)

Study Title, Author, Year	Study Area	Study Design	Sample Size: N	Paternal/Maternal Exposures	Results
Hyacinth, 2012)					
Fathers Count: The Impact of Paternal Risk Factors on Birth Outcomes (Meng and Groth, 2018)	United States	Retrospective cross-sectional analysis	N = 36 731	Paternal education, race, ethnicity and age	Paternal race/ethnicity was statistically significant with PTB, LBW, SGA and HBW. Older paternal age was associated with increased odds (OR 1.012, 95% CI 1.003–1.022) of LBW. Less paternal education was significantly related to an elevated risk of PTB, LBW, and SGA
Maternal Height and Child Growth Patterns (Addo *et al.*, 2013)	Brazil, Guatemala, India, the Philippines, and South Africa	Pooled analysis of maternal height and offspring growth	N = 7 630	Parental height, household income, child sex, birth order and study site	In unadjusted analyses, younger (≤19 years) and older (≥35 years) maternal age was associated with lower birth weight and gestational age. After adjustment, associations with younger maternal age remained for low birth weight (odds ratio [OR] 1·18 (95% CI 1·02-1·36)], preterm birth (1·26 [1·03-1·53]), 2-year stunting (1·46 [1·25-1·70])
Evidence of genetic regulation of foetal longitudinal growth **(Knight et al., 2005)**	United Kingdom	A prospective cohort study	N = 567	Paternal height, weight and biochemistry factors	Paternal height was correlated with birth weight (r = 0.19) independent of maternal factors. Maternal height showed similar correlations with birth weight (r = 0.18). Additionally, maternal BMI was correlated with birth weight (r = 0.27)

The table 1 presents a summary of the literature reviewed regarding the influence that parental factors have on BW. Studies show a higher association between maternal factors and BW, compared to paternal factors. Literature suggests that paternal height, weight, low SES, race/ethnicity, diabetes and father's living in rural areas had a significant association with the infants BW. Occupational exposure may have an association with BW; however more research needs to be conducted on this variable. Maternal factors that had significant associations include weight, height, BMI, age, low levels of education, unemployment, stress, depression and chronic diseases (Table 1).

b. Birth length

i. Public health significance of birth length

The following sections summarize the literature on the public health significance of paternal and maternal factors on BL. An offspring's BL is of great importance as there appears to be growing evidence of the increased connections between slow growth in height early in life and impaired health and education performance later on in life (Dewey and Begum, 2011). Research has shown a negative association between childhood stunting and cognitive development and risk of NCDs in later life. These adverse health issues related to poor cognitive development include impaired brain development, lower IQ, weakened immune system diabetes and cancer (Woldehanna, Behrman and Araya, 2017).

ii. Associations between paternal and maternal factors and BL

Table 2: Summary of the literature review – paternal and maternal factors related to BL

Study Title, Author, Year	Study Area	Study Design	Sample Size: N	Paternal/Maternal Exposures	Results
Paternal Metabolic and Cardiovascular Risk Factors for Foetal Growth Restriction (Hillman, Peebles and Williams, 2013)	United Kingdom	A case-control study	N = 119	Paternal BMI, BP, waist circumference, endothelial function (flow-mediated dilatation), lipid profile, weight, and smoking habit.	Fathers of foetal growth–restricted offspring [mean (SD) 1.8th (2.2) customized birth centile] were more likely to have insulin resistance, hypertension, central adiposity, and smoked cigarettes compared with fathers of normal grown offspring. After multivariable analysis, paternal insulin resistance and smoking remained different between the groups. Compared with fathers of normal grown offspring, men who fathered pregnancies affected by foetal growth restriction had an OR 7.68 (95% CI 2.63–22.40; P < 0.0001) of having a 1-unit higher log HOMA-IR value and 3.39 (1.26–9.16; P = 0.016) of being a smoker
Paternal Metabolic and Cardiovascular Risk Factors for Foetal Growth Restriction **A case-control study** (Hillman, Peebles and Williams, 2013)	London	Case-control study	N = 119 (42 cases and 77 controls)	All fathers had measures of insulin resistance (HOMA index), blood pressure, waist circumference, endothelial function (flow-mediated dilatation), lipid profile, weight, and smoking habits	Compared with fathers of normal grown offspring, men who fathered pregnancies affected by foetal growth restriction had an OR 7.68 (95% CI 2.63–22.40; P < 0.0001) of having a 1-unit higher log HOMA-IR value and 3.39 (1.26–9.16; P = 0.016) of being a smoker

Study Title, Author, Year	Study Area	Study Design	Sample Size: N	Paternal/Maternal Exposures	Results
Maternal and paternal height and BMI and patterns of foetal growth: The Pune Maternal Nutrition Study (Wills *et al.*, 2010)	India	Community based prospective study	N = 2 675	Parental weight, height and socio-economic status	There was a positive association between paternal height and femur length
Gestational age and newborn size according to parental social mobility: an intergenerational cohort study (Gigante *et al.*, 2015)	Brazil	Two population-based birth cohort studies	N = 266	Parental socio-economic status, weight, height and gestation age	Amid mothers, childhood poverty was strongly associated with smaller birth size (about 400 g in weight and 1.5 cm in height) and shorter gestations (about 2 weeks)
Maternal Height and Child Growth Patterns (Addo *et al.*, 2013)	Brazil, Guatemala, India, the Philippines, and South Africa	Pooled analysis of maternal height and offspring growth	N = 7 630	Parental height, household income, child sex, birth order and study site	There was an association between maternal height with birth weight and length. Short mothers (<150.1 cm) were more likely to have a child who was stunted at 2 years (prevalence ratio = 3.20 (95% CI: 2.80-3.60) and as an adult (prevalence ratio = 4.74, (95% CI: 4.13-5.44)
Relationships of maternal and paternal anthropometry with neonatal body size, proportions and adiposity in an Australian cohort (Pomeroy *et al.*, 2015)	Australia	Analysis of neonatal and parental anthropometry from the Mater-University of Queensland Study of Pregnancy (MUSP) dataset	N = 1041	Parental height and weight	Paternal height may relate to neonatal limb length as a means of increasing foetal growth without exacerbating the risk of obstetric complications

Study Title, Author, Year	Study Area	Study Design	Sample Size: N	Paternal/Maternal Exposures	Results
Evidence of genetic regulation of foetal longitudinal growth **(Knight et al., 2005)**	United Kingdom	A prospective cohort study	N = 567	Paternal height, weight and biochemistry factors	Paternal height was correlated with birth length (r = 0.33) independent of maternal height. Maternal height showed similar associations (r = 0.26). Maternal BMI was also correlated with birth length (r = 0.15)

Table 2 summarizes the literature reviewed regarding the influence that parental factors have on BL. Studies show a higher association between paternal factors and BL, compared to maternal factors. Literature suggests that paternal height, insulin resistance, hypertension and central adiposity had a significant association with the infants BL. There was a very strong association found between paternal smoking and BL in various studies listed in the table above. Maternal factors that had significant associations include height, BMI and childhood poverty. The same table reflects inconsistent literature on the various factors (both paternal and maternal) and their contribution to BL.

c. Head circumference

i. Public health significance of head circumference

The following sections summarize the literature on public health significance of paternal and maternal factors on HC. HC is a significant birth indictor that measures infant growth and development as it monitors brain volume and is a key predictor of cognitive and intelligence development of a child (Sutan *et al.*, 2018). HC is a key predictor of microcephaly – a disproportionately small HC (below the 5^{th} centile in children) for a given GA, which results in an under developed brain (McElrath *et al.*, 2010). Due to the small and under developed brain, microcephaly has a multitude of adverse cognitive conditions that depend on its severity. The medical conditions include dwarfism, mental retardation, developmental delays in speech and movement, facial distortions and difficulties with balance and coordination (Chiriboga *et al.*, 2003).

Associations between paternal and maternal factors and HC

Table 3: Summary of the literature review – paternal and maternal factors related to HC

Study Title, Author, Year	Study Area	Study Design	Sample Size: N	Paternal/Maternal Exposures	Results
Head Circumference of Infants Born to Mothers with Different Educational Levels; The Generation R Study (Bouthoorn *et al.*, 2012)	Netherlands	A population-based prospective cohort study	N = 3383	Maternal education levels	During the first month of birth, children of mothers with a low education had a smaller HC than those with a high education (20.42 SD; 95% CI: 20.54, 20.30)
Relationships of maternal and paternal anthropometry with neonatal body size, proportions and adiposity in an Australian cohort (Pomeroy *et al.*, 2015)	Australia	Analysis of neonatal and parental anthropometry from the Mater-University of Queensland Study of Pregnancy (MUSP) dataset	N = 1041	Parental height and weight	Results suggest that the HC and limb trunk length of an infant is positively associated by maternal anthropometry factors such as height and BMI
Maternal and paternal height and BMI and patterns of foetal growth: The Pune Maternal Nutrition Study (Wills *et al.*, 2010)	India	Community-based prospective study	N = 2 675	Parental weight, height and socio-economic status	There was an observed positive significance between parental height and head circumference. The paternal height associations were detected much earlier during gestation (17 – 29 weeks) compared to mothers. Mothers with a high BMI, had foetuses who have a smaller mean head circumference at 17 weeks

Study Title, Author, Year	Study Area	Study Design	Sample Size: N	Paternal/Maternal Exposures	Results
Evidence of genetic regulation of foetal longitudinal growth (Knight *et al.*, 2005)	United Kingdom	A prospective cohort study	N = 567	Paternal height, weight and biochemistry factors	Paternal height has an independent influence on size at birth, predominantly skeletal growth and head circumference
Maternal Anthropometric measurements, Pre-pregnancy Body Mass Index, and Foetal Growth Parameters - A Rural Experience (Tayade *et al.*, 2018)	India	A hospital based, cross-sectional, observational study	N = 500	Maternal age, weight and height	Positive correlation was found between pre- pregnancy BMI and head circumference
Low Preconception Body Mass Index Is Associated with Birth Outcome in a Prospective Cohort of Chinese Women (Ronnenberg *et al.*, 2003)	China	A prospective study	N = 575	Pre-pregnancy BMI	There was significant association between severe maternal underweight BMI and smaller head circumference

Table 3 contains a summary of the literature reviewed regarding the influence that parental factors have on HC. Studies show a higher association between maternal factors and HC, compared to paternal factors (Wills *et al.*, 2010; Pomeroy *et al.*, 2015; Tayade *et al.*, 2018). Literature suggests that there is a positive significant association between paternal height and HC (Knight *et al.*, 2005). There was a very strong and significant association found between maternal pre-pregnancy BMI and low levels of education and HC (Table 3). The same table reflects inconsistent literature on the various factors (both paternal and maternal) and their contribution to HC.

d. Ponderal index

i. Public health significance of Ponderal index

The following sections summarize the literature on the public health significance of paternal and maternal factors on PI. Birth parameters such as PI are important indicators of intrauterine environment and prenatal nutritional status. Additionally, compared to BMI, PI is thought to provide a better gauge of weight in relation to height and body composition (Brutsaert et al. 2011). When an infant is born and has a low PI, they are likely to be malnourished whereas an infant that has a high PI is more likely to be obese (diagnosed with foetal macrosomia) (Roje et al. 2004). The long-term health implications related to PI include obesity, decreased muscle strength, coronary heart disease, hypertension and diabetes (Akram & Arif 2005).

ii. Associations between paternal and maternal factors and PI

Table 4: Summary of the literature review – paternal and maternal factors related to PI

Study Title, Author, Year	Study Area	Study Design	Sample Size: N	Paternal/Maternal Exposures	Results
Parental body size and early weight and height growth velocities in their offspring (Botton *et al.*, 2010)	Ireland	Population based study	N = 235 parent-child trios	Parental weight and height	Ponderal index at birth is significantly associated with maternal BMI and height
Small for Gestational Age, Ponderal Index and Neonatal Polycythemia: A study of their association with maternal hypertension among Nigerian women (Onyiriuka and Okolo, 2005)	Nigeria	A case control study	N = 1069	Maternal hypertension	SGA infants born to mothers with chronic hypertension had normal mean ponderal index (≥2.3) while their counterparts born to mothers with pregnancy-induced hypertension had low mean ponderal index (<2.3).
Low Preconception Body Mass Index Is Associated with Birth Outcome in a Prospective Cohort of Chinese Women (Ronnenberg *et al.*, 2003)	China	A prospective study	N = 575	Pre-pregnancy BMI	Being severely underweight was associated with lower ponderal index compared to a mother with a normal weight

Study Title, Author, Year	Study Area	Study Design	Sample Size: N	Paternal/Maternal Exposures	Results
Maternal Anthropometric measurements, Pre-pregnancy Body Mass Index, and Foetal Growth Parameters - A Rural Experience (Tayade *et al.*, 2018)	India	A hospital based, cross-sectional, observational study	N = 500	Maternal age, weight and height	There was a negative association between ponderal index and pre-pregnancy BMI

Table 4 summarizes the literature reviewed regarding the influence that parental factors have on PI. Based on the conducted literature review, the studies obtained only reported associations between maternal factors and PI. There significant associations between maternal BMI (underweight mothers), pre-pregnancy BMI, height, hypertension and PI. Further studies need to be conducted to assess the associations between paternal factors and PI (Table 4). The same table reflects inconsistent literature on the various factors (both paternal and maternal) and their contribution to PI.

i. Public health significance of gestational age

The following sections summarize the literature on the public health significance of paternal and maternal factors on GA

ii. Associations between paternal and maternal factors and GA

GA was not one of the measured birth outcomes in this current study, however it is an important covariate as it is directly associated with BW, BL, HC and PI. In this study, GA was the only consistently significant variable in all linear regression models, except for PI; but literature stresses the importance of GA in all birth outcomes including PI (Oluwafemi *et al.*, 2013). GA is of great importance as that is the time where the foetus grows and develops in the mother's uterine. Normal foetus development is vital as that reduces the risks of LBW, short BL, small HC and low PI. Optimal GA of delivery reduces pregnancy complications and neonatal mortality and morbidity (Bollen, Noble and Adair, 2013). Furthermore, it decreases the risk of an infant developing various NCDs and cognitive developmental disabilities in the later stages of an infant's life due to adverse birth outcomes. Both maternal and paternal factors play a crucial role in the intrauterine environment and genetic makeup of a foetus (Vlachadis, Tsamadias and Economou, 2013).

Figure 3 presents the plausible mechanisms that capture the trans-generational effect from mother to neonate that are known to exist for chronic conditions. Epigenetic modification of gene expression affects the development of key organs, which results to an increased risk of NCDs (i.e. diabetes, hypertension and cognitive ability) in later adulthood.

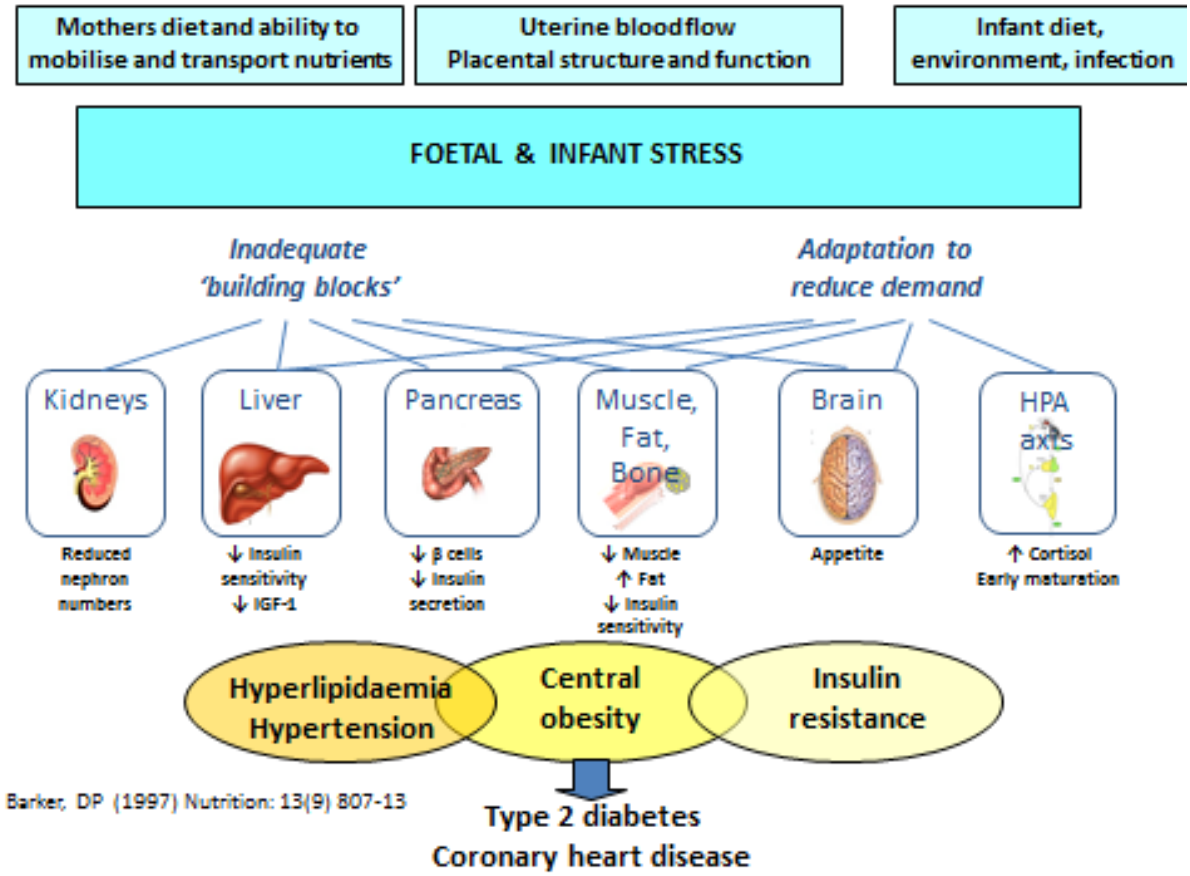

Figure 3: Plausible mechanisms for the trans-generational effect from mother to child that is known to exist for chronic conditions

2.3 Research gaps in literature

Existing literature mainly focuses on maternal factors and based on the literature review that was conducted, there appears to be a lack of robust epidemiological data that investigates the associations between paternal factors and birth outcomes – especially in South Africa. Of the 20 million LBW infants that are born globally more than 15% of them are born in the Sub-Saharan African Region (World Health Organization, 2014). However, the reported prevalence is regarded as an underestimation due to the notion that the LICs – which happen to be the higher risk groups- are more likely to be underreported as they are least likely to be included in hospital or urban-based data (Sharma *et al.*, 2015). The novelty of this this research emanates from the excessive burden of neonatal deaths and atypical birth outcomes in LMICs like South Africa. Therefore, this study will not only add towards the much-

needed literature on paternal determinants of birth outcomes; but also inform primary interventions that are tailor-made to alleviate the factors that place children at risk of poor birth outcomes and policies that are pro-paternal wellbeing and health in addition to maternal health.

CHAPTER 3: METHODOLOGY

3.1 Introduction

The methodology section is presented by objectives as articulated in Chapter 1. Under each objective, the following aspects are comprehensively covered where applicable:

(i) Study design, setting and population, including inclusion and exclusion criteria
(ii) Data collection
(iii) Measures
(iv) Informed consent
(v) Ethics clearance
(vi) Statistical analysis

3.2 Study design, Setting and Study population

The current study is cross-sectional in nature utilising secondary data. Data were primarily collected by the University of Witwatersrand (Wits) Developmental Pathways for Health Research Unit (DPHRU) under the following two major projects conducted in Soweto, Chris Hani Baragwanath hospital, in South Africa from 2013-2015 (i) the S1000 study and (ii) Father of baby study. For the purposes of the current objectives presented in the dissertation, a secondary database was created from the two projects mentioned above. Comprehensive details of the two projects including their (i) primary aims, design and methods, data collection procedures, and primary outcomes are explained elsewhere (Wrottesley, Lamper and Pisa, 2016). The following selection criterion was set for the current study:

Inclusion Criteria

- Fathers should who are over the age of 18 years
- Pregnant females who are over the age 18 years
- Man's partner should be pregnant with singletons
- Naturally conceived pregnancy
- Women should be screened at foetal medicine
- Women should have at least one scan (from foetal medicines' unit)
- Women should be ≤ 20 weeks GA (entry to the study can occur at any point before 20 weeks GA)
- Any BMI

- Fathers should be from Soweto and Greater Soweto (Orange farm)
- Individuals being able to give consent

Exclusion Criteria

- Fathers who are younger than the age of 18 years
- Pregnant females who are younger than the age of 18 years,
- White, Indian, Coloured men and women
- Non-Singleton pregnancies (Twins, triplets etc.),
- Men on fertility treatment
- Pregnancies where foetal abnormalities have been detected
- Fathers residing in Lenasia & anywhere out of Soweto
- Fathers with Epilepsy & Type 1 diabetes and any other known chronic condition

3.3 Data collection

3.3.1Paternal and Maternal Factors

Dual-Energy X-ray Absorptiometry Scan

A full body scan taken utilizing the Dual-Energy X-ray Absorptiometry (DEXA) Scan (Hologic DiscoveryA S/N 86254) to derive body composition measures of fat and lean mass using standard procedures.

Father's Anthropometric Data

The father's anthropometric data was collected according to height (cm), weight (kg), waist and hip circumference (cm), pulse and blood pressure (BP). Pulse was classified as beats per minute and BP was recorded as systolic BP (mmHg) and diastolic BP (mmHg).

Father's Biochemical Measurement

Blood was drawn and stored for analysis on lipids, insulin, C - reactive protein (CRP), spare blood, glycated haemoglobin (HbA1c) and glucose levels from each father of the baby using standard procedures.

Father's Socioeconomic Status Questionnaire

The father of the baby answered questions about their social and economic status. The questionnaire has been modified to conform to the South African population and was based on standard measures used by Demographic and Health surveys (DHS). The measures included paternal education, occupation, smoking habits and the chronic history of the respondent, the respondent's parents and grandparents.

Mother's Confounding Factors

Maternal factors such as the mother's age, parity, number of children was considered as covariates and were collected and answered through a social demographic questionnaire.

3.3.2 Offspring Factors

The offspring's birth weight was measured at delivery using a digital scale to the nearest 0.1kg. GA was derived from the expected dates of confinement (EDC) which will be calculated on the basis of both early ultrasound and the date of the last menstrual period (Janssen *et al.*, 2007). The length of the baby was measured using a stadiometer, a hard plastic platform with a vertical headboard against which the crown of the baby's head will be placed. The measurement from the crown to the soles of the feet was taken using the centimetre scale on the stadiometer.

3.4 Informed Consent

The study was explained to eligible participants and they were asked to give written informed consent. All study subjects that withdrew midway through the research had their data disregarded in the final analysis stage. All participants were requested for a written consent that highlighted their voluntary participation.

3.5 Ethics Clearance

This protocol was submitted to the University of the Witwatersrand Human Research Ethics Committee (Medical) for ethical clearance before commencing the study and ethical clearance was obtained (Clearance certificate number: M161189). For

confidentiality and anonymity purposes, dataset for this study was granted to the student without identifiers.

3.6 Statistical analysis

Differences in the study populations infants' characteristics were compared by sex, using T independent and Pearson chi squared tests for continuous and categorical variables, respectively. Descriptive statistics were calculated to summarise further summarise paternal and maternal characteristics. Data are presented as mean (SD) and percentages (%) for continuous and categorical data, respectively. Bivariate associations between paternal and maternal factors with the following infant birth outcomes birth length, weight, head circumference and ponderal index were computed.

Furthermore, to explore independent and adjusted associations of paternal and maternal factors with birth outcomes, multivariate linear regression models were computed. Two crude models associating paternal and maternal factors with all above mentioned birth outcomes without adjusting for any covariates (Model 1 and Model 3) are presented. Additionally, M1 and M3 are adjusted models for neonatal factors (GA and sex of infant at birth) and are as presented as M2 and M4. A full model that includes paternal, maternal and neonatal factors as independent factors, and all birth outs as dependent variables, are reflected as M5. In the M5 the following variables are specifically included:

- **Paternal factors**: Age, BMI, WC, total number of education (years), employment status, smoking habits and living with partner status
- **Maternal factors:** Age, BMI, education, HIV status, GDM status and marital status
- **Neonatal factors:** GA and sex at birth

Regression coefficients and adjusted beta values (and their 95% CIs) are presented. Adjusted partial R^2 values were computed to express the proportion variance explained by each model with infant birth outcomes. The statistical package STATA version 13 was used for analysis. All statistical significance was defined using a 2 tailed P value <0.05.

CHAPTER 4: RESULTS

4.1 Introduction

The results section presents tables and narratives for the set objectives of the current study. Table 5 compares the demographic, anthropometric and other selected characteristics of infants at birth by sex. No significant differences were observed by sex for GA, birth weight, head circumference and ponderal index among infants. However, the results displayed that males were 1.7cm significantly taller than the females. Significant differences were observed by sex, for preterm/term status as well as mode of delivery. However, only 11.7% of the total sample reported as preterm deliveries and 45% were vaginal deliveries. A total of 79.1% of the infants had the recommended GA, whilst 13% were defined as SGA. As for BW, 13.9% were lower than 2,5kg.

Table 5: ***Demographic, anthropometric and other selected characteristics of infants at birth by sex*** **(N=120)**

Variables		**Male (N= 72)**	**Female (N = 48)**	
	Mean (SD)/%	**Mean (SD)/%**	**Mean (SD)/%**	**p value**
Gestational Age at Delivery (wks.)	38.2 (2.3)	38.3 (2.2)	38.1 (2.3)	0.611
Birth Weight (g)	3062.4 (507.1)	3109.8 (466.3)	2991.3 (560.6)	0.222
Birth Length (cm)	48.5 (3.8)	49.2 (3.8)	47.5 (3.6)	**0.017**
Birth Head Circumference (cm)	34.2 (1.7)	34.5 (1.5)	33.8 (2.1)	0.066
Ponderal Index(kg/m3)	2.7 (0.4)	2.6 (0.5)	2.8 (0.3)	0.094
Weight to Length Ratio (kgm)	6.3 (0.8)	6.3 (0.7)	6.6 (0.9)	0.839
Fat Mass (g)	438.1 (194.7)	403.5 (166.6)	509.3 (232.5)	0.074
Preterm delivery (<37 weeks gestation)	120	72	48	**0.054**
Yes	14 (11.7%)	8 (11.1%)	6 (12.5%)	
No	106 (88.3%)	64 (88.9%)	42 (87.5%)	
Mode of Delivery (collapsed)	120	72	48	**0.022**
Vaginal delivery	54 (45.0%)	32 (44.4%)	22 (45.8%)	
Caesarean section	66 (55.0%)	40 (55.6%)	26 (54.2%)	
Birth size categories by weight (based on Intergrowth 21st calculations)	115	69	46	1.772
Small for gestational age (SGA) (<10th centile)	15 (13.0%)	10 (14.5%)	5 (10.9%)	
Appropriate for gestational age (APA)	91 (79.1%)	52 (75.4%)	39 (84.8%)	
Large for gestational age (LGA) (>90th centile)	9 (7.8%)	7 (10.1%)	2 (4.3%)	
Low Birth Weight (g)	115	69	46	0.774
Yes	16 (13.9%)	8 (11.6%)	8 (17.4%)	
No	99 (86.1%)	61 (88.4%)	38 (82.6%)	

Table 6 – 7 describes the paternal and maternal characteristics. The fathers in the study had a mean age of 32.5 (6.7) years, BMI of 23.7 (4.8), WC of 82.3 (11.2). Of

the father's sample, 33.5% were considered overweight and obese combined. About 99% fathers reported having a secondary and tertiary education whilst 77.9% of those fathers were employed. Approximately 69.3% fathers were living with their partners and approximately 46% of the study participants were current smokers. In table 7, the mothers in the study had a mean age of 28.8 (5.9) and BMI 27.2 (6.4). Of the mother's sample, 63.4% were considered overweight and obese. About 98% mothers reported having secondary and tertiary education whilst 46% are married or cohabitating with their partners. Approximately 26.8% and 13% of the mothers were HIV positive and had GDM respectively.

Table 6: Demographic, anthropometric and other selected characteristics of paternal factors (N = 164)

Variables	**Total**
Age (yrs.) [Mean (SD)]	32.5 (6.7)
Height (cm) [Mean (SD)]	172.4 (6.2)
Weight (cm) [Mean (SD)]	70.4 (14.6)
BMI (Kg/m2) [Mean (SD)]	23.7 (4.8)
Waist Circumference (cm) [Mean (SD)]	82.3 (11.2)
Total Number of Education (yrs.) [Mean (SD)]	12.6 (2.6)
Diastolic blood pressure [Mean (SD)]	79.7 (10.9)
Systolic blood pressure [Mean (SD)]	123.6 (16.0)
Total body fat [Mean (SD)]	14720.1 (8631.4)
Total % body fat [Mean (SD)]	21.9 (8.1)
Total body mass [Mean (SD)]	63338.4 (13929.3)
Whole body total mass [Mean (SD)]	68389.7 (14251.9)
Total body lean [Mean (SD)]	48618.3 (6830.8)
Whole body total lean [Mean (SD)]	52595.9 (7088.7)
Visceral fat cm2 [Mean (SD)]	5.8 (2.1)
Subcutaneous fat cm2 [Mean (SD)]	1.2 (0.6)
BMI	164
Underweight n (%)	17 (10.4%)
Normal (≤24.9 kg/m2) n (%)	92 (56.1%)
Overweight (25-29.9kg/m2) n (%)	35 (21.3%)
Obese (≥ 30 kg/m2) n (%)	20 (12.2%)
Highest Level of Education	163
Primary n (%)	2 (1.2%)
Secondary n (%)	83 (50.9%)
Tertiary and post qualification n (%)	78 (47.9%)
Employment Status	163
No n (%)	36 (22.1%)
Yes n (%)	127 (77.9%)
Living with Partner	163
No n (%)	50 (30.7%)

Yes n (%)	113 (69.3%)
Smoking Habits	162
Never smoked n (%)	65 (40.1%)
Used to smoke but stopped n (%)	23 (14.2%)
Current smoker n (%)	74 (45.7%)

Table 7: Demographic, anthropometric and other selected characteristics of maternal factors (N = 168)	
Variables	Total
Age (yrs.) [Mean (SD)]	28.8 (5.9)
Weight (cm) [Mean (SD)]	68.9 (15.7)
Height (cm) [Mean (SD)]	159.2 (8.0)
BMI (Kg/m2) [Mean (SD)]	27.2 (6.4)
BMI category at enrolment	123
Normal (≤24.9 kg/m2) n (%)	45 (36.6%)
Overweight (25-29.9kg/m2) n (%)	49 (39.8%)
Obese (≥ 30 kg/m2) n (%)	29 (23.6%)
Marital Status (categories)	123
Single n (%)	67 (54.5%)
Married/Cohabiting n (%)	56 (45.5%)
Highest Level of Education	123
Primary n (%)	2 (1.6%)
Secondary n (%)	82 (66.7%)
Tertiary n (%)	39 (31.7%)
HIV status at enrolment	123
Negative n (%)	90 (73.2%)
Positive n (%)	33 (26.8%)
Gestational Diabetes Status	123
Non-GDM n (%)	107 (87.0%)
GDM n (%)	16 (13.0%)

Table 8: Bivariate associations between neonatal factors paternal, maternal variables with selected birth outcomes

Birth Outcomes	Birth Weight (g)			Birth Length (cm)			Head Circumference (cm)			Ponderal Index		
Variables	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p-value**	**β**	**95%CI**	**p value**
NEONATAL FACTORS												
Sex												
Male	Reference											
Female	-118.39	-309.20;72.42	0.222	-1.70	-3.09; -0.31	**0.017**	-0.61	-1.26;0.04	0.066	0.14	-0.03;0.31	0.094
GA (weeks)	148.56	113.57;183.55	**0.001**	1.003	0.73;1.28	**0.001**	0.44	0.31;0.57	**0.001**	-0.03	-0.06;0.01	0.211
Preterm status												
Term	Reference											
Preterm	-742.39	-1017.35; -467.44	**0.001**	-5.08	-7.16; -3.01	**0.001**	-2.20	-3.17; -1.23	**0.001**	0.12	-0.15;0.39	0.388
PATERNAL FACTORS												
Age (yrs.)	2.91	-11.54;17.36	0.691	-0.02	-0.12;0.09	0.789	0.01	-0.04;0.06	0.647	0.00	-0.01;0.02	0.648
Height (cm)	13.47	-0.60;27.55	0.060	0.02	-0.09;0.13	0.723	0.04	-0.01;0.09	0.111	0.01	-0.01;0.02	0.352
Weight (g)	2.24	-3.86;8.34	0.468	0.00	-0.04;0.05	0.948	-0.01	-0.03;0.01	0.544	0.00	0.00;0.01	0.593
Waist Circumference (cm)	4.41	-3.68;12.50	0.283	0.01	-0.05;0.08	0.641	0.00	-0.03;0.03	0.853	0.00	-0.01;0.01	0.697
BMI	2.11	-16.77;20.99	0.825	0.00	-0.12;0.14	0.954	-0.04	-0.10;0.28	0.268	0.00	-0.02;0.02	0.841
Total Number of Education (yrs.)	5.87	-28.87;40.62	0.738	0.03	-0.23;0.29	0.831	0.01	-1.11;0.13	0.865	0.00	-0.03;0.03	0.857
Employment Status												
Employed	Reference											
Not Employed	89.57	-142.47;321.61	0.446	-0.01	-1.73;1.71	0.990	0.09	-0.71;0.89	0.823	0.10	-0.10;0.30	0.327
Living with Partner												
Not married (No)	Reference											
Married (Yes)	157.90	-42.51;358.30	0.121	-0.05	-1.55;1.45	0.949	0.28	-0.41;0.98	0.418	0.18	0.00;0.35	**0.050**
Smoking Habits												
Never (ref)	Reference											
Quit	125.44	-146.31;397.19	0.362	0.90	-1.14;2.94	0.385	-0.63;1.27	-0.63;1.27	0.505	-0.04	-0.28;0.21	0.773
Current	-164.83	-366.39;36.72	0.108	-0.80	-2.32;0.72	0.298	-0.82;0.60	-0.82;0.60	0.756	0.28	-0.15;0.21	0.758
Average Systolic	1.60	-4.24;7.43	0.589	0.00	-0.04;0.05	0.907	0.00	-0.02;0.02	0.675	0.00	0.00;0.01	0.778
Average Diastolic	1.75	-7.20; 10.69	0.700	0.01	-0.06;0.08	0.736	-0.01	-0.04;0.02	0.500	0.00	-0.01;0.01	0.905
Medial (visceral) fat	20.83	-27.25;68.90	0.392	0.05	-0.31;0.42	0.780	-0.04	-0.20;0.13	0.668	0.01	-0.03;0.05	0.673
Subcutaneous fat	13.73	-144.41;171.86	0.864	0.04	1.14;1.22	0.948	-0.12	-0.66;0.42	0.665	0.02	-0.12;0.16	0.775
Total Fat	0.00	-0.01;0.01	0.742	0.00	0.00;0.00	0.706	0.00	0.00;0.00	0.153	0.00	0.00;90.00	0.849

Table 8: Bivariate associations between neonatal factors paternal, maternal variables with selected birth outcomes

Birth Outcomes	Birth Weight (g)			Birth Length (cm)			Head Circumference (cm)			Ponderal Index		
Variables	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p-value**	**β**	**95%CI**	**p value**
Total Lean	0.00	-0.01;0.02	0.601	0.00	0.00;0.00	0.789	0.00	0.00;0.00	0.366	0.00	0.00;0.00	0.540
Total fat percentage	-2.58	-14.36;9.20	0.665	-0.02	-0.11;0.70	0.697	-0.03	-0.68;0.01	0.184	0.00	-0.01;0.01	0.828
Total Mass	0.00	-0.00;0.01	0.966	0.00	0.00;0.00	0.717	0.00	-0.00;0.00	0.187	0.00	0.00;0.00	0.682
Whole body total mass	0.00	-0.01;0.01	0.735	0.00	0.00;0.00	**0.040**	0.00	0.00;0.00	0.152	0.00	0.00;0.00	0.843
Whole body total lean	0.00	-0.01;0.02	0.602	0.00	0.00;0.00	0.784	0.00	0.00;0.00	0.377	0.00	0.00;0.00	0.538
Whole body total mass percentage	-2.87	-15.46;9.72	0.652	-0.02	-0.11;0.08	0.689	-0.03	-0.07;0.01	0.178	0.00	-0.01;0.01	0.828
MATERNAL FACTORS												
Age (yrs.)	-15.07	-30.94;0.79	0.062	-0.13	-0.24; -0.01	**0.036**	-0.04	-0.10;0.01	0.119	0.01	-0.01;0.02	0.351
Weight (cm)	10.07	4.44;15.70	**0.001**	0.02	-0.03;0.07	0.411	0.01	-0.00;0.03	0.213	0.01	0.00;0.01	**0.054**
Height (cm)	2.81	-8.77;14.40	0.631	-0.01	-0.10;0.08	0.838	0.01	-0.03;0.04	0.801	0.00	-0.01;0.01	0.501
BMI (Kg/m2)	21.51	7.54;35.49	**0.003**	0.05	-0.07;0.16	0.413	0.03	-0.03;0.08	0.312	0.01	0.00;0.02	0.125
Level of Education												
Tertiary	Reference											
Primary	-340.92	-1062.08;380.24	0.351	-0.36	-5.74;5.02	0.894	0.40	-2.10;2.91	0.751	-0.22	-0.86;0.43	0.503
Secondary	-203.27	-401.21; -5.33	**0.044**	-1.76	-2.91;0.17	0.082	-0.31	-1.00;0.39	0.381	0.08	0.10;0.26	0.371
HIV status at enrolment												
Negative	Reference											
Positive	-93.63	-304.98;117.71	0.382	-0.46	-2.03;1.12	0.566	-0.06	-0.78;0.67	0.880	-0.02	-0.20;0.17	0.859
Gestational Diabetes Status												
Non-GDM												
GDM	-6.81	-278.71;265.09	0.961	-0.43	-2.50;1.64	0.683	0.05	-0.90;1.01	0.915	0.00	-0.24;0.25	0.968
Marital status												
Single	Reference											
Married	114.81	-72.76;302.38	0.228	-0.07	-1.47;1.34	0.924	0.16	-0.49;0.81	0.619	0.13	-0.03;0.30	0.110

Table 8 presents bivariate associations between neonate, paternal, maternal variables with selected birth outcomes. Neonatal factors specifically GA and preterm birth were respectively associated with all birth outcomes except for PI. As for sex at birth, all associations were not significant except for BL. As for paternal factors, living with partner and body composition (WBTF) significantly associated with PI and BL respectively.

With regards to the mother's anthropometric variables, maternal weight and BMI had a significant association with the BW of the infant. Maternal weight also showed a positive association with the infant's PI. Significant associations were observed between maternal age and the infant's birth length. Maternal education was significantly associated with BL.

Table 9 - 12 presents adjusted models for multiple linear regressions between paternal and maternal factors with birth length, BW, BL, HC and PI. In table 9, the full model represents 51.9% of the variance in BW. However, the key drivers to this model were neonatal factors (34.7%) and maternal factors (15%). Paternal factors had the lowest contribution of 11.6%. GA and maternal BMI were the only consistent significant predictors in birth weight in the models where they were included. GA findings were as follows (M2: β: 141.05; 95%CI: 103.96; 178.14; p = 0.001), (M4: β: 141.44; 95%CI: 107.61; 175.26; p = 0.001), (M5: β: 132.54; 95%CI: 95.08; 170.0; p = 0.001). The following are the results for maternal BMI: (M3: β: 21.66; 95%CI: 7.71; 35.61; p = 0.003), (M4: β: 22.49; 95%CI: 11.54; 33.43; p = 0.001), (M5: β: 21.43; 95%CI: 9.39; 33.48; p = 0.001). Additionally, there was an observed significant association in M3 between maternal age and birth weight (β: -17.10; 95%CI: -33.96; -0.23; p = 0.003). In model 2, fathers who live with their partners and paternal WC were also a significant predictor in birth weight: (β: 194.00; 95%CI: 14.71; 373.26; p = 0.034), (β: 19.32; 95%CI: 2.21; 36.42; p = 0.027), respectively.

In table 10, the full model (including paternal, maternal and neonatal factors) represents 38.5% of the variance in BL. However, the key drivers were neonatal factors (31.7%) and maternal factors (5.7%). Paternal factors had the lowest variance of 4.2%. In all models, GA and sex at birth were consistently significant predictors of BL in the models they were included: GA: [(M2: β: 0.98; 95%CI: 0.69;1.27; p = 0.001), (M4: β: 0.93; 95%CI: 0.65;1.21; p = 0.001), (M5: β: 0.91;

95%CI: 0.06;1.23; p = 0.001)] and sex at birth: [(M2: β: -1.36; 95%CI: -2.59;-0,13; p = 0.031), (M4: β: -1.46; 95%CI: -266; -0.26; p = 0.018), (M5: β: -1.52; 95%CI: -2.80; -0.23; p = 0.021)].

In table 11, the full model represents 32.6% of the variance in HC. However, the key drivers to this are neonatal factors (29.1%) and paternal factors (5.5%). Unlike the previous association with BW and BL, maternal factors had the lowest contribution of about 4.2%. Paternal BMI was significantly associated with HC (β: -0.15; 95%CI: -0.28;-0.01; p = 0.042), however the association diminished once neonatal and maternal factors were added to the models GA was a consistent significant predictor in birth HC with the following results: (M2: β: 0.44; 95%CI: 0.30; 0.58; p = 0.001), (M4: β: 0.43; 95%CI: 0.29; 0.56; p = 0.001), (M5: β: 0.42; 95%CI: 0.27; 0.56; p = 0.001).

In table 12, the full model represents 10.1% of the variance in PI. However, the key drivers in this model were maternal factors (4.8%) and paternal factors (4.2%), unlike in the previous outcomes in which neonatal factors had the highest contribution. In all models, no significant predictors were observed for PI.

Table 9: Multivariate linear regressions between paternal and maternal factors and birth weight

	Model 1 (Paternal Factors)			Model 2 (Model 1 + Neonatal factors)			Model 3 (Maternal Factors)			Model 4 (Model 3 + Neonatal factors)			Model 5 (Paternal + Maternal + Neonatal factors)		
Variables	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**
PATERNAL FACTORS															
Age (yrs.)	-9.77	-27.24;7.70	0.270	-5.60	-19.78;8.57	0.435							1.77	15.69;19.23	0.841
BMI	-41.00	-79.77; -2.30	**0.038**	-13.93	-46.28;18.42	0.395							-9.74	-41.23;21.75	0.541
WC	19.32	2.21;36.42	**0.027**	8.85	-5.27;22.97	0.217							3.66	-10.52;17.84	0.610
Total Number of Education (yrs.)	11.35	-24.76;47.45	0.535	15.35	-13.79;44.48	0.299							7.88	-20.87;36.63	0.588
Employment Status															
No (Ref)	Ref	Ref	Ref	Ref	Ref	Ref							Ref	Ref	Ref
Yes	61.56	-176.70;299.83	0.610	-8.46	-201.79;184.87	0.931							29.44	-161.10;219.98	0.760
Smoking Habits															
Never smoked (ref)	Ref	Ref	Ref	Ref	Ref	Ref							Ref	Ref	Ref
Used to smoke but stopped	181.48	-98.19;461.14	0.201	149.29	-76.81;375.38	0.193							159.65	-58.77;378.07	0.150
Current smoker	-175.33	-382.71;32.06	0.097	-32.73	-203.93;138.46	0.705							-31.47	-200.86;137.93	0.713
Living with Partner															
No (Ref)	Ref	Ref	Ref	Ref	Ref	Ref							Ref	Ref	Ref
Yes	199.65	-22.67;421.97	0.078	194.00	14.71;373.26	**0.034**							111.89	-92.45;316.23	0.280
R^2	0.17														
NEONATAL FACTORS															
Sex															
Male (ref)				Ref	Ref	Ref				Ref	Ref	Ref	Ref	Ref	Ref

Table 9: Multivariate linear regressions between paternal and maternal factors and birth weight

	Model 1 (Paternal Factors)			Model 2 (Model 1 + Neonatal factors)			Model 3 (Maternal Factors)			Model 4 (Model 3 + Neonatal factors)			Model 5 (Paternal + Maternal + Neonatal factors)		
Variables	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**
Female				-55.42	-211.95;101.11	0.484				-79.18	-224.39;66.02	0.282	-85.69	-239.54;68.16	0.272
GA (weeks)				141.05	103.96;178.14	**0.000**				141.44	107.61;175.26	**0.000**	132.54	95.08;170.0	**0.000**
R^2				0.44											
MATERNAL FACTORS															
Age (yrs.)							-17.10	-33.96; -0.23	**0.047**	-7.85	-21.15;5.45	0.245	-11.84	-30.60;6.93	0.214
BMI (Kg/m2)							21.66	7.71;35.61	**0.003**	22.49	11.54;33.43	**0.000**	21.43	9.39;33.48	**0.001**
Level of Education															
Tertiary (Ref)							Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Education up till Secondary School							134.99	-57.57;327.56	0.168	71.72	-78.72;222.17	0.347	52.31	-108.87;213.49	0.521
HIV status at enrolment															
Negative (Ref)							Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Positive							45.72	-169.53;260.98	0.675	36.50	131.01;204.01	0.667	12.22	-167.71;192.15	0.893
Gestational Diabetes Status															
Non-GDM (Ref)							Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
GDM							0.56	-266.30;267.42	0.997	-8.73	-217.58;200.13	0.934	-16.92	-240.83;207.0	0.881
Marital Status (categories)															
Married/Cohabiting (Ref)							Ref	Ref	Ref	Ref	Ref	Ref			
Single							147.75	-37.2;332.75	0.116	131.47	-12.94;275.87	0.074			
R^2							0.150			0.50			0.52		

Table 10: Multivariate linear regressions between paternal and maternal factors and birth length

	Model 1 (Paternal Factors)			Model 2 (Model 1 + Neonatal factors)			Model 3 (Maternal Factors)			Model 4 (Model 3 + Neonatal factors)			Model 5 (Paternal + Maternal + Neonatal factors)		
Variables	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**
PATERNAL FACTORS															
Age (yrs.)	-0.05	-0.19;0.08	0.427	-0.03	-0.14;0.08	0.574							0.02	-0.12;0.17	0.743
BMI	-0.17	-0.48;0.13	0.256	0.05	-0.21;0.31	0.694							0.06	-0.21;0.32	0.683
WC	0.09	-0.05;0.22	0.200	0.00	-0.11;0.12	0.958							0.00	-0.12;0.12	0.969
Total Number of Education (yrs.)	0.04	-0.24;0.32	0.777	0.06	-0.17;0.29	0.589							0.03	-0.21;0.27	0.806
Employment Status															
No (Ref)	Ref	Ref	Ref	Ref	Ref	Ref							Ref	Ref	Ref
Yes (Ref)	-0.04	-1.87;1.80	0.968	-0.46	-1.98;1.06	0.546							-0.34	-1.93;1.25	0.672
Smoking Habits															
Never smoked (ref)	Ref	Ref	Ref	Ref	Ref	Ref							Ref	Ref	Ref
Used to smoke but stopped	1.05	-1.11;3.21	0.337	0.92	-0.86;2.70	0.310							0.85	-0.98;2.69	0.357
Current smoker	-0.86	-2.47;0.75	0.292	0.17	-1.19;1.53	0.803							0.04	-1.38;1.47	0.954
Living with Partner															
No (Ref)	Ref	Ref	Ref	Ref	Ref	Ref							Ref	Ref	Ref
Yes (Ref)	0.25	-1.46; -1.96	0.775	0.24	-1.17;1.64	0.740							0.20	-1.51;1.91	0.813
R^2	0.04														
Sex															
NEONATAL FACTORS															
Male (ref)				Ref	Ref	Ref				Ref	Ref	Ref	Ref	Ref	Ref
Female				-1.36	-2.59; -0.13	**0.031**				-1.46	-2.66; -0.26	**0.018**	-1.52	-2.80; -0.23	**0.021**
GA (weeks)				0.98	0.69;1.27	**0.000**				0.93	0.65;1.21	**0.000**	0.91	0.06;1.23	**0.000**

Table 10: Multivariate linear regressions between paternal and maternal factors and birth length

	Model 1 (Paternal Factors)			Model 2 (Model 1 + Neonatal factors)			Model 3 (Maternal Factors)			Model 4 (Model 3 + Neonatal factors)			Model 5 (Paternal + Maternal + Neonatal factors)		
Variables	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**
R^2				0.37											
MATERNAL FACTORS															
Age (yrs.)							-0.12	-0.25;0.01	0.072	-0.06	-0.17;0.05	0.268	-0.09	-0.24;0.07	0.277
BMI (Kg/m2)							0.05	-0.06;0.17	0.354	0.07	-0.02;0.17	0.129	0.06	-0.04;0.17	0.227
Level of Education															
Tertiary (Ref)							Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Education up till Secondary School							0.75	-0.77;2.27	0.330	0.31	-0.94;1.56	0.625	0.16	-1.20;1.51	0.818
HIV status at enrolment															
Negative (Ref)							Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Positive							0.26	-1.42;1.94	0.760	0.15	-1.24;1.53	0.835	0.08	-1.43;1.58	0.920
Gestational Diabetes Status															
Non-GDM (Ref)							Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
GDM							0.08	-2.06;2.22	0.941	-0.07	-1.84;1.69	0.936	-0.14	-2.05;1.77	0.887
Marital Status (categories)															
Married/Cohabiting (Ref)							Ref	Ref	Ref	Ref	Ref	Ref			
Single							0.25	-1.20;1.69	0.737	0.06	-1.13;1.26	0.918			
R^2							0.06			0.37			0.39		

Table 11: Multivariate linear regressions between paternal and maternal factors and birth head circumference

	Model 1 (Paternal Factors)	Model 2 (Model 1 + Neonatal factors)	Model 3 (Maternal Factors)	Model 4 (Model 3 + Neonatal factors)	Model 5 (Paternal + Maternal + Neonatal factors)

Variables	β	95%CI	p value	β	95%CI	p value	β	95%CI	p value	β	95%CI	p value	β	95%CI	p value
PATERNAL FACTORS															
Age (yrs.)	0.01	-0.07;0.05	0.766	0.00	-0.05;0.05	0.936							0.02	-0.05;0.09	0.497
BMI	-0.14	-0.28; -0.01	**0.042**	0.05	-0.17;0.07	0.403							-0.06	-0.19;0.07	0.342
WC	0.05	-0.01;0.11	0.114	0.01	-0.04;0.07	0.599							0.02	-0.04;0.07	0.569
Total Number of Education (yrs.)	0.04	-0.08;0.17	0.496	0.06	-0.05;0.16	0.314							0.05	-0.06;0.16	0.389
Employment Status															
No (Ref)	Ref	Ref	Ref	Ref	Ref	Ref							Ref	Ref	Ref
Yes	0.17	-0.67;1.01	0.683	-0.03	-0.75;0.69	0.934							-0.01	-0.76;0.74	0.984
Smoking Habits															
Never smoked (ref)	Ref	Ref	Ref	Ref	Ref	Ref							Ref	Ref	Ref
Used to smoke but stopped	0.35	-0.65;1.34	0.490	0.27	-0.57;1.11	0.528							0.28	-0.58;1,.14	0.520
Current smoker	-0.23	-0.97;0.51	0.532	0.22	-0.42;0.86	0.496							0.23	-0.45;0.90	0.507
Living with Partner															
No (Ref)	Ref	Ref	Ref	Ref	Ref	Ref							Ref	Ref	Ref
Yes	0.37	-0.42;1.15	0.360	0.35	-0.31;1.02	0.295							0.25	-0.55;1.06	0.535
R^2	0.06														
NEONATAL FACTORS															
Sex															
Male (ref)				Ref	Ref	Ref				Ref	Ref	Ref	Ref	Ref	Ref
Female				-0.39	-0.97;0.19	0.188				-0.47	-1.04;0.11	0.111	-0.41	-1.02;0.19	0.179
GA (weeks)				0.437	0.30;0.58	**0.000**				0.43	0.29;0.56	**0.000**	0.42	0.27;0.56	**0.000**
R^2				0.336											
MATERNAL FACTORS															
Age (yrs.)							-0.05	-0.12;0.01	0.083	-0.03	-0.08;0.03	0.315	-0.05	-0.12;0.03	0.200
BMI (Kg/m2)							0.03	-0.02;0.08	0.257	0.04	-0.01;0.08	0.107	0.03	-0.02;0.08	0.223
Level of Education															
Tertiary (Ref)							Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Education up till Secondary School							0.10	-0.62;0.80	0.801	-0.10	-0.70;0.50	0.734	-0.09	-0.73;0.55	0.791
HIV status at enrolment															
Negative (Ref)							Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Positive							0.20	-0.59;0.98	0.622	0.16	-0.51;0.82	0.643	0.02	-0.69;0.73	0.960

Table 11: Multivariate linear regressions between paternal and maternal factors and birth head circumference

	Model 1 (Paternal Factors)			Model 2 (Model 1 + Neonatal factors)			Model 3 (Maternal Factors)			Model 4 (Model 3 + Neonatal factors)			Model 5 (Paternal + Maternal + Neonatal factors)		
Variables	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**
Gestational Diabetes Status															
Non-GDM (Ref)							Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
GDM							0.19	-0.81;1.18	0.709	0.15	-0.69;0.99	0.728	0.26	-0.64;1.16	0.565
Marital Status (categories)															
Married/Cohabiting (Ref)							Ref	Ref	Ref	Ref	Ref	Ref			
Single							0.28	-0.40;0.95	0.417	0.21	-0.36;0.78	0.465			
R^2							0.04			0.33					

Table 12: Multivariate linear regressions between paternal and maternal factors and birth ponderal index

	Model 1 (Paternal Factors)			Model 2 (Model 1 + Neonatal factors)			Model 3 (Maternal Factors)			Model 4 (Model 3 + Neonatal factors)			Model 5 (Paternal + Maternal + Neonatal factors)		
Variables	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**
PATERNAL FACTORS															
Age (yrs.)	0.00	-0.02;0.01	0.681	0.00	-0.02;0.01	0.686							0.01	-0.03;0.01	0.487
BMI	-0.01	-0.04;0.03	0.761	-0.02	-0.05;0.02	0.425							-0.02	-0.05;0.02	0.455
WC	0.00	-0.01;0.02	0.801	0.01	-0.01;0.02	0.518							0.00	-0.01;0.02	0.713
Total Number of Education (yrs.)	0.00	-0.03;0.03	0.935	0.00	-0.03;0.03	0.930							0.00	-0.04;0.03	0.954
Employment Status															
No (Ref)	Ref	Ref	Ref	Ref	Ref	Ref							Ref	Ref	Ref
Yes	0.08	-0.14;0.30	0.471	0.084	-0.13;0.30	0.443							0.10	-0.13;0.33	0.399
Smoking Habits															
Never smoked (ref)	Ref	Ref	Ref	Ref	Ref	Ref							Ref	Ref	Ref
Used to smoke but stopped	0.00	-0.26;0.25	0.984	-0.01	-0.26;0.25	0.955							0.01	-0.25;0.27	0.936
Current smoker	0.03	-0.16;0.22	0.761	0.00	-0.19;0.20	0.993							0.01	-0.19;0.22	0.891
Living with Partner															
No (Ref)	Ref	Ref	Ref	Ref	Ref	Ref							Ref	Ref	Ref
Yes	0.18	-0.02;0.39	0.078	0.18	-0.02;0.38	0.079							0.10	-0.14;0.35	0.415
R^2	0.04														
NEONATAL FACTORS															
Sex															
Male (ref)				Ref	Ref	Ref				Ref	Ref	Ref	Ref	Ref	Ref
Female				0.13	-0.04;0.31	0.133				0.13	-0.04;0.31	0.134	0.13	-0.06;0.32	0.167
GA (weeks)				-0.03	-0.07;0.02	0.238				-0.02	-0.06;0.02	0.373	0.02	-0.07;0.03	0.372

Table 12: Multivariate linear regressions between paternal and maternal factors and birth ponderal index

	Model 1 (Paternal Factors)			Model 2 (Model 1 + Neonatal factors)			Model 3 (Maternal Factors)			Model 4 (Model 3 + Neonatal factors)			Model 5 (Paternal + Maternal + Neonatal factors)		
Variables	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**	**β**	**95%CI**	**p value**
R^2				0.08											
MATERNAL FACTORS															
Age (yrs.)							0.00	-0.01;0.02	0.646	0.00	-0.01;0.02	0.738	0.01	-0.02;0.03	0.632
BMI (Kg/m2)							0.01	-0.00;0.02	0.152	0.01	-0.01;0.02	0.232	0.01	-0.01;0.02	0.269
Level of Education															
Tertiary (Ref)							Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Education up till Secondary School							-0.05	-0.23;0.14	0.620	-0.03	-0.21;0.15	0.728	-0.03	-0.22;0.17	0.794
HIV status at enrolment															
Negative (Ref)							Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Positive							-0.03	-0.23;0.17	0.782	-0.02	-0.22;0.18	0.846	-0.02	-0.24;0.19	0.835
Gestational Diabetes Status															
Non-GDM (Ref)							Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
GDM							-0.05	-0.31;0.20	0.683	-0.04	-0.29;0.22	0.788	-0.02	-0.30;0.25	0.876
Marital Status (categories)															
Married/Cohabiting (Ref)							Ref	Ref	Ref	Ref	Ref	Ref			
Single							0.13	-0.05;0.30	0.151	0.14	-0.03;0.31	0.113			
R^2							0.05			0.08			0.10		

CHAPTER 5: DISCUSSION, PUBLIC HEALTH IMPLICATIONS AND CONCLUSION

5.1 Introduction

There is a growing body of evidence that strongly suggests the role of maternal factors (through various plausible mechanisms) on key birth outcomes including BW, BL, HC and PI. However, little is known on the contribution if any of paternal factors, moreover interactions with maternal factors. This study systematically explored the association of paternal and maternal factors with key birth outcomes and additionally explores their role independent of each other. To our knowledge, this is the first study to explore the role of paternal and maternal factors on birth outcomes, independent of each other in an African population undergoing a known epidemiological health transition.

5.2 Main study findings

In the current study, we observed that fathers who were cohabitating with their partners significantly associated with lower BW after adjusting for neonatal factors... No other paternal factors significantly associated with BW and other neonatal outcomes in all other models. In addition, maternal BMI and age were significant predictors for BW, but after adjusting for paternal and neonatal factors only Maternal BMI remained a significant predictor of BW. As for BL, both maternal and paternal factors were not significant predictors across all models. Paternal BMI was significantly associated with HC, however the association diminished once neonatal and maternal factors were added to the model. As for PI, both maternal and paternal factors were not significant predictors across all models. It is noteworthy that GA and sex at birth were key proximal factors for most birth outcomes in this current study. GA was a consistent significant predictor for BW, length and HC. Whilst sex at birth consistently had a significant association with only BL.

Association of paternal and maternal factors with birth weight

A systematic review conducted by Shah (2010) reported findings in various countries on paternal factors that had an association with the infant's birth weight. In the review, height and birthweight had a positive association with BW, whilst extreme paternal had a negative association or positively associated with LBW. However, in

our study only cohabitating status was the only significant paternal predictor for BW. There was a positive association found between extreme paternal age and BW (Shah, 2010) and this finding supports the results obtained from our study from the bivariate regressions that showed a significant association with BW. The study also further confirmed that paternal factors such as age and low levels of education were associated with LBW – both variables were also included in the current study but did not significantly predict weight (Meng and Groth, 2018).

In the current study, maternal BMI was a significant predictor in BW after adjusting for both paternal and maternal factors. These results are comparable to studies that reported a strong association between an increasing maternal BMI and BW of the infant (Upadhyay *et al.*, 2011) and mothers who had a lower BMI were reported to have infants who had a lower birth weight compared to the group of mothers who had a higher BMI. Additionally, the overall risk of pre-term birth was increased in the cohort study of underweight women which was conducted by Yang and colleagues (Yang, Zhai and Yang, 2010) . The systematic review and meta-analysis also established that underweight women in both developed and developing countries had an increased risk of having a LBW infants technically agreeing and aligned to our findings (Han *et al.*, 2011).

Additionally, there was a positive significant association between maternal age and BW that disappeared after adjusting for other covariates. This relates to findings reported by Kent *et al* who reported that maternal age was a significant predictor in BW (Kent *et al.*, 2013). However, results from a cohort study done in Brazil, reported a U-shaped association between maternal age and odds of LBW, and in the same study findings suggested that social disadvantage was a key driver in unfavourable birth outcomes for young mothers (<16 years) as compared to older mothers (≥35 years) (Restrepo-Méndez *et al.*, 2015). Older mothers were more likely to experience adverse birth outcomes (PTB and LBW infants) due to biological factors such as aging maternal tissues and systems, obstetrical history or cumulative effects of disease (Aras, 2013) and this pattern of results was assessed in both low and high income countries (Goisis *et al.*, 2017).

In the current study results reported that fathers who live with their partners had a positive and significant effect on BW. In relation to this, a study by Misra *et al*.

reported that mothers who were married or cohabitating with their partners had higher spent financial rates on prenatal care which had a positive association with the BW of the infant. This attribution could explain the findings in the current study. The study also reported that single mothers were at a higher risk of having babies with a LBW in comparison with mothers who were married or cohabitating with their partners (Misra *et al.*, 2010). However dissimilar findings were detected in a study that explored the association between partner support in African American families and its influence on pregnancy outcomes and prenatal health behaviours (Straughen *et al.*, 2013). The difference in findings could be explained by the variance on the different indicators used to measure partner or father involvement, population setting and diversity in ethnic groups.

Association of paternal and maternal factors with birth length

Our findings on BL as an outcome summarized above, are similar to study conducted in North India that showed a significant (positive) relationship between BL and GA (Magon and R.S, 2013). Additionally, our findings are similar, with a study done in North America where results exhibited that higher prenatal exposure to unconjugated bisphenol A had an association with sex-specific increase in BL (Veiga-Lopez *et al.*, 2015). A study conducted by Jukic *et al*. reported different results that suggested a lack of association between the length of a woman's pregnancy and an offspring's sex (Jukic *et al.*, 2013). Like our findings, a study reported through a multivariate analysis that GA and sex of the newborn had significant effects on BL of the neonates (Shajari *et al.*, 2006).

Association of paternal and maternal factors with head circumference

Few studies are available on associations with HC. Studies have observed significantly different HC sizes at birth between males and females, males having a larger HC average than female infants (Janssen *et al.*, 2007). These findings are in line with this study's results as males had larger HC compared to females. In our analysis, the full model represented 51.9% of the variance in HC. However, the key drivers to this were neonatal factors (GA and sex at birth) and paternal factors. Unlike the previous associations with BW and BL, maternal factors had the lowest contribution to HC of about 4.2%. Though none of the paternal and maternal factors were significant predictors of HC in the full model, Bouthroom *et a*l (2012) showed

that maternal educational levels were associated with HC sizes in infants (Bouthoorn *et al.*, 2012). Additionally, it has been reported that neonatal HC is associated with maternal anthropometric factors, that is; height and maternal BMI during pregnancy (Wills *et al.*, 2010; Tayade *et al.*, 2018). As for paternal anthropometric factors, studies suggest little contribution towards HC at birth (Pomeroy *et al.*, 2015). However, a study by Knight *et al* suggests contribution of paternal factors to HC (Knight *et al.*, 2005). In the current study, all models showed GA as the only significant predictor in birth HC. Similar to this study's findings, a study reported through a multivariate analysis that GA and sex of the newborn had significant effects on HC of the neonates (Shajari *et al.*, 2006).

Association of paternal and maternal factors with ponderal index

According to Roje *et al* (2004) the PI of a male infant is usually higher than a female (Roje *et al.*, 2004) – findings which were opposite to this study's findings as female infants had higher PI compared to males. The full model in the current analysis showed that maternal factors (4.8%) and paternal factors (4.2%) were comparable drivers for PI. Available literature on PI indicate that maternal chronic conditions such as hypertension had a negative impact on the PI of an infant due to the mother's high risk of having a neonate who is SGA (Onyiriuka and Okolo, 2005). However, the current study gestational diabetes, did not predict PI the sane results.

Biological plausible mechanisms for paternal and maternal influence on birth outcomes

Interest in the degree to which paternal and maternal factors influence neonatal outcomes exists; however, most studies including known interventions are solely centred on mothers. Genetically, the both fathers and mothers contribute to the chromosomal make-up of a newborn. Evidence has indicated that paternal genetic components are associated with the quality of the embryo and have a contribution towards foetal development (Yang *et al.*, 2015; Constantinof, Moisiadis and Matthews, 2016). Paternal factors have also been associated with maternal depression and anxiety during pregnancy, which has shown to have adverse effects on birth outcomes such as preterm birth (Liu *et al.*, 2016). According to Zhang *et al* (2015), the positive association between maternal height and the following neonatal

birth outcomes – BW and BL, is due to foetal genetics. The study indicated that shorter mothers delivered their infants at earlier GA stages with a lower BW and BL. However, the association between maternal height and GA is more likely to be causal (Zhang *et al.*, 2015). An additional study reported similar findings where women of shorter stature (< 163 cm) had a shorter pregnancy length and lower risk of post-term birth and were at a higher risk of PTB in comparison to taller women (Myklestad *et al.*, 2013). Positive associations with paternal height and pregnancy length, or with the risks of pre- and post-term births have been reported (Myklestad *et al.*, 2013). Additionally, findings have been reported in another study where paternal height was correlated with BW (r = 0.19) and BL (r = 0.33), independent of maternal (Pomeroy *et al.*, 2015).

Associations between GA and birth weight

GA remained a primary predictor of BW, regardless of model presented the current study. It is known that GA and/or preterm or term deliveries are key drivers to birth outcome especially BW (Quinn *et al.*, 2016). GA in the current study was not treated as a primary outcome. However, it was treated as a covariate and assisted in investigating the independent role of both paternal and maternal factors on birth outcomes regardless of age at birth. A study conducted in Turkey with a large sample size of 68 255 live singletons confirms our findings in that it reported a positive and significant relationship between GA and BW.(Topçu *et al.*, 2014). Additional studies reported similar findings that showcased the increases in BW as GA increased (Oken *et al.*, 2003). Like the current study's, a study reported through a multivariate analysis that gestational age and sex of the newborn had significant effects on BW of the neonates (Shajari et al 2006)

Public Health Relevance

Data shows, infants who are born prior to 37 completed weeks of gestation denote substantial public health concern as PTB, LBW and SGA, are the leading cause of neonatal deaths that are not related with child defects (Gavin, Nurius and Logan-Greene, 2012). Infants born with a LBW have higher morbidity and mortality rates than infants born with a normal weight. Furthermore, they are also at a higher risk of developing adverse health and chronic diseases across the life course, such as diabetes, hypertension and heart disease (Goldenberg and Culhane, 2007) as well

as poor mental and psychological development during school-going age (Hack *et al.*, 2009). HBW may not be directly linked to the high risk of infant mortality; however it has been related to long term health issues such as obesity and cancer (Holman, Buchanan and Cancer Prevention During Early Life Expert Group, 2016). Infant mortality and PTB are a key measurement and indicator of a population's health status and life expectancy (Kim & Saada 2013). These birth outcomes not only have adverse effects on the infant; however also have detrimental health effects on the wellbeing of the mother (Ruiz *et al.*, 2015).

Limitations and strengths of the study

To our knowledge, this is the first study to evaluate the relationship between paternal and maternal risk factors and birth outcomes within the South African context. The cross-sectional nature of the study means causality cannot be inferred. Since this is a secondary data analysis not all known maternal and paternal factors that have been shown to influence birth outcomes were collected and included in the analysis. However, the study shows that most paternal and maternal factors were explored. Paternity was self-reported and could not be verified by DNA for ethical reasons hence this might have introduced bias in the main findings. Another limitation of the study is the small sample size, which may have affected the power to detect some associations that were reported in larger sample size studies. The strength of the study also exists in the analysis performed, which included multivariate regression modelling, that could ascertain contribution and comparison of maternal and paternal factors to various birth outcomes, alone and independent of each other.

Implications of public health findings

the relationship s between paternal and maternal factors and neonatal outcomes in this study indicate a need for targeted interventions for both paternal factors especially adiposity measures, however pregnancy related variables especially GA seems to be key intermediate determinants of neonatal outcomes. It is vital to understand the variations in birth measurements in relation to their impact of outcomes in both the perinatal period and later life, to provide opportunities for tailor made prevention strategies and public health interventions. The work done on the developmental origins of health and adult disease strongly indicate the importance of

better birth outcomes as a strong intervention for reducing chronic diseases later in life. Interventions have been targeted to improve better maternal health as a means of improving birth outcomes.

Conclusion

Fathers who were cohabitating with their partners positively associated with BW after adjusting for neonatal factors. As for BL, both maternal and paternal factors were not significant predictors across all models. Whilst for HC, only paternal BMI was significant predictor in a bivariate association. Findings affirm the important role of both paternal, maternal and neonatal factors in relation to birth outcomes. A significant amount of existing research, policies and obstetrical care do not routinely take paternal factors into account. Going forward, paternal characteristics should be incorporated into studies, policies and programs targeted at reducing adverse birth outcomes as well as the inclusion and engagement of fathers directly into prenatal care. Furthermore, the current evidence supports the existence of paternal effects on birth outcomes; however, it needs to be further researched and strengthened and a need for longitudinal studies to confirm that the association as causal In order to achieve this, further studies with more comprehensive paternal and maternal factors, such as physical activity and chronic diseases should be conducted. The inclusion of a larger sample size would also be of great benefit in order to obtain a valid representation of the targeted population and a smaller margin of error.

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APPENDICES:

Appendix i: Ethics clearance certificate

R14/49 Miss Tadiwa Melinda Mashonganyika et al

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)

CLEARANCE CERTIFICATE NO. M161189

NAME: (Principal Investigator)	Miss Tadiwa Melinda Mashonganyika et al
DEPARTMENT:	MRC/Wits Developmental Pathways for Health Research Unit Chris Hani Baragwanath Academic Hospital
PROJECT TITLE:	Dads Matter Too: The Role of Paternal Factors on Birth Outcomes
DATE CONSIDERED:	25/11/2016
DECISION:	Approved unconditionally
CONDITIONS:	
SUPERVISOR:	Juliana Kagura
APPROVED BY:	Professor P. Cleaton-Jones, Chairperson, HREC (Medical)
DATE OF APPROVAL:	01/03/2017

This clearance certificate is valid for 5 years from date of approval. Extension may be applied for.

DECLARATION OF INVESTIGATORS

To be completed in duplicate and **ONE COPY** returned to the Research Office Secretary in Room 10004,10th floor, Senate House/2nd floor, Phillip Tobias Building, Parktown, University of the Witwatersrand. I/We fully understand the the conditions under which I am/we are authorised to carry out the above-mentioned research and I/we undertake to ensure compliance with these conditions. Should any departure be contemplated,from the research protocol as approved, I/we undertake to resubmit to the Committee. **I agree to submit a yearly progress report**. The date for annual re-certification will be one year after the date of convened meeting where the study was initially reviewed. in this case, the study was initially review in November and will therefore be due in the month of November each year. Unreported changes to the application may invalidate the clearance given by the HREC (Medical).

Principal Investigator Signature

Date 02/03/2017

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES

Appendix ii: FOB clinical data collection checklist

Soweto Fetal Growth Study –
Father of the Baby
Clinical Data Collection Checklist

Patient Label

Father ID: | | | | | | | | | | | |

Date: D D M M Y Y

1. Height | | | | . | | cm

2. Weight | | | | . | | kg

3. Waist circumference | | | | . | | cm

4. Pulse and blood pressure

1. Pulse | | | | beats per minute
   Systolic bp | | | | mmHg
   Diastolic bp | | | | mmHg

2. Pulse | | | | beats per minute
   Systolic bp | | | | mmHg
   Diastolic bp | | | | mmHg

3. Pulse | | | | beats per minute
   Systolic bp | | | | mmHg
   Diastolic bp | | | | mmHg

Research Assistant: _____ **Date:** _____

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5. Full body DXA Scan (please attach report)	YES	NO
6. Abdominal fat ultrasound	YES	NO

Medial (visceral) | | | | . | | cm

Subcutaneous | | | | . | | cm

Radiographer/Research Assistant:_____ **Date:**_____

Appendix iii: FOB SES questionnaire

FV: FBSES

University of the Witwatersrand, Johannesburg
Developmental Pathways for Health Research Unit
Department of Paediatrics and Child Health

Soweto Fetal Growth Study:
Father of the Baby's Socioeconomic Status Questionnaire

Father ID: _____

Interviewer's name: _____

Interview date: _____

Page 1 of 3

Father of the Baby's Socioeconomic Status Questionnaire

1. **Does he live with his female partner (the mother of his unborn baby)?**

   _____ Yes

   _____ No

2. **What is his highest level of education attended?**

   _____ No school attended _____ Technical training

   _____ Primary _____ College

   _____ Secondary _____ University

3. **Total number of years of formal education** _____
   Show the number of school years + tertiary education eg. 12 (school) + 4 (university)
   (school PLUS tertiary education (college, technicon, university). Formal education is certificate or degree based.)

4. **Is he currently employed?**
   _____ Yes

   _____ No

   If yes, specify occupation_____

5. **On average, how much does he smoke (mark one only)?**

   _____ Never smoked

   _____ Used to smoke but stopped

   _____ 1-9 cigarettes a day

   _____ 10-19 cigarettes a day

   _____ 20 or more cigarettes a day

6. **Indicate whether he and his biological parents and grandparents have/had the following:**

	No one	Self	Mother	Father	Maternal grandmother	Maternal grandfather	Paternal grandmother	Paternal grandfather
Weight problem/ obesity								
High blood pressure								
Heart problems								
Diabetes (sugar)								
Depression								

Appendix iv: Turnitin report

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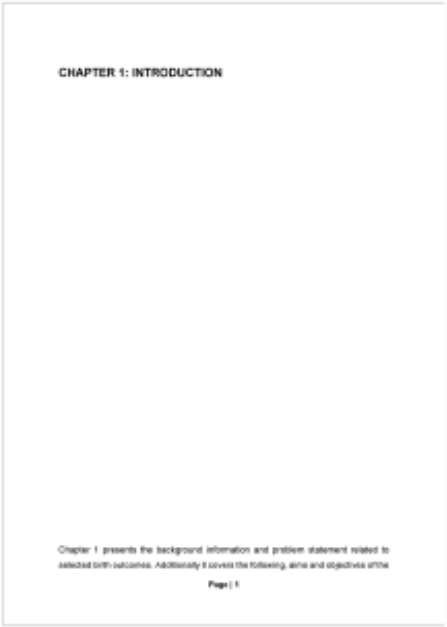

CHAPTER 1: INTRODUCTION

Chapter 1 presents the background information and problem statement related to selected birth outcomes. Additionally it covers the following, aims and objectives of the

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CHAPTER 1: INTRODUCTION Chapter 1 presents the background information and problem statement related to selected birth outcomes. Additionally it covers the following, aims and objectives of the Page | 1 study, related conceptual frameworks, structure of the dissertation and contribution of the candidate to the MSc to the study. 1.1 Background and Problem Statement Globally, the prevalence of infants born before 37 completed weeks of gestation and with a low birth weight (LBW) -weight less than 2500g- continues to increase despite extensive prevention and screening programmes (Altenhöner et al. 2016). On a worldwide scale, early infant deaths (deaths that occur during the first week of life) are accountable for at least 75% of all of all neonatal deaths and 40% of them are due to premature births (Blencowe et al. 2013). Newborns that are born prematurely or small for gestational age (SGA) are at a higher risk of perinatal death and severe disability (Behrman et al. 2007), this is due to the fact that LBW infants are more susceptible to infectious diseases as compared to an infant born with a normal and healthy birth weight (Muchemi et al. 2015). Additionally, they stand a higher chance of developing chronic diseases such as Type II Mellitus Diabetes and cardiovascular diseases in their later stages of life (Barker et al. 2005) as well as poor mental and psychological development during school-going age (Hack et al. 2009). An infant's BW is one of the most misunderstood, yet most accessible variables in epidemiology as a baby's weight at birth is strongly associated with mortality risks during the first year of life and, to a lesser degree, with development complications in childhood and the risk of various chronic diseases in adulthood (Wilcox 2001). Usually, the lower the weight is at birth, the higher a baby's risk of infant mortality, making PTB, SGA and LBW a major cause of infant mortality (Darmstadt et al. 2009). In early childhood, PTB babies who survived usually experience a variety of health and developmental issues including, motor delay, cerebral palsy, poor vision, educational deficits such as low IQ, limited academic skills, poor social adaptive skills and respiratory illnesses such as asthma (Mayor 2005; McCormick et al. 2011). The Developmental origins of health and disease (DoHaD) concept sheds light on how early life environment can impact the long-term risk of NCDs